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Review Article

The nasopharynx, eustachian tube and otitis media

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Abstract
It has been classically hypothesized that a mass in the nasopharynx causes an obstacle to air flow through the eustachian tube, thereby creating a negative pressure in the middle ear followed by an effusion. However, examination of the relevant data concerning the supposed obstruction of the eustachian tube by nasopharyngeal carcinomas, choanal polyps and adenoids does not seem to support this cause and effect relationship. Evidence points to other more sophisticated mechanisms which cause negative pressure and an effusion in pathological middle ear conditions. While the hypothesis of a nasopharyngeal mass as the usual obstructive cause of middle ear effusion is hard to maintain, evidence does exist to support the origin of middle ear infection, as seen in acute and secretory otitis media, as being associated, at times, with an ascending infection from the nasopharynx.

Key words: Otitis media with effusion; Otitis media; Eustachian tube; Adenoids; Atelectasis; Cholesteatoma; Nasopharyngeal neoplasms; Nasopharynx

Physiological pressure in the middle ear is a function of the gaseous balance between its air intake through the eustachian tube and gas diffusion from the middle ear into the circulation. This gaseous balance is traditionally thought to be predominantly influenced – for better or for worse – by the adequacy of ventilation through the eustachian tube. If a smaller amount of air reached the middle ear, a negative pressure would result. The eustachian tube opening into the nasopharynx has been viewed traditionally as a strategic point from which air may potentially be obstructed in its ascent into the middle ear cleft. Many or most of the inflammatory disorders of the middle ear are usually considered to be related to such an obstruction (Politzer, 1903; Goodhill, 1979).

The notion that nasopharyngeal pathology represents a potential danger to middle ear ventilation mainly stems from the association of nasopharyngeal carcinoma with middle ear effusion and the association of enlarged adenoids with secretory otitis media. Obstruction of the nasal passages was also thought to hamper middle ear ventilation. As a consequence, the relief or treatment of nasal obstruction (i.e. correction of a deviated septum or removal of nasal polyps) was thought to promote middle ear ventilation in patients suffering from deficient middle ear pressure. This attitude is seldom encountered today. However, the basic premise that an impediment to air flow by some space-occupying lesion in the nasopharynx will usually jeopardize middle ear aeration has not, so far, been critically examined. When one looks carefully at the facts concerning the obstruction by a carcinoma and adenoids of the air flow into the middle ear, the cause–effect relationship appears less convincing.

Nasopharyngeal carcinomas often present themselves as small tumours, which seem most unlikely to interfere with the passage of air into the eustachian tube (Zöllner, 1942; Honjo, 1988; Sato *et al.*, 1988; Van Hasselt and Gibb, 1991). Furthermore, postmortem histological examination of the eustachian tube in patients with nasopharyngeal carcinoma reveals that while sometimes the tumour infiltrates the eustachian tube submucosa and may cause obstruction (Cundy *et al.*, 1973); it usually infiltrates the eustachian tube muscles and does not involve the eustachian tube opening or its lumen at all (Zöllner, 1942; Cundy *et al.*, 1973; Takahara *et al.*, 1986). Various studies (Honjo, 1988; Sham *et al.*, 1992) demonstrated a direct relationship between the frequency of middle ear effusion in nasopharyngeal carcinoma and the extent of its infiltration to the parapharyngeal region where the eustachian tube muscles are most probably infiltrated. Myers *et al.* (1984) and Wei *et al.* (1988) also pointed to the presence of middle ear effusion in cases of other head or neck tumours such as maxillary sinus carcinoma and its surgery. They showed that, in these cases also, damage to the eustachian tube muscles rather than obstruction of the

