Paparella: Volume II: Otology and Neuro-Otology

Section 3: Diseases of the Ear

Part 3: Middle Ear and Mastoid

Chapter 27: Otitis Media with Effusion

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Middle ear effusions are referred to in the literature by a variety of synonyms or, in some instances, pseudonyms. These terms include serous otitis media, catarrh of the middle ear, tympanic hydrops, tubotympanic catarrh, hydrotubotympanum, glue ear, exudative catarrh, otitis media with effusion, tubotympanitis, allergic otitis media, and mucoid ear. In this chapter we discuss fluids that arise from the eustachian tube, middle ear, and mastoid but exclude otorrheic cerebrospinal fluid and allude to acute purulent otitis media only briefly for the sake of completeness, since it is fully discussed in the chapter that follows.

There are various causative and contributory factors related to the formation of fluids in the middle ear, some of which are not clearly understood. Many of these causative factors are shared by other inflammatory diseases of the middle ear cleft, including chronic otitis media, cholesteatoma, tympanosclerosis, and cholesterol granuloma. In addition, middle ear diseases with effusions in children can lead to chronic inflammatory disease in the adult, thus representing the beginning of a continuum of changes. Problems with fluid in the middle ear are very common, especially in children, and on that basis alone merit our careful consideration and early diagnosis and treatment, not only to cure but to prevent more serious sequelae.

Historical Considerations

The problem of fluid in the middle ear has probably been recognized for hundreds of years. In 1869, Politzer described a condition that he termed "otitis media catarrhalis". In his classic book, *The Diseases of the Ear* (1902), he devoted 60 pages to the subject. His descriptions of clinical features were complete and remain relevant to date. He recognized secretory and adhesive forms of the condition. The therapy that he advocated consisted of insufflation of air and paracentesis of the middle ear, which were intended to equalize the atmospheric pressure on both sides of the tympanic membrane. The principles of both ventilation and drainage of the middle ear have remained basic in the management of middle ear fluids to date. Following the descriptions by Politzer, these fluids received little attention until the time of the Second World War. Since then, many articles have been written on the subject.

The clinical "challenge" of this disorder was described by Hoople (1950) in an article in which the features of middle ear effusions were clearly elucidated for the otolaryngologist. He appropriately emphasized the importance of diagnostic paracentesis so that many of these conditions, especially in children, would not escape detection. Laboratory and animal studies, including chemical analyses of fluids, were described by Senturia and colleagues (1958, 1962). An important contribution to the management of middle ear effusions was introduced

by Amrstrong in 1954 when he advocated the use of an indwelling polyethylene tube through the drumhead in order to achieve ventilation and drainage, as espoused earlier by Politzer.

Epidemiology

Otitis media is one of the most common pediatric health problems, especially among young children. Koch and Dennison (1974) reported that otitis media was the most frequent diagnosis for illness and was the most frequent reason for office visits excluding visits for well-baby and well-child care. Howie and associates (1975) reported that two-thirds of the children visiting their office's practice had at least one episode of otitis media by the age of 2, and one in seven children had more than six episodes. There are only a few studies dealing with the incidence of persistent effusions of the middle ear. Approximately one-third of children with acute otitis media were found to have middle ear effusions that persisted for 4 or more weeks (Pelton et al, 1977). In a Boston study (Teele et al, 1980a), persistent effusion in the middle ear was found in 70 per cent of the children at two weeks after the first episodes of otitis media, 40 per cent at 1 month, 20 per cent at 2 months, and 10 per cent at 3 months.

Age

Otitis media is predominantly a disease of infants and young children. In the Boston study (Teele et al, 1980b) of more than 2500 children from birth to age three, 13 per cent had at least one episode of otitis media by 3 months of age, 25 per cent had one or more episodes by 6 months of age, 47 per cent had one or more episodes by 12 months of age, and 65 per cent had otitis media by 24 months of age. In general, the highest incidence of acute otitis media occurs between 6 and 24 months of age. The incidence declines with age except for a slight upward trend between 5 and 6 years of age. Persisten effusions in the middle ear were found to bemore common in younger children (Pelton et al, 1977). Fifty per cent of children 2 years old or younger had middle ear effusions that lasted for four weeks or more after an episode of acute otitis media, whereas only 20 per cent of children older than two years of age had persistent otitis media.

Sex

Most studies reported male predilection for single, recurrent, and persistent middle ear effusions (Stewart et al, 1984; Teele et al, 1980b).

Race

Eskimos, American Indians, and Hispanic children have a higher incidence of otitis media than American whites. Blacks have a lower incidence of otitis media than whites. Eskimos and American Indians have a higher incidence of otorrhea (Teele et al, 1980b; Wiet et al, 1980). These racial differences may be explained by anatomic differences in the length, width, and angle of the bony eustachian tube in American blacks, whites, and Indians (Doyle, 1977). However, socioeconomic and cultural factors may also play an important part in racial differences in the incidence of otitis media.

Socioeconomic Conditions

Socioeconomic factors that cause crowding, poor hygiene, inadequate nutrition, delay in seeking medical attention, and poor compliance with treatment plans may increase the incidence and severity of otitis media. These speculations have been supported by a number of studies. Children living in urban areas contracted otitis media significantly more than those living in rural areas. The reason for this is considered to be the higher density of population in urban areas, especially those with day-care centers (Pukander et al, 1984). In the Boston study, children living in households with many members were more likely to have otitis media than were children living in households with few members (Teele et al, 1980b).

Genetic Factors

Several studies support genetic predisposition as a risk factor for otitis media. Teele and coworkers (1980b) found that children who had single or recurrent episodes of otitis media were more likely to have siblings or parents with a history of otitis media than were children who had no episodes of otitis media.

Season

The incidence of otitis media peaks at its highest during the winter and spring. The epidemiologic association between otitis media and viral upper respiratory infections was shown by Henderson and colleagues (1982).

Breast-Feeding

Breast-feeding seems to protect an infant from bouts of otitis media, probably because of the transmission of specific immunoglobulins. However, opinions are contradictory on the protective effect against otitis media of breast-feeding. Some reported no correlation between the duration of breast-feeding and the frequency of otitis media, whereas Schaeffer (1971) and Pukander and coworkers (1984) reported a lower incidence among those breast-fed longest.

Etiology and Pathogenesis

Otitis media with effusion is caused by multiple factors and complex interactions of biochemical, immunologic, and inflammatory mediators in the middle ear cavity. Among these factors, abnormal function of the eustachian tube, mucosal changes, presence of microorganisms, the effect of inflammatory cells, and inflammatory mediators seem to have the most influence on the etiology and pathogenesis of otitis media with effusion.

Eustachian Tube

The eustachian tube has at least three physiologic functions with respect to the middle ear. These are (1) protection from nasopharyngeal secretions and pressure, (2) clearance of secretions from the middle ear into the nasopharynx, and (3) ventilation of the middle ear to equlibrate air pressure with atmospheric pressure and to replenish oxygen that has been absorbed (Bluestone and Klein, 1983). Testing the physiologic function of the eustachian tube is a problem that has not been completely solved. Normally the air in the middle ear space

is gradually absorbed because of the greater total partial pressure of gases in the middle ear compared with the bloodstream when the eustachian tube is closed. This results in a subatmospheric pressure in the middle ear space. Hence, when the eustachian tube is momentarily opened with each swallow, atmospheric air is drawn into the middle ear from the nasopharynx to equalize the pressure.