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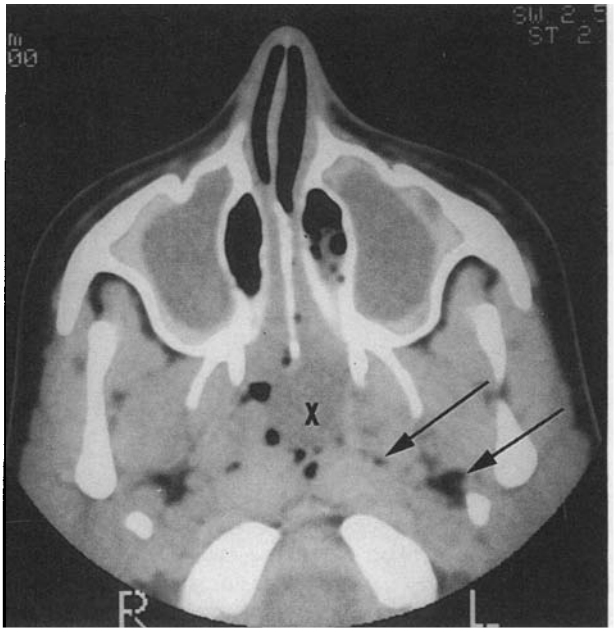

FIG. 1

Choanal polyp (X) filling the nasopharynx. Note air in the eustachian tube and anterior part of the middle ear (arrows). Hearing and tympanic membrane were normal in this patient.

opening, or the lumen, of the tube led to the effusion. Finally, Casselbrandt *et al.* (1988) produced middle ear effusions experimentally by damaging the tensor velo palatini muscles in monkeys. It would therefore seem that a middle ear effusion in cases of nasopharyngeal carcinoma is usually the consequence of faulty middle ear aeration, due to the inability to introduce air through the tube because of its muscles being affected – and not because of its opening in the nasopharynx being blocked by a tumour.

It is interesting to note that common nasal and choanal polyps (Figure 1), which may fill the nasonpharynx to a much greater extent than carcinomas do, are usually not associated with middle ear effusion. Out of 36 consecutive cases of choanal polyps followed up, 31 had normal middle ears, one had a bilateral atelectasis diagnosed years before the polyp appeared, and four (11 per cent) presented with secretory otitis media – one on the opposite side of the polyp. All these patients were children, and their secretory otitis media was noted as being of a mild and fleeting nature.

Other illustrative cases which demonstrate from time to time the lack of a mandatory association between a large mass in the nasopharynx and middle ear effusion are also encountered – such as the patient with a nasopharynx which was almost completely filled by a sarcoma in the absence of a middle ear effusion (Figure 2), or a huge nasopharyngeal teratoma originating from the inner cartilagenous part of the eustachian tube in the presence of an aerated middle ear.

Adenoids usually reach their peak size in children at about the same age as secretory otitis media is most prevalent, i.e. between the ages of two and four years. This is, however, also the time when teeth erupt, and indeed many mothers and physicians see a pathogenic correlation between teething and otitis media. This correlation is usually not accepted by otologists. Thus, what looks like a correlation should not be taken on its own and accepted uncritically as a cause and effect relationship, without some additional supporting evidence.

While there are, so far, no anatomical studies to demonstrate an obstruction of the eustachian tube or its opening by adenoids, there are quite a few studies which raise doubts that adenoids are actually capable of doing so. Critical surveys of the various quantitative studies (Sadé and Fuchs, 1986; Sadé and Luntz, 1991) which have attempted to evaluate the influence of adenoids, their size, and the influence of adenoidectomy on secretory otitis media reveals the following findings:

(a) There is no correlation between adenoid size and secretory otitis media (Roydhouse, 1980; Gates *et al.*, 1988), (see Figure 3).
(b) There is no correlation between the weight of adenoids and secretory otitis media (Gerwat, 1975).
(c) Many children with secretory otitis media were found to be devoid of adenoids or had previously undergone adenoidectomy (Dawes, 1970; Mawson and Fagan, 1972; Kokko, 1974).
(d) Adults with secretary otitis media are usually devoid of adenoids, as are most adults suffering from atelectasis and cholesteatoma.