Ingelstedt (1963) reported that if the eustachian tube is suddenly occluded, the intratympanic pressure will, within 1 hour, be about 10 to 20 mm of mercury less than the extratympanic pressure. He has also shown that the amount of gas drawn into the tympanic cavity during the act of swallowing decreases as negative pressure in the closed space of the ear increases. A negative pressure of -25 mm Hg in the middle ear with a positive pressure of 5 mm Hg in the nasopharynx represents the same pressure gradient as a negative pressure of -30 mm Hg alone in the middle ear. The air flow within the former has been shown to be eight times greater than within the latter. Ingelstedt calls the mechanism of this phenomenon "occlusion by suction" and says it is due to narrowing of the isthmus by direct suction of the lowered pressure in the tympanic cavity. The slight positive pressure in the nasopharynx apparently overcomes this negative suction effect.

Studies have been done on patients with acute catarrhal symptoms of the common cold. A small intra-aural negative pressure of -5 mm Hg in these patients could not be eliminated by swallowing. In healthy volunteers, on the other hand, intratympanic pressure had to be reduced to -30 or -50 mm Hg before a "locking" of the eustachian tube occurred. Slight positive nasopharyngeal pressure, however, relieved this locking. It is apparent, therefore, that mucosa swollen by acute inflammation accentuates occlusive action through the mechanism of suction mentioned before. There are, of course, other mechanisms of obstruction that can occlude the eustachian tube (eg, developmental and anatomic factors, tumors, infections).

The result of any tubal occlusion is a decrease in the intratympanic pressure due to oxygen resorption from the middle ear space. Because the walls of the middle ear cannot collapse, the negative intratympanic pressure is maintained and transudation of fluid from the vasculature into the tympanum may result. This results in edema of the mucoperiosteum and decreased diffusion of oxygen into the bloodstream. The final result might be an actual decrease in the oxygen tension below that of venous blood as a result of oxygen consumption by surface epithelium of the mucosa. This environment lowered in oxygen might affect certain microorganisms present and permit long-term, low-grade infections.

Mucosal Changes

The lining of the normal middle ear space is an extension and modification of the respiratory epithelial membrane of the nasal cavity and its accessory sinuses, the larynx, pharynx, trachea, and bronchi, down to the smallest terminal bronchiole. Ciliated cells are found in the nasal cavity, accessory sinuses, anterior nasopharynx, larynx, and trachea and along the entire length of the bronchi. In the bronchioles that measure from 0.3 to 0.4 millimeter in diameter, the predominating epithelium is cuboidal and low columnar, with many nonciliated cells interspersed with ciliated cells. The presence of cilia depends on the diameter of the bronchiole, and in bronchioles 1 millimeter in diameter, ciliated cells outnumber any other type. Goblet cells are frequently scattered among the ciliated epithelial

cells. These findings in the lower respiratory tract compare in a general way with those in the middle ear, in that the epithelial lining of the middle ear participates in the same transition of histoarchitectural changes as does that leading to the terminal bronchiole. In this case, the transition of cellular type extends from the eustachian tube to the tympanum, to the antrum, and finally to the mastoid air cells (Kawabata and Paparella, 1969a, b).

Ciliated cells in the middle ear are especially prevalent near the opening of the eustachian tube and the inferior aspect of the middle ear, and they decrease gradually posteriorly toward the mastoid. Ciliated cells do not generally appear to line the medial surface of the tympanic membrane, the posterior superior part of the promontory, the posterior attic, the aditus, or the mastoid air cell system. The distribution of ciliated cells appears variable in normal subjects. The presence of cilia can be confirmed by watching the evacuation within minutes following introduction of foreign material into the middle ear, as it goes through the eustachian tube to the nasopharynx (Sadé, 1966).

Three basic types of epithelial cells are found in the mucoperiosteum lining the middle ear and mastoid: ciliated, nonciliated, and basal cells. *Ciliated cells* are usually cuboidal or columnar, with dark cytoplasm resulting from high concentration of intracellular organelles. Some *nonciliated cells* are cuboidal or cylindrical, but most of them are flat. Microvilli instead of cilia project into the middle ear cavity from their surface. The nonciliated cells become cuboidal or squamous as they approach the antrum and mastoid. In humans, some nonciliated cells contain secretory granules. *Basal cells* are found below the ciliated and nonciliated cells and rest on the basement membrane. They may serve as precursors for ciliated and nonciliated epithelial cells.

The subepithelial layer, or lamina propria, is a thin layer between the periosteum of the temporal bone and the basement membrane of the epithelial cells. Within this space lie collagen fibers, fibroblasts, capillaries, and nerve filaments represening branches of the tympanic nerve plexus of the glossopharyngeal cranial nerve.

Pathogenesis is dependent on various extrinsic factors of etiopathogenesis, whereas the form that otitis media takes seems to rely mostly upon relative activity of the subepithelial space and epithelium. Pathologic changes in the epithelium during serous and purulent otitis media consist primarily of dilatations of the intercellular space (Juhn et al, 1977; Lim et al, 1979; Paparella et al, 1970b). The subepithelial space is observed to increse in thickness in *serous* otitis media due to edema associated with some degree of capillary dilatation and vascular congestion and no, or only a few, inflammatory cells, whereas in *purulent* otitis media the increased thickness is due to severe edema, capillary dilatation, and vascular congestion with infiltration of large numbers of neutrophils and an increase in fibroblasts (Paparella et al, 1985). In *mucoid* otitis media, the epithelium shows a remarkable proliferation, an increase in goblet cells, and intraepithelia and subepithelial glandular formations (Juhn et al, 1977; Tos and Bak-Pederson, 1972, 1973), and the subepithelial space thickens due to a proliferation of fibroblasts, fibrosis, edema, vascular dilatation, increased vascularization, and abundant infiltration of lymphocytes, plasma cells, and macrophages (Paparella et al, 1985).

The similarity of findings among the groups suggests a strong relationship between them. The ability of certain types of otitis media to evolve into another type substantiates the concept of the continuum for some patients. This concept has been demonstrated in longitudinal histopathologic studies of experimentally induced otitis media in animals from day 1 of experimentation to 6 months (Goycoolea et al, 1979; 1980a). A continuum of mucoperiosteal changes that was gradual and consistent was demonstrated experimentally using various techniques including immunocytochemistry. Using these observations as a base, the authors postulated and documented the concept of a middle ear defense system. Nonspecific and specific defense systems as well as localized mucoperiosteal immunity were described. It was also observed that effusions followed a continuum.

Microorganisms

The most commonly isolated bacterial pathogens are *Streptococcus pneumoniae* and *Haemophilus influenzae* (Liu et al, 1975). Other organisms identified in middle ear effusion include Group A *Streptococcus, Staphylococcus aureus, Branhamella catarrhalis,* gram-negative enteric bacilli, and *Staphylococcus epidermidis* (Giebink et al, 1979).

Inflammatory Cells

Inflammatory cells are present in both mucosa and effusions of the middle ear. In acute purulent otitis media, polymorphonuclear leukocytes predominate at first, but in a few days lymphocytes and macrophages appear. The macrophages increase in number in the course of otitis media in humans (Palva et al, 1978) and in experimental otitis media (Juhn et al, 1984). Lim and associates (1979) reported that the cytologic profile of an effusion correlates well with the results of bacterial culture. Neutrophilic counts were higher in culture-positive middle ear effusion than in culture-negative effusions. Phagocytes were noted in 37 per cent of culture-positive middle ear effusions and 23 per cent of sterile effusions. The presence of immunoglobulin-producing plasma cells and macrophages has been reported (Goycoolea et al, 1980d). These cells interact with each other and produce numerous humoral mediators such as chemotactic factor, interferon, and migration-inhibitory factors (Bernstein, 1976).

Inflammatory Mediators

Inflammatory mediators including histamine, prostaglandins, and leukotrienes have been identified in middle ear effusions. These inflammatory mediators cause vasodilation and increase the permeability of the mucosa and blood vessels of the middle ear. Berger and coworkers (1984) reported that concentrations of histamine in middle ear effusions were higher than in blood. Histamine levels in mucoid middle ear effusion were higher than in the serous type of middle ear effusion. There was no difference in histamine levels of allergic versus nonallergic patients. Berger and colleagues postulated that the histamine is released from mast cells located in the subepithelial layers of tympanic mucoperiosteum. Since there was no difference in histamine levels between allergic and nonallergic patients, they suspected that the mechanisms of histamine release may not be solely the type I immediate type of hypersensitivity but may include type III, which involves immune complexes through activation of the complement system producing anaphylatoxin, which may degranulate mast cells and release histamine.

Arachidonic acid metabolites such as prostaglandins (PGs) and leukotrienes (LTs) appear to play an important role in the pathogenesis of middle ear effusion. In earlier studies, concentrations of PGs were measured in human middle ear effusion (Bernstein et al, 1976; Jackson, 1971). Arachidonic acid metabolites in experimental serous otitis media and purulent otitis media have been determined (Jung et al, 1982). As a further evidence of the involvement of PGs in otitis media, PG-forming cyclo-oxygenase was localized in middle ear tissue (Jung et al, 1983). Studies on the effects of inhibitors of arachidonic acid metabolism on the pathogenesis of otitis media have suggested the importance of lipoxygenase products in otitis media even more than that of PGs (Jung et al, 1984). Indeed, leukotrienes and other lipoxygenase products have been identified in middle ear effusions (Jung and Juhn, 1986; Jung, 1988). Some of the lipoxygenase products, especially leukotrienes, are potent bronchial constrictors and chemotactic agents. They can cause plasma exudation and increase vascular permeability. The presence of these inflammatory mediators in middle ear effusion suggests an important role of these potent agents in the pathogenesis of otitis media with effusion. The various middle ear fluids result from different factors and related clinical conditions, some of which are discussed herewith.

Related Clinical Conditions

Adenoid Hypertrophy

The ages at which incidence peaks for recurring middle ear effusions correspond to the period of maximum lymphoid hyperplasia in the nasopharynx. There are at least two possible mechanical factors responsible for this. The first factor is the effect of direct closure of the eustachian tubal orifice by excessively enlarged adenoids. Since this is seldom found, the second factor - obstruction of lymphatics draining the middle ear and eustachian tube - may be of greater importance. Chronic adenoiditis provides a focus of infection adjacent to the eustachian tubal orifice, imposing change of eustachian tubal function as well as retrograde inflammation to the tube. Based on these observations, adenoidectomy, in selected cases, is a very useful procedure. Gates and coworkers (1989) recommended adenoidectomy as the initial surgical management for chronic otitis media with effusion. However, the specific indications and effectiveness remain controversial (Bluestone, 1984; Lemon, 1962).

Cleft Palate

In children who have or have had a cleft palate, there is an increase in the incidence of middle ear effusions as well as other forms of otitis media. This is believed to be secondary to dysfunction of the tensor and levator veli palatini muscles rather than a stenosis of the eustachian tube. A secondary reaction in the middle ear to contamination of the eustachian tube is also a probable factor (Paradise et al, 1969; Stool and Randall, 1967).

Tumors

Unilateral middle ear effusion in an adult should alert the physician to the possibility of a nasopharyngeal tumor. This may be the result of mechanical obstruction alone or that which produces secondary lymphatic obstruction and mucosal edema (Davison, 1958).

Barotrauma (Aerotitis)

This phenomenon is most common after travel by air and is still prevalent in this age of pressurized jet aircraft. Barotrauma can also occur from traveling over hills or mountains, elevator rides down from high levels, or descent into deep water. The problem occurs from rapid descent, during which time atmospheric pressure on the outside of the tympanic membrane and in the nasopharynx is greater than pressure of air in the middle ear. This requires an active opening of the eustachian tube through contraction of the levator and tensor veli palatini muscles, allowing air to be drawn into the middle ear and thereby equalizing pressure. During ascent (pressure higher in the middle ear than in the nasopharynx), air is passively transmitted from the middle ear through the tube and into the nasopharynx, a task that the eustachian tube readily accomplishes under usual conditions. Individuals who have eustachian tubal dysfunction or who have upper respiratory tract infections (eg, eustacheitis, nasopharyngitis, sinusitis) are especially prone to barotrauma.

Barotrauma produces a negative pressure in the middle ear that causes a retraction of the tympanic membrane, pain, immobility of the tympanic membrane and ossicles, conductive hearing loss, and, if the middle ear pressure is not equalized, transudation of serum and bleeding. If the eustachian tube does not quickly ventilate the middle ear to counteract the negative pressure, what little air remains in the middle ear continues to be absorbed and the situation is exacerbated.

Barotrauma can be readily studied in animals. Cats and monkeys in our laboratory were subjected to changes in barometric pressure; in addition, the same findings were reproduced by applying simple direct suction to the middle ear through the eustachian tube. Resultant pathologic findings consisted of an accumulation of blood and serum below an elevated mucoperiosteum and in the middle ear space. Serous fluid was produced as a transudate from intact vessel walls, whereas the hydrostatic pressure gradient between vessels and air in the middle ear was so great as to cause rupture of certain vessel walls and bleeding. These findings were significantly greater in anesthetized animals, suggesting that patients in an airplane, especially during descent, should be awake and seated rather than in a recumbent position.

In barotrauma, the tympanic membrane may appear similar to that seen in serous otitis media or there may be evidence of a hemotympanum with purplish discoloration. Treatment consists of restoring eustachian tube ventilation and drainage, using politzerization and other methods, and is essentially the same as described for serous otitis media. Resolution usually occurs with self-administered Valsalva maneuvers and general medical management to decongest the tube. Chewing of gum is advised to encourage swallowing and tubal ventilation. This effectively doubles the number of swallows per unit of time. Chewing gum and sucking on candy are both helpful preventive measures when flying. A systemic antihistamine-decongestant or nasal decongestant to be used prior to flight time can be prescribed for patients with upper respiratory infection.

Inflammation and Antibiotics

As mentioned above, chronic adenoiditis can act as a focus for spread of infection to the middle ear. Other regional upper respiratory inflammations such as sinusitis,

nasopharyngitis, and rhinitic can behave similarly. In the past, several authors have attributed the entire pathogenesis of recurrent middle ear effusions to the hydrops ex vacuo theory. They either found the fluid in the middle ear to be sterile or attributed any growth to contaminants. Senturia and colleagues (1958) found a very high percentage of bacterial growth in the mucopurulent form of middle ear effusions. Others believe that the apparent increase in the incidence of this condition is due to inadequate antibiotic therapy. This results in a lingering, low-grade infection within the middle ear. The mucosal response to this stimulus is an outpouring of fluid and cells, and eventually even metaplasia of the mucoperiosteal lining of the middle ear, producing goblet cells and mucous glands (Paparella and Lim, 1967).

Allergy

Some of the proponents of the hydrops ex vacuo theory believe allergic edema of the mucosa is the principal cause of eustachian tubal obstruction. Derlacki (1952) reported that "the majority of the cases of chronic secretory otitis with either a clear serum or a much thicker mucus were due to a definitve allergy to house dust or to a food". Jordan (1949) found that 74 per cent of middle ear effusions in 123 patients were due to allergy on the basis of eosinophils in nasal smears, skin tests, and response to allergic therapy. Draper (1967) and Visscher and coworkers (1984) reported that the allergic child is at increased risk of otitis media. Draper concluded that, although allergy is not the only etiologic factor in middle ear effusions, it is an important one that should be considered in appropriate cases.

Iatrogenic Causes

Antibiotic Therapy

Many authors believe that inadequate antibiotic therapy for acute purulent otitis results in a lingering, low-grade exudative infection. This was previously believed to be the major cause of the increased incidence of the condition (Seuhs, 1952). However, recent studies by Van Cauwenberge and colleagues (1984) did not find any correlation between the antibiotic consumption by the child and the prevalence of otitis media with effusion.