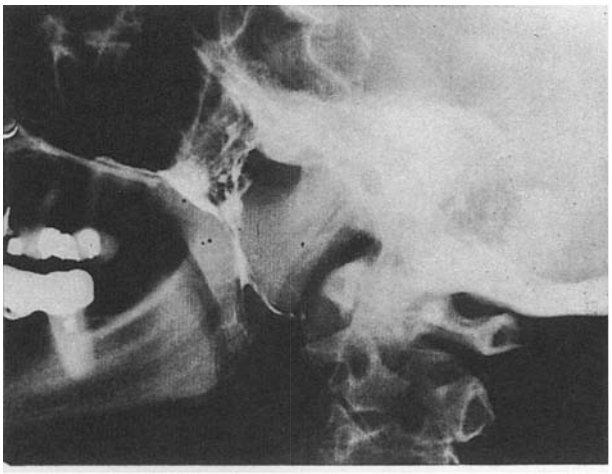

FIG. 2

Sarcoma filling the nasopharynx. The contours of the tumour are delineated with radiopaque material. Hearing and tympanic membrane were normal.

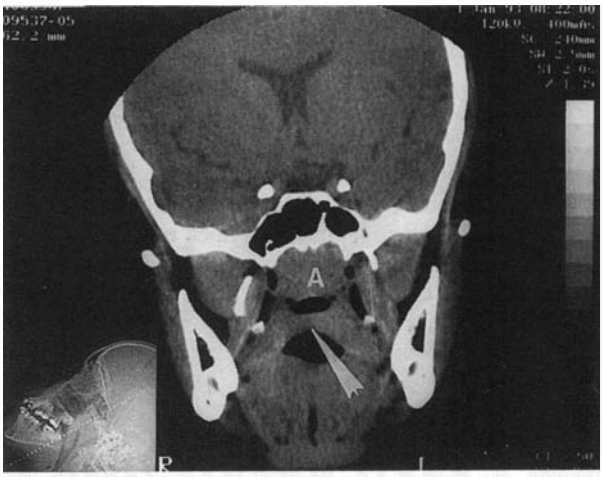

FIG. 3

CT scan of the nasopharynx showing it filled by an adenoidal mass (A). The palate is visible (arrow). This child was not suffering from secretory otitis media.

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TABLE I

Ear	Average Pre-operation	Audiometry Post-operation	12 hours Wet	12 hours Dry	24 hours Wet	24 hours Dry	72 hours Wet	72 hours Dry
R (aspirated)	40 dB	12.3 dB	6	20	3	23	2	24
L (not aspirated)	47 dB	14.4 dB	5	21	2	24	2	24

*Ventilating tubes were inserted into the tympanic membranes of 26 patients (52 ears) with secretory otitis media. Mucus in the right ear was aspirated, but mucus in the left ear was not. When only a paracentesis was performed, the effusion, quite often, protruded through the incision. Thus it was sometimes necessary to remove the obstructing mucus 'plug' before the ventilating tube was inserted. Besides this, no effort was made to remove the mucus from the right ear. Prompt clearance of mucus occurred in both groups. Audiometric levels in the right and left ears were the same before the operation and showed similar improvements post-operatively.

(e) Patients with secretory otitis media show no immediate hearing improvement after adenoidectomy, although there is an immediate improvement after a ventilating tube is introduced (Sadé *et al.*, 1976; Sadé and Biran, 1977; Honjo *et al.*, 1985), as discussed in this paper.

(f) On the other hand, several reports describe a certain reduction in the rate of recurrences of both acute and secretory otitis media in a year or two following adenoidectomy, in comparison to patients who have not undergone adenoidectomy. This can be viewed as a possible long-term benefit of the operation (McKee, 1963; Sadé and Biran, 1977; Gates *et al.*, 1987; Paradise *et al.*, 1987; Maw and Parker, 1988).

Finally, not only is the obstructive role of adenoids in the pathogenesis of secretory otitis media lacking, but a quantitative histological study of the eustachian tube lumen in secretory otitis media and acute otitis media also failed to show any obstruction of the lumen or a significant difference between the size of the lumen in secretory otitis media, acute otitis media and nonpathological specimens (Sadé and Luntz, 1989).