Radiation Therapy

Effusions of the ears are commonly found after radiation therapy to the head. This usually results in a serous type of effusion, probably on the basis of disturbed lymphatic drainage of the nasopharynx with secondary mucosal edema and eustachian tubal obstruction (Gamble et al, 1968).

Trauma

Trauma to the torus during adenoidectomy can result in permanent cicatrization and tubal obstruction and secondary middle ear effusion. However, an unusual amount of surgical trauma would need to be inflicted to produce such changes. Nevertheless, the torus tubarius should be carefully protected during adenoidectomy.

Other Factors

Viral infection, immunologic deficiency, and metabolic disturbances have been suggested as possible causes of middle ear effusion. For example, a viral etiology for serous effusion has been clinically suspected but not proved. It is known that patients with hypothyroidism can have effusion, presumably as a result of tubal edema. As diagnostic methods improve in the fields of virology and immunology, the role these factors play in the etiology of middle ear effusions will be better understood.

Classification of Middle Ear Fluid and Otitis Media

Classification of middle ear fluid is helpful to the clinician in understanding the disease process. Four basic types of middle ear fluid can be found: (1) serous, (2) mucoid, (3) bloody, and (4) purulent. Any combination of the four is possible. For classification, it is important to collect the fluid in a glass tube and to look at it with the naked eye against a bright light. This alone usually suffices. Culture and sensitivity studies may be done in selected individual problem cases. It may be necessary to submit the fluid for microscopic cytologic study or, if desired, for chemical study.

Classification of otitis media is synonymous with a differential diagnosis of otitis media. As further information is provided through research, improved methods of diagnosis and management of otitis media should follow. Based on clinical and laboratory studies, the following classification is useful at the present. We have adopted the terminology for these various forms of otitis media as agreed upon at the First International Symposium on Recent Advances in Middle Ear Effusions (1976), which includes (1) acute purulent otitis media (POM), (2) serous otitis media (SOM), and (3) mucoid secretory otitis media (MOM). Although these types can occur as distinct clinical entities with characteristic fluid findings, combinations of POM, SOM, and MOM can occur in clinical practice, so that a given patient may have seromucinous or mucopurulent otitis media. Chronic otitis media (COM) refers to chronic suppurative otitis media, which in patients suggests otorrhea, perforation of the tympanic membrane, and active, irreversible pathologic conditions in the tissue of the middle ear cleft, such as cholesteatoma or granulation tissue. Serous otitis media or mucoid otitis media can be acute or chronic. Chronic is defined as a condition that lasts 3 months or longer.

It is our observation that the various forms of otitis media may be dynamically interrelated in etiologic origin and in pathogenesis or pathophysiology, in that certain transient forms of otitis media can develop into more permanent forms of otitis media. Thus, for example, as seen in animal models and occasionally seen in humans, POM can lead to SOM, to MOM, and finally to COM. As mentioned, there is often overlap between types, especially between SOM and MOM. In most children, in whom fluid in the middle ear predominates, both transudative and secretory activity play a role, and the fluid can often be described as seromucinous.

Serous Fluid

Serous fluid appears clinically as a sterile, pale yellow-colored transudate with low viscosity, resembling serum that is found in serous otitis media, aerotitis, and barotrauma. Here the term "serous otitis media" correctly applies. This is a type of fluid most often found

in the patient with a history of illness of short duration. This type of fluid is commonly found in older children and adults. If bubbles or a fluid level are seen behind the drumhead, the fluid most likely is serous in nature. No mucous strands are present, and the yellow-colored fluid resembles serum. The liquid will clot within a few minutes after removal and exposure to air.

Mucoid Fluid

Clinically, mucoid fluid is a cloudy exudate resulting from cell secretion that occurs in secretory otitis media, glue ear, and mucoid otitis. It is commonly seen in younger children, and the term "mucoid otitis media" may be used. The fluid may be yellowish or dirty gray and, when stagnant for a long time, becomes so tenacious as to resemble glue. Often the history is of infection of longer duration than in serous otitis media. Here the mechanism involves initial alteration of pressure with superimposed cellular activity often related to adjacent foci of infection. Laboratory studies will show numerous mucous strands and a few cells, mostly phagocytes.

Bloody Fluid

Bloody middle ear fluid is found in "idiopathic hemotympanum", barotrauma, chronic serous otitis media, and cholesterol granuloma. It is clear that blood in the middle ear (hemotympanum) can result from tumor (glomus jugulare), trauma (fracture of the temporal bone), or bleeding dyscrasia (eg, leukemia). It is not so clear that blood in the middle ear can result from aerotitis or long-standing serous otitis media. The pathogenesis is similar to that of serous otitis media and involves subepithelial vascular leakage. The difference is that in serous otitis media the vessel wall remains intact, allowing only plasma to escape, whereas in more abrupt, more forceful alterations in pressure, the vessel wall ruptures, allowing blood to spill out, first into the submucosal space and then into the middle ear cleft. The clinically described entity known as idiopathic hemotympanum (Ranger, 1949) falls into this category. This term is misleading and should not be used, since the other effusions described in this chapter are equally "idiopathic".

In cases of chronic serous otitis media it is, therefore, common to see a "gunmetal bluish" discoloration of the drumhead or indeed a purple drumhead resulting from old blood in the middle ear (Paparella and Lim, 1967). If blood predominates over serum, the term "hemotympanum" can be used, although "serous otitis media with bleeding" suffices. Very long-standing obstructions of ventilation of the middle ear may lead to development of cholesterol granuloma. Thus, when the eustachian tube of the monkey is obstructed, the early finding is serous effusion, whereas the late finding after 6 months is cholesterol granuloma (Main and Lim, 1970). The latter will be discussed in more detail later in this chapter.

Purulent Fluid

Here we refer to acute and chronic otitis media (sometimes termed purulent otitis media) in which pus bathes the middle ear. Purulent fluid is included in this classification for the sake of completeness, but it is not our primary intent to discuss purulent otitis media except as it relates to the aforementioned types of fluid. Laboratory examinations of this fluid will reveal many neutrophils and a moderate number of cellular remnants.

Clinical Features

History

In children, in whom middle ear effusions are commonly seen, the history is one of conductive hearing loss and, occasionally, of earaches. Often there are recurrent colds and sore throats. There may be attacks of acute otitis media, between which the ears never really return to normal. The air conduction hearing loss may be noted in a school audiogram or subjectively by observant parents. Children seldom volunteer any hearing difficulty. Middle ear effusions are more common in children with cleft palate, strong family histories of allergy, and symptoms of adenoid enlargement.Adults with middle ear fluid usually describe their symptoms quite specifically. They complain of a "plugged up" feeling in their ears and decreased hearing acuity. They often relate the onset of these to an upper respiratory infection. They may have autophony with their poor hearing. If the middle ear is only partially filled with fluid, such patients will note an improvement when they lie on the abdomen or sit far forward. This allows return of air to the region of the round window. In mild cases "popping and cracking" are noted, indicating altered eustachian tubal function. Dizziness is rarely a problem. Tinnitus due to movement of middle ear fluid may be present.

Some patients have seasonal hearing loss, with other characteristic symptoms indicative of inhalant allergies. In these patients a much more detailed history, including a pertinent family history, is indicated. As previously mentioned, patients who have received radiation therapy to the head often have recurring middle ear effusions.

Physical Examination

Because of the many etiologic possibilities, each patient with middle ear fluid merits a complete otorhinolaryngologic examination.

Tympanic Membrane

The changes noted on otoscopic examination of the drumhead can be classified into five categories according to frequency:

1. An opaque amber or yellow color of the drumhead replaces the normal translucent appearance. The normal cone of light in the anterior inferior quadrant is missing. With this there may be an increase in capillary vascularity of the drumhead. In mucoid and mucopurulent cases, the color may actually be more creamy than amber.