Whereas evidence for the obstructive theory of secretory otitis media (i.e. the *ex vacuo* effect) is lacking, the inflammatory nature of secretory otitis media has begun to reveal itself. The middle ear lining was found to be a true respiratory mucosa at least in the anterior part of the middle ear (Sadé, 1966a). The respiratory mucosa was then found to undergo in secretory otitis media a mucous metaplasia, explaining the origin of the glycoproteins and mucoexudate in the middle ear effusion (Sadé, 1966b; Vered *et al.*, 1972). The mucosa was also found to be studded with inflammatory cells (Sadé, 1966b). Finally, bacteria (mostly dead) and viruses were also found in the effusion (Lim, 1979; Freijd *et al.*, 1984; Chonmaitree *et al.*, 1986; Bernstein *et al.*, 1989). The inflammatory origin of the middle ear effusion in secretory otitis media was better understood, but the question of why the exudative mucus in the middle ear had difficulties in clearing itself still remained unanswered.

TABLE II

Group	No. of ears	Adenoids	Ventilating tubes	Aspiration
A	27	+	+	+
B	23	+	+	–
C	32	–	+	+
D	34	–	+	–
Total no. of ears	116			

*Four groups of children were treated by insertion of ventilating tubes into the tympanic membranes. Two groups had mucus sucked out of the middle ear and two did not. Also, two groups underwent adenoidectomy and two did not.

Much was learned from the effect of ventilating tubes inserted in the tympanic membranes of patients with secretory otitis media. In a study (Sadé *et al.*, 1976), 26 patients with bilateral secretory otitis media (52 ears) had ventilating tubes inserted in both ears. In the right ear, each patient had the mucus aspirated from the middle ear, while in the left ear the effusion was not aspirated. None of the patients underwent an adenoidectomy. Inspection of the ears 12 and 72 hours after the operation showed that all ears were free of fluid (Table I). The pre-operative hearing was the same in both ears, and the post-operative improvement was also similar, irrespective of whether the effusion was aspirated or not. Post-operative inspection of these ears through the opening of the ventilating tube, usually showed a pinkish mucosa and a dry tube opening. A few ears, both from the aspirated group and nonaspirated group, drained into the external ear canal for several days. The amount of fluid drained into the external canal, when present, was a fraction of that usually suctioned from middle ears with secretory otitis media. These observations suggest that, once a ventilating tube was inserted, the effusion cleared from the middle ear through the eustachian tube and not into the external ear canal. In actual clinical practice I aspirate some mucus from the middle ear to facilitate the insertion of the ventilating tube, hoping also that this will minimize obstruction of the ventilating tube. The amount sucked out is only a small part of what is left behind in the various middle ear recesses, attic, antrum, etc. Similar studies were conducted recently by Youngs and Gatland (1988) and Dawes *et al.* (1991) giving the same results.

In 1977 Sadé and Biran conducted an additional study, also taking into account the factor of adenoidectomy. Four groups of children suffering from secretory otitis media were studied. Each patient had a ventilating tube inserted in both ears. In two of the groups, as much mucus as possible was removed by suction from the middle ear during the operation, and in the other two groups the mucus was left undisturbed. From each of these two sets of groups, one group underwent an adenoidectomy and the other did not (Tables II and III). Two weeks after the operation the average hearing improvement was found to be similar in all four groups, and all of them demonstrated similar, almost complete, closure of the air–bone gap. Post-operative inspection of the ears showed that, in general, the external ears were clean, indicating that the mucus clearance had occurred via the eustachian tube. As in the previous study only a few ears from each group presented a slight wetness or slight drainage around the opening of their ventilating tubes.

The conclusion from these four studies (Sadé *et al.*, 1976; Sadé and Biran, 1977; Youngs and Gatland, 1988; Dawes *et al.*, 1991) is that clearance of the middle ear

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TABLE III

Group	No. of ears	Air–Bone Gap (dB)		
		Before operation	After operation	Long-term
A	27	33.75	9.5	9.5
B	23	33.0	8.4	10.0
C	32	32.5	8.3	12.7
D	34	30.2	6.2	12.7
Total no. of ears	116			

*The air–bone gap was similar in all four groups before, as well as after, the operation. However, 1.5 years later the two groups of patients who had undergone adenoidectomy fared better.

mucus is not hampered by obstructing adenoids (nor indeed by any other obstructive factor in the lumen of the eustachian tube, (Sadé and Luntz, 1989) nor by ciliary deficiency, as the mucus disappeared relatively swiftly and efficiently regardless of whether the adenoids were removed or the mucus was aspirated. The single factor which brought about clearance of mucus from the middle ear was the ventilating tube.