2. A chalky-appearing malleus handle, stressed by Hoople (1950), has been a valuable finding, in our experience.

3. Retraction of the tympanic mebrane resulting in apparent shortening of the malleus' handle is seen in most patients. In adhesive otitis media, this may result in the drumhead lying upon the incudostapedial joint or the promontory. The retraction may be localized to the pars flaccida, with the main part of the drumhead appearing amber in color.

4. Fluid levels or bubbles are usually seen in the serous form of middle ear effusion. The bubbles are not usually seen unless the patients has had positive pressure applied to a closed nasopharynx. Generally, this implies a favorable prognostic sign, because transudation and not exudation with associated chronic inflammation has probably occurred.

5. Blue tympanic membrane. The blue or purple drumhead is classically seen in cases of hemotympanum due to several causes (temporal bone fracture, leukemia, middle ear vascular tumors, and so forth). A bluish drumhead may also be seen in cases of refractory chronic "serous" otitis, aerotitis, or barotrauma, and so-called idiopathic hemotympanum (Paparella and Lim, 1967).

Adjunctive diagnostic procedures are used to confirm the diagnosis. Mobility of the drumhead is assessed with a pneumatic otoscope. Positive pressure is applied in the ear canal after obtaining an adequate seal. If there is air in the tympanum, it will be compressed and the drumhead will move with the application of positive pressure.

Politzerization in cases of retraction may result in a bulging tympanic membrane. If the eustachian tube is too obstructed or if the adhesions causing the retraction are too firm, the drumhead remains in the same position. If the diagnosis of middle ear fluid is not readily established, paracentesis should not be delayed.

Nasal and Nasopharyngeal Examination

To complete the physical examination, the nose must be viewed anteriorly and posteriorly. Posterior nasopharyngeal examination is adequately achieved with a small mirror in most cases, although some patients require topical anesthesia and retraction of the soft palate or a flexible nasopharyngoscope to complete the examination. Cases of unilateral middle ear effusion in adults are assumed to be due to nasopharyngeal tumor until proved otherwise.

Audiologic Examination

A conductive hearing loss can be confirmed quickly during examination by means of a negative Rinne test with a 512-cps tuning fork. Maximum sound will be perceived on the fluid ear in unilateral cases when performing a Weber test. The tympanogram typically shows flat type-B curve with absent stapedial reflex.

Pure-tone audiometry on conduction by air and bone is sufficient to follow the progress of most patients. If the diagnosis is doubtful, speech testing should be included. Characteristically, the audiogram shows a mild to moderate flat conductive loss in the range of 10 to 40 dB. If a greater conductive hearing loss is present, suspicion should be raised of the possibility of ossicular fixation or discontinuity. There may be a sensorineural high-tone hearing loss associated with the conductive loss, which is believed to be due to increased tension and stiffness of the round window membrane. This loss also disappears on evacuation of the fluid.

Radiologic Studies

X-rays of the mastoid are not routinely obtained in patients with middle ear effusions. Long-standing eustachian tubal dysfunction is usually associated with a poorly pneumatized mastoid. Middle ear fluid in a well-aerated mastoid appears as a clouding of the air-cell system. X-rays of the nasopharynx may be useful ni assessing adenoid hypertrophy in children in whom physical examination of this area is impossible.

Allergic Testing

Allergy can be an adjunctive cause of recurring middle ear effusions. There are definite exceptions in which allergy seems to play a primary etiologic role. We find that most patients are well controlled by the measures outlined in the next section. In patients with other signs of allergy (allergic rhinitis and sinusitis) and a strong family history of allergies, evaluation for allergy is essential to controlling recurring middle ear effusions.

Treatment

Medical Management

An attempt at conservative therapy should be made before surgical intervention. Cases of serous otitis presenting after an upper respiratory infection or acute otitis media often are cured by medicine alone. Mucoid otitis and chronic bloody effusions are controlled less often by medical therapy. Blood and serous fluid from barotrauma will usually clear with conservative management. Medical therapy includes antibiotics, antihistamines, decongestants, exercises to ventilate the eustachian tube, and allergic hyposensitization.

Antibiotics

Antibiotics are definitely indicated in patients with purulent otitis media. They are useful in those patients to whom inadequate therapy was given during the initial stages of the acute otitis media or in whom mucopurulent fluid or seropurulent fluid persists. Occasionally, in certain long-standing cases of mucoid otitis media (secretory otitis media), and especially when purulent otitis media becomes superimposed intermittently, we employ a trial of antibiotics for a period of 10 days. Indiscriminate use of antibiotics for all cases of serous or mucoid otitis media is not indicated. Microbiology of middle ear effusion and selection of appropriate antibiotics are dealt with in Chapters 28 and 30.

Antihistamines and Decongestants

Antihistamines and decongestants either alone or in combination are still among the most popular medications for the treatment of otitis media with effusion. The rationale for the use of these drugs is to reduce congestion of the eustachian tube as well as regional shrinkage of mucosa in the nose and sinuses. A short course of topical nasal decongestant (no longer than 5 days at a time) such as 1 per cent ephedrine or 0.25 per cent neosynephrine can be useful for the shrinkage of eustachian tubal mucosa. However, the efficacy of these drugs in the treatment of middle ear effusion has not been established and remains controversial (Cantekin et al, 1983). Side effects of these drugs such as hyperactivity or hypertension

(decongestants) and drowsiness (antihistamines) should be considered, and long-term indiscriminate use should be avoided.

Exercises to Ventilate the Eustachian Tube

In the absence of upper respiratory infection, whenever possible the patient should be instructed in the Valsalva maneuver. This may be advised several times a day to "unlock" the eustachian tube and to hasten clearance of the middle ear. A milder but similar passive effect can be achieved by having the patient chew gum, which encourages frequent swallowing of saliva, or squeeze the nose and swallow (Toynbee maneuver). Politzerization is a useful method in the office for ventilating the eustachian tube. Cannulating the eustachian tube to insufflate air may be traumatic and is unnecessary.

Allergic Hyposensitization

This is done when definite allergies are demonstrated by skin testing. Dietary elimination is advised when food allergy is demonstrated. Antihistamines are helpful when the diagnosis of allergy is not well established and less specific clinical control seems indicated.

Other Medical Treatment

The use of corticosteroids either in systemic form or as a topical nasal spray has been tried for the treatment of otitis media with effusion (Persico et al, 1978; Schwartz et al, 1980). From these studies it appears that in some children a short course of corticosteroid therapy may be of some help in treating chronic otitis media with effusion. Corticosteroids may work, probably because of anti-inflammatory action and inhibition of certain inflammatory mediators such as prostaglandins and leukotrienes.

Vaccinations against pneumococcal otitis media have been tried but the results have been disappointing so far (Karma et al, 1984). Mucolytic agents have been used, but their efficacy has not been established.

Surgical Management

Therapy includes control of regional inflammatory and obstructive foci when indicated. This may include intranasal or sinus procedures and adenoidectomy with or without tonsillectomy. Indications for adenoidectomy should be reviewed, with special caution in chidren with cleft palate. If deemed absolutely necessary in such children, the midline mass is left untouched and lymphoid tissue is removed from the lateral gutters. In bona fide cases of respiratory tract allergy, removal of the tonsils and adenoids may lead to an exacerbation of the allergic state. Special considerations are also necessary for the patient with a bleeding diathesis or other serious systemic illness. Diagnostic paracentesis, as mentioned previously, determines the presence of middle ear fluid; however, the type of middle ear fluid can be determined by examination, which is additionally diagnostic. This allows the institution of proper medical therapy (eg, antibiotics). If the fluid is serous, paracentesis alone with adjunctive medical measures is usually curative.

If the fluid is cloudy, mucoid, mucopurulent, or bloody, some form of myringotomy is usually indicated. In children, this is often done under general anesthesia in combination with adenoid surgery. In cooperative older children and in adults, this is a routine office procedure without anesthesia. (It has been said that the best anesthesia is a small sharp myringotomy knife.) In the occasional patient local anesthesia may be necessary. This can be accomplished by the topical iontophoresis technique or a single injection of 1 per cent lidocaine (Xylocaine), using a 27-gauge needle beneath the skin of the posterior bony canal wall, as in stapedial surgery. Another method is to make a tiny, pinpoint incision through the drumhead and to apply a small pledget of cotton soaked in 4 per cent cocaine, allowing the agent to anesthetize the adjacent undersurface of the drumhead, following which the myringotomy is lengthened as desired. Either the operating microscope or a hand otoscope with at least 6-power magnification should be used.

Only a limited myringotomy incision in the inferior aspect of the drumhead is necessary. It is well to stay away from the umbo, which is closer to the promontory. The "wide smile" myringotomy incision previously used for obstinate cases of acute otitis media with or without complications is not necessary for these conditions. Available today are tubes made of silicone rubber, Teflon, and plastic in many shapes and forms. We prefer using the silicone rubber tubes because of their inertness, malleability, and ease of introduction.

A small myringotomy incision is made in the inferior part of the drumhead just large enough to admit a tube with mild pressure. Great care is taken to have one flange of the tube on either side of the tympanic membrane. Especially with thick fluid, this may necessitate application of the "beer can" principle, in which a counterincision is made in the anterior superior quadrant to allow air to pass into the middle ear, facilitating fluid removal through both openings with a large (at least 5-gauge) suction tip. When the fluid is viscid, irrigation of the middle ear space helps to promote liquefaction and removal. In our experience, sterile water, saline, or corticosteroid solutions have been used with equal effectiveness.

The tube is usually left in place until it is spontaneously extruded, usually a minimum of several months. Occasionally the tubes will remain in place for a year or more, which is desirable in patients who have an established chronic problem.

In obstinate cases in which thick fluid quickly blocks and extrudes the tube, the following additional measures can be taken: (1) insertion of two tubes after complete evacuation of the middle ear, (2) insertion of a No 2 tube with a larger lumen and flange, or (3) insertion of a tube beneath the annulus and adjacent canal skin (Feuerstein, 1966). Occasionally, elevation of a tympanomeatal flap, as in stapedial surgery, is indicated in order to evacuate loculated and inspissated fluid thoroughly from the middle ear and eustachian tube and to inspect for other pathologic conditions.

If the prosthetic tube becomes plugged by dried secretions or epithelial overgrowth, fluid will recur. It has been suggested that a patent lumen is not necessary and that air enters the tympanum around the blocked tube. We have not found this to be true when plugged tubes were deliberately inserted in human subjects.

Rarely, untreated or persistent fluid in the middle ear with associated inflammatory and immunologic factors will lead to more serious pathologic states in the middle ear and mastoid.

This may include hypertrophic and fibrous changes, metaplasia, profuse glandular formation, cholesterol granuloma, and even cholesteatoma. In such cases, roentgenograms frequently reveal sclerotic, small mastoids, which are indicative of poor tubal function and development since early childhood.

Tympanostomy Tubes

Since Armstrong's (1954) pioneering contribution to the management of the middle ear effusions, innovative otologists have developed tympanostomy or ventilation tubes of many different materials, shapes, and sizes. The physiologic features they share in common are that they provide a substitute for eustachian tubal function and, indeed, that they function in the same order of importance as the eustachian tube - first, ventilation and second, drainage. Tympanostomy tubes have been used most widely for so-called "clean" middle ear effusions (in serous and mucoid otitis media) of chronic duration (longer than 2 or 3 months). Studies in our laboratories indicate that middle ear effusions that occupy the middle ear cleft for a prolonged period of time are not innocuous, but rather frequently lead to permanent sequelae and irreversible hearing losses, thus reinforcing the concept of the continuum of changes that may occur from one form of otitis media to another. There are those who view a hearing aid as a rehabilitative device that does nothing to interrupt the disease's pathogenesis or to prevent further long-term sequelae and complications.

Our studies, as well as others, demonstrate a higher incidence of positive cultures for bacteria in clean effusions from the middle ear without obvious clinical evidence of infection (Liu and Lim, 1975). This can amount to an incidence of 50 per cent or more in some cases. The role of active versus inactive bacteria in these cases is an interesting factor to consider. It is our opinion that tympanostomy, properly utilized, serves a most significant function, not only in helping to ameliorate the problem of otitis media, but in preventing further, more serious, forms of otitis media or sequelae. As is true of any tool, however, tympanostomy must be judiciously and properly used. The major indication for myringotomy and insertion of a tympanostomy tube is chronicity or failure of medical therapy.

A few principles regarding myringotomy are noteworthy. It is important never to do a myringotomy incision in the posterior-superior quadrant because of the regional location of important anatomic structures. The preferable place for a myringotomy and/or myringotomy tube is in the inferior quadrants. We prefer to place a tube in the anterior-inferior quadrant, which places it closer to the eustachian tube. It should not be located too close to the annulus, since it will extrude more readily.

Rarely there will be an exposed jugular bulb, and this should be noted as a bluish fullness in the lower aspect of the tympanic membrane. It should be recalled that the distance between the umbo and the promontory is approximately 2 millimeters, whereas it is double that toward the hypotympanum. There is still a place for paracentesis. This can be done with a needle or a very small incision to see if the fluid is present. In case of purulent, thick, tenacious material, a larger myringotomy incision and suction tip should be utilized.

There are many good myringotomy tubes (tympanostomy) made from a variety of materials. We usually use tympanostomy tubes of two sizes made of soft silicone rubber (Jurgens and Paparella, 1982), referred to as No 1 and No 2 tubes. The No 3 tube is never

used. The No 1 tube is used 95 per cent of the time. The No 1 tube has a lumen of 1.0 millimeters, and the No 2 has a lumen of 1.27 millimeters. The inner flange is thinner and larger in the No 2 tube, which allows it to remain in place for a much longer period of time. This tube is used for obstinate cases.

The tubes are never removed but rather are extruded in time by themselves. In our experience, the No 1 tube in most instances stays in place for an average of a year and the No 2 tube for an average of 2 years or longer; indeed, we have had one patient with chronic recurrent mucoid otitis media in whom the No 1 tube has remained in place for 7 years without any difficulty. When inserting the tube, it is important that the inner flange always be placed in the middle ear; if not, the tube will extrude readily. The tube extrude by epithelial migration, which occurs radially from the center of the tympanic membrane circumferentially toward the ear canal.

Clinical Applications

Purulent Otitis Media

Occasionally children, especially within the first 2 years of life, but up to 5 or more years of age, have frequent and repeated bouts of acute purulent otitis media. A frustrated mother will bring in her sick child to see the pediatrician or family physician, sometimes several times a month. We have seen children literally on constant antibiotic coverage. Between bouts of asbcess in the middle ear, the tympanic membrane usually will appear normal, or there may be evidence of fluid. Among the many multifactorial considerations of etiopathogenesis, eustachian tubal dysfunction plays an important role for these children. For these patients, we have found myringotomy and insertion of a tympanostomy tube to be of great adjunctive value.

The following procedure is used. The child is maintained on an appropriate antibiotic, such as ampicillin or erythromycin, for at least 10 days prior to the scheduled date of procedure. Occasionally a conservative midline adenoidectomy will accompany implacement of tympanostomy tubes if there is evidence of hypertrophy or focus of infection. A tube must not be inserted if there is active middle ear infection, and the procedure should be cancelled and rescheduled. We have seen children with as many as 10 to 20 bouts of purulent otitis media per year who, when treated as described above, have had few or no bouts of purulent otitis media in the subsequent year.