The way in which the ventilating tube brings about mucus clearance is most probably related to the relief of negative pressure in the middle ear. This can also be deduced from the return of a retracted drum to its physiological position after insertion of a ventilating tube (Buckingham, 1982). The association of retraction of the tympanic membrane with a long-standing middle ear effusion has also been recognized and documented (Sadé, 1979a).

We see that secretory otitis media is characterized by the synthesis and production, of a sero-mucoexudate (Vered *et al.*, 1972), and by an inflamed metaplastic middle ear mucosa (Sadé, 1966a; Sadé, 1966b). Accumulation of this exudate in the eustachian tube will, by itself, obstruct the ascendance of air to the middle ear, as verified also by fibre optic endoscopy (Magnan *et al.*, 1989). Propagation of mucus through the eustachian tube will block the tube and may leave behind a middle ear negative pressure. Any gas left in the space behind the mucus blob in the middle ear will be absorbed thus increasing the negative pressure created by the forward (i.e., outward), piston-like movement of the mucus through the eustachian tube (Figure 4). This mechanism may be viewed as a pseudo-obstruction, or secondary obstruction of the eustachian tube, which is the result of the mucus and effusion produced in otitis mainly from the middle ear mucosa (Sadé, 1966b). Experimental production of this mechanism was provided by Hilding (1944) and later by Murphy (1979).

Secretory otitis media was previously thought to be a sterile, noninflammatory entity unrelated to microorganisms. Today, however, it is considered to be an inflammatory disease and a sequel to upper respiratory or middle ear infection (Sadé, 1966a; Dawes, 1970; Hinchcliffe, 1972; Lim, 1979; Maw and Parker, 1988). While 70 per cent of ears with acute otitis media yield microorganisms on culture (Freijd *et al.*, 1984; Chonmaitree *et al.*, 1986), secretory otitis media is mostly characterized by the find-