Serous Otitis Media

Effusions from the middle ear in cases of serous otitis media tend to be clear, watery transudates. Occasionally bubbles or a meniscus (fluid level) will be seen through a semitranslucent drumhead. Unlike those with mucoid otitis media, these cases of middle ear effusion have a better chance of resolving spontaneously or with conservative management. Therefore, tympanostomy tubes need not be used quite as readily as for patients with mucoid otitis media. However, tympanostomy tubes for cases of serous otitis media appear to have an improved chance of arresting the pathologic process. Serous otitis media can occur as a result of aerotitis or barotrauma, and blood can be combined with the plasma in the middle ear to provide a bluish appearance or hemotympanum. Occasionally, chronic cases of

refractory serous otitis media will result in deposition of blood, deposits of cholesterol, and foreign-body reaction, which may result in the pathologic conditions of cholesterol granuloma. Thus, serous otitis media can last for long periods of time and can also be associated with irreversible pathologic conditions in the tissue. If serous otitis media lasts 3 months or longer with appropriate medical therapy, then it is advisable that a tube be inserted.

Mucoid Otitis Media

In our experience, we see mucoid otitis media more commonly than serous otitis media in younger children. The fluid in mucoid, or secretory, otitis media appears as a thick, cloudy exudate. Even though clinical evidence of infection is not present, bacteria commonly are cultured. The fluid can be thick, tenacious, and inspissated and can result in so-called glue ear. Increased numbers of goblet cells and other secretory structures such as cysts and glands are seen throughout the mucoperiosteum of the middle ear cleft. In cases of mucoid otitis media, we routinely employ a trial of antibiotic therapy along with other medical aspects of management. If it is known that the patient has mucoid otitis media and will require the placement of a tympanostomy tube, he or she is placed on appropriate antibiotic therapy for at least 1 week prior to the scheduled date of the procedure. These cases are generally more reactive and advanced pathologically than cases of serous otitis media, with more frequent subsequent complications. Use of a No 2 tube may be considered, especially in obstinate cases of mucoid otitis media. As previously mentioned, the No 2 tube has a slightly larger lumen and a larger inner flange and remains in place a longer period of time.

Chronic Otitis Media

It is well known to otologic surgeons that eustachian tubal dysfunction is the chief nemesis in the surgical correction (tympanoplasty) of chronic otitis media. Clinical observation suggests that many patients had eustachian tubal dysfunction earlier in their lives. By the time they receive a tympanoplasty as an adult, the eustachian tube may appear to function appropriately. Unfortunately, tubal dysfunction usually continues to persist in some form or another. For this reason, and whenever possible, we use a tympanostomy tube in the anterior drumhead remnant while performing the various types of tympanoplasty.

The tube must not be inserted through the graft. Silastic sheeting is routinely used in the middle ear, and it is imperative that the fascial graft that extends to, and in some instances around, the tube does not obstruct its internal lumen. This appears to assist the healing process in postoperative tympanoplasty patients and seems to promote improved hearing results. The tympanostomy tube that we utilize has a small tag on it, so that, if the epithelium attempts to grow along the outer surface of the tube, it is still identifiable and the lumen can be maintained. We have attempted to use longer tubes under tympanomeatal flaps in tympanoplasty cases; they uniformly became obstructed and ultimately were removed.

Complications

The following complications may follow the use of tympanostomy tubes:

1. Infection or postoperative drainage.

2. Permanent perforation of the tympanic membrane.

3. Atrophic, healed, retracted replacement membrane at the site of the tube.

4. Cholesteatoma (keratoma) of the middle ear.

5. Sensorineural hearing loss.

Birck and Mravec (1976) reported an incidence of 15.1 per cent of draining ears treated successfully with aqueous Merthiolate irrigations. They also report an incidence of 14 perforations of the tympanic membrane, out of 736 children receiving tubes. Kokko and Palva (1976) described five patients out of 181 who had a dry perforation postoperatively. They described cholesteatoma in four ears; however, in their opinion, only one of those was related to the location of the tube at the posterior margin of the drum.

In our own experience, the most common complication, as was found also by Birck and Mravec, is postoperative infection or a draining ear. Although not tabulated, our incidence would appear to be somewhat lower. Ears that are draining following tympanostomy tube insertions are treated with frequent suction cleaning and antibiotic otic drops; intravenous antibiotics to combat infection with *Pseudomonas* should be given to patients with persistent otorrhea. When all medical therapy fails, mastoidectomy with exploratory tympanostomy should be considered.

We have had a few cases of nonhealed perforation and have often chosen to leave them alone, since such a perforation will function as well as a tympanostomy tube. If it becomes a problem, then a myringoplasty closure with a tympanostomy tube inserted in another part of the tympanic membrane may be considered.

We have seen no cases in which we could definitely attribute to the use of the tube the genesis of cholesteatoma, although this is described in the literature. We have seen four cases in which congenital cholesteatoma existed prior to the use of the tube. It could be that in certain patients a pre-existent congenital cholesteatoma without clinical manifestations, and not the tube, might be the cause for cholesteatoma. Needless to say, it is important that tubes be used only in patients who require them. They should be placed toward the center of the tympanic membrane and not near the edge, so as not to irritate the annulus, which might encourage ingrowth of the epithelium from the adjacent canal wall into the middle ear, thus leading to a cholesteatoma.

The possibility of a sensorineural hearing loss exists if treatment with otic drops for external otitis is undertaken while tympanostomy tubes are in place. A hearing loss, most severe in the high frequencies, and hair cell damage in the basal turn of the cochlea were observed in chinchillas receiving Corticosporin otic drops following placement of

tympanostomy tubes (Meyerhoff et al, 1983). When a dyed preparation of the drops was applied topically in the external auditory canal, it readily passed through a patent tympanostomy tube into the middle ear and onto the round window membrane. Sensorineural hearing loss in humans with tympanostomy tubes has been reported following treatment of external otitis with eardrops containing neomycin (Lind and Kristiansen, 1986).

Exploratory Tympanotomy

We have seen a number of patients in whom tympanostomy tubes have neen inserted, perhaps several times over a period of years, and following which a conductive loss persisted even though fluid was absent in the middle ear. We have seen and reported evidence that otitis media can exist in a subclinical state which we called "silent otitis media" (Paparella et al, 1980, 1984a). With proper treatment, the majority of cases with otitis media with effusion will resolve to a normal state. In some patients, otitis media with effusion persists in a chronic state and over time leads to intractable pathologic conditions in the tissue and sequelae. When this is suspected, exploratory tympanotomy is indicated (Paparella and Koutroupas, 1982).

For example, we have seen patients in whom the conductive loss (eg, 50 dB speech reception threshold) was greater than that which would be caused by fluid alone. In such instances it is likely that middle ear effusions caused osteolysis and ossicular disruption or ossicular fixation as a result of fixation of the tensor tympani tendon and other ligaments in the middle ear, or osteoneogenesis or tympanosclerosis. In any case, once a conductive loss of that magnitude is realized in the absence of fluid, exploratory tympanotomy is recommended along with the insertion of a tympanostomy tube. The contents of the middle ear are microscopically assessed. The lesion is identified and conservatively corrected as the individual situation warrants. In these cases, Silastic sheeting is generally placed in the middle ear; this helps to maintain an air space between the eustachian tube and the round window. In cases in which the round window membrane seems thin or deficient or if sensorineural hearing loss is associated with the otitis media, we graft the membrane with Gelfoam to thicken it and render it less permeable to inflammatory toxins. Occasionally, in these cases, if the drumhead is markedly atrophic, a fascial underplant procedure will be used to reinforce the tympanic membrane as well (Paparella and Jung, 1981).

Sequelae

Problems with effusions of the middle ear in children may lead to various developmental and behavioral sequelae. The complications that occur in the middle ear may also result in a continuum of late otologic sequelae, including atrophic drumhead, ossicular erosion, tympanosclerosis, chronic otitis media, cholesterol granuloma, and sensorineural hearing loss.

Developmental and Behavioral Sequelae

Recurrent middle ear effusion may result in developmental and behavioral sequelae. Delays in speech and language development (Downs, 1975; Holm and Kunze, 1969), deficits in auditory processing (Brandes and Ehinger, 1981; Welsh, 1983; Zinkus et al, 1980), learning disabilities (Kaplan et al, 1973), and reading disorders and educational difficulties (Brandes

and Ehinger, 1981; Zinkus et al, 1978) as well as behavioral problems (McGee et al, 1982) may occur. However, when hearing impairment is mild or of relatively brief duration, or both, evidence of adverse developmental consequences is lacking (Paradise, 1980).

Atrophic Drumhead

Long-standing effusions of the middle ear and negative pressure result in a "melting" of the membrane propria (fibrous layer) of the pars tensa and flaccida, leaving only the epithelial surface lining both sides of the tympanic membrane. The drumhead becomes very transparent and may become attached to the ossicles or promontory. This is a common finding, and unless associated symptoms exist, treatment is not indicated.

Ossicular Erosion

A small group of patients with chronic and persistent effusions in the middle ear require periodic (perhaps once every year or two) insertion of a ventilation tube. However, when the tube is patent and in its proper position, and in the absence of fluid in the middle ear, a conductive loss of 35 dB or more occasionally will be found to persist. In such cases, erosion of the lenticular process of the incus should be suspected and may be confirmed by exploratory tympanotomy. In some instances a fibrous union replaces the missing bone. This results from infection and enzymatic osteolysis or ischemia due to interrupted blood supply. Interposing a sculptured bone-wedge to replace the lenticular process may re-estabish ossicular continuity and hearing.

Tympanosclerosis

Hyalinized collagen, or tympanosclerosis, typically is seen in the pars tensa in patients with a previous history of effusions or infections in the middle ear (Hussl and Lim, 1984). Such tissue has an uncertain pathogenesis but can exist throughout the middle ear, enveloping ossicles and especially the stapes, thereby causing fixation and conductive deafness, and involving the promontory and the eustachian tube. Management of extensive cases can be difficult. It is known that collagen exists in the fibrous layer of the normal pars tensa and in the wide subepithelial space, which has undergone fibrosis in cases of chronic serous otitis media. Irritative fators such as infection and recurrent fluid and increased vascularity and transudation, as well as exudation as seen in the "glue ear", may help to precipitate the formation of such sclerotic changes.

Chemical changes also probably play a role. Occasionally there may be a mild amount of calcification, although frequently this is absent. Immature tympanosclerosis appears soft in consistency, whereas mature tympanosclerosis hardens and approaches the consistency of cartilage. Typically, when tympanosclerosis envelops the stapes it does so in layers, as in an onion skin, and these flakes can be peeled off with a fine oval-window hook to mobilize the stapes. Extensive tympanosclerosis, as is usually seen on the promontory, may invade the bone.

Chronic Otitis Media and Cholesteatoma

The same fundamental factors characterized by eustachian tubal dysfunction may predispose to the development of chronic otitis media and even secondary cholesteatoma. Thus, early and adequate treatment of serous otitis media may preclude more serious chronic inflammatory disease of the middle ear and mastoid.

Cholesterol Granuloma and Glandular Formation

Patients with chronic refractory serous otitis media and with cholesterol granuloma requiring mastoid surgery have been studied (Paparella and Lim, 1967). The pathologic findings in both of these conditions were found to be essentially the same. Cholesterol granuloma was found in both; in addition, glandlike structures having the appearance of cysts or ducts were seen in both groups. It is probable that such structures develop from a trapping of metaplastic epithelium beneath the surface of a hyperplastic mucosal response in the middle ear and mastoid and help to lead to an intractable state. It was concluded that these problems do not represent specific diseases but rather different phases of the same condition.

If the eustachian tube is mechanically obstructed in an animal, serous otitis media develops in 20 to 24 hours, followed by chronic serous otitis media in weeks, and cholesterol granuloma in the middle ear and mastoid after 3 to 6 months. If blood is placed in the middle ear cavity of experimental animals after mechanical obstruction of the eustachian tube, cholesterol granuloma is observed consistently as early as 3 months (Goycoolea et al, 1980a). A similar course of events may occur in humans. The diagnosis applied to this disorder in some instances has been "idiopathic hemotympanum". This disorder may result in chronic serous otitis media in which vessels rupture and bleeding develops as well as transudation of serum. Because the cilia cannot evacuate the contents of the middle ear, stagnation and breakdown of blood occur, leading to formation of granulation tissue with deposition of cholesterol. In this case, irreversible pathologic conditions or cholesterol granuloma develops, obviating any possibility of more conservative treatment.

After extended attempts at conservative therapy, including tympanotomy for purposes of exploration and evacuation, it may be necessary in selected cases to consider mastoid surgery. If the temporal bone is reasonably pneumatized, a complete simple mastoidectomy may be done, whereas if the mastoid is markedly sclerotic, a modified radical mastoidectomy is preferable. Granulation tissue that is brown, appearing like axle grease, will be found, particularly in such regions as the sinus tympani, facial recess, aditus ad antrum, and retrofascial cells. The procedure should be accompanied by a tubal placement through the drumhead to continue to assist ventilation of the middle ear.

Sensorineural Hearing Loss

High-frequency sensorineural hearing loss can occur in patients with otitis media with effusion. This type of hearing loss has been reported in patients with chronic otitis media with effusions of the serous (Avil, 1982; Münker, 1977), mucoid (Arnold et al, 1977; Münker, 1977; Palva and Johnsson, 1984), and purulent types (Paparella et al, 1970a; Paparella et al, 1984b). The incidence of sensorineural hearing loss in patients diagnosed as having otitis media with effusion has been reported to be as high as 35.8 per cent in children and 87.9 per

cent in adults (Draf and Schultz, 1980). Thresholds for bone-conducted hearing have been found to decrease with the severity and duration of the disease (English et al, 1973).

Paparella et al (1970a) suggested that it is the passage of toxic substances of infectious agents from the middle to the inner ear via the round window membrane that causes damage to the cochlea and results in high-frequency hearing loss. Evidences presented for this concept are the various types of substances that have been shown to permeate the normal round window membrane, including proteins (Goycoolea et al, 1980b; Tanaka and Motomura, 1981), toxins (Goycoolea et al, 1980c; Schachern et al, 1981), antibiotics (Harada et al, 1986; Okuno and Nomura, 1984), and local inhibitors (Rybak et al, 1984). Morphologic evidence, other than the presence of inflammatory cells in the scala tympani (Paparella et al, 1972), concerning the mechanism for the sensorineural loss has not been found. Walby and colleagues (1983) have suggested that the high bone-conduction thresholds frequently seen in patients with chronic otitis media may be due to changes in the mechanics of sound transmission.

In patients with a normal round window membrane and in children with acute otitis media, the round window membrane is thin and potentially permeable to toxic substances of middle ear effusions. The round window has been shown to thicken, due to fibrosis, in human temporal bones with chronic otitis media (Sahni et al, 1987). Round window membranes from cats with experimentally induced chronic otitis media were also thicker than membranes from normal cats or from cats with acute otitis media and were less permeable to a tracer substance (Schachern et al, 1987b). Furthermore, grafting of normal feline round window membranes, with Gelfoam, also resulted in a thickened round window membrane that was less permeable to a tracer (Schachern et al, 1987a).

During surgical procedures on the middle ear, such as exploratory tympanotomy, stapedectomy, and tympanoplasty for conductive losses, especially when these are accompanied by a sensorineural hearing loss, we carefully inspect the round window niche and membrane. In those cases in which the membrane appears thin or deficient, or if sensorineural hearing loss appears to exist as a concomitant of otitis media, our policy is to pack Gelfoam into the round window niche to encourage an increase in the thickness of the membrane and a decrease in the membrane's permeability.