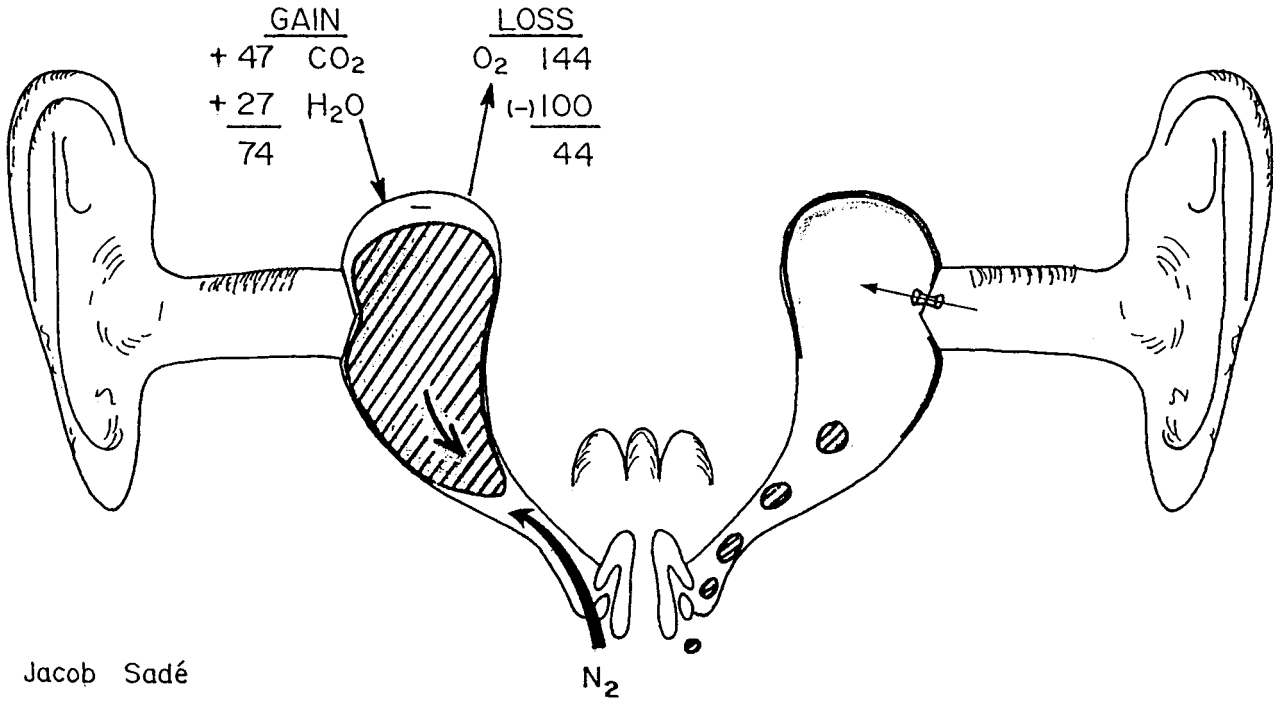

FIG. 4

Schematic representation of mucus, produced in the middle ear, travelling for some distance in the eustachian tube and obstructing it. Behind the mucus, which is stranded in the middle ear, a vacuum is formed, and is augmented by a negative gas balance. The gas balance is the result of H_2O and CO_2 inflow yet a greater O_2 outflow (Sadé and Luntz, 1993). Note the relief of negative pressure in the left ear upon insertion of a ventilating tube.

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ing of dead bacteria and may be viewed as a residual outcome of acute otitis media and upper respiratory infections or as a subacute otitis media. This is a clinically extinguished, inflammatory reaction.

Nasopharyngeal microorganisms are usually nonpathogenic, yet it is often possible to also isolate *Haemophilus influenzae*, *Streptococcus sp.* and *Branhamela catharalis* from the nasopharynx. The period during which these bacteria are most often detected in the nasopharynx corresponds to the period of peak incidence of acute otitis media in children, i.e. at around two years of age (Chonmaitree *et al.*, 1986; Freijd *et al.*, 1984; Stenfors and Raisanen, 1991). The specificity of the nasopharyngeal microorganisms in respect to otitis media was further established in a study by Loos *et al.* (1989), who found that the specific outer membrane protein profile and the DNA profile of nasopharyngeal microoganisms correspond to those detected in the inflamed ears of the same individuals.

Whereas no immediate hearing relief is seen after adenoidectomy in secretory otitis media patients (Honjo *et al.*, 1985), some studies showed an average greater hearing improvement after a longer follow-up, up to 18 months (McKee, 1963; Sadé and Biran, 1977; Paradise *et al.*, 1987; Gates *et al.*, 1988; Maw and Parker, 1988), in patients who underwent adenoidectomy compared to those who did not. This observation suggests that adenoidectomy will reduce, in a certain percentage of cases (10–15 per cent), reinfection of the middle ear.

It seems that the eustachian tube and its aperture are structured in a way which makes them difficult to obstruct (Sadé *et al.*, 1959; Sadé, 1979b). It appears that while the negative pressure in secretory otitis media cannot usually be attributed primarily to an eustachian tube obstruction in the nasopharynx, there is sufficient evidence to implicate the nasopharynx as a source of infection that may ascend to the middle ear, causing acute otitis media. This may later develop into a subacute form, or secretory otitis media.

Finally, the view that middle ear negative pressure or middle ear effusion should have only one pathogenic origin is somewhat simplistic, as we see it associated with a carcinomatous muscular infiltration in nasopharyngeal carcinoma, middle ear inflammation in secretory otitis media, extreme relative pressure differences in barotrauma, or excessive gas loss by diffusion (Sadé and Luntz, 1993) coupled with an underdeveloped mastoid in atelectasis and cholesteatoma (Sadé, 1992). These four separate clinical entities are most probably related to different pathogenic mechanisms especially as far as their middle ear effusion is concerned.

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