Recent Advances in the Pathogenesis, Diagnosis, and Management of Otitis Media

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Otitis media is the most frequent diagnosis made by physicians who care for children. Acute otitis media is usually suppurative or purulent, but serous middle ear effusions may also have an acute onset. Chronic otitis media with effusion has many synonyms, including such terms as secretory, serous, nonsuppurative, and "glue ear". A chronic effusion may be serous, mucoid, or even purulent. In some instances, the eardrum may be retracted or collapsed without a middle ear effusion, which is termed *atelectasis* of the tympanic membrane, and is the result of persistent or intermittent negative middle ear pressure. It is often difficult to determine from the history and visual inspection of the tympanic membrane the precise type of otitis media present since in most patients, especially infants and young children, the disease is a continuum of the different stages. Some patients may have recurrent acute attacks without an apparent effusion in-between, whereas others may have only chronic otitis media with effusion, and still others may have recurrent acute episodes superimposed on a persistent middle ear effusion. Atelectasis of the tympanic membrane may represent the only pathology in some patients but in others the condiction can be present between episodes of otitis media with effusion. Chronic otitis media with perforation and otorrhea is one of the sequelae of acute or chronic otitis media with effusion.

Epidemiology

Infants and young children are at highest risk for the acquisition of otitis media, with the peak prevalence rate occurring between 6 and 36 months, and a lesser peak between 4 and 7 years. One study of 2565 children followed for the first three years of life found that only 29 per cent of infants failed to develop at least one attack of otitis media, whereas about one-third had three or more episodes. In addition, the study showed that after the first episode, 40 per cent of children had a middle ear effusion that persisted for four weeks and 10 per cent had an effusion that was still present at three months. Infants in whom otitis media with effusion develops in the first years of life have an increased risk of recurrent acute or chronic middle ear effusions. The overall prevalence of the disease in children has been estimated to be between 15 and 20 per cent. However, the incidence and prevalence of the disease tend to decrease as a function of age after the age of 6 years. The incidence is higher in males, lower socioeconomic groups, Alaskan natives (Eskimos), American Indians, children with cleft palate and other craniofacial anomalies, and is higher in whites than in blacks. The incidence is also higher in winder and early spring.

Physiology and Pathophysiology of the Eustachian Tube Related to Pathogenesis of Otitis Media

The pathogenesis of otitis media with effusion appears to be related to abnormal function of the eustachian tube. Investigation into the exact nature of this dysfunction requires an understanding of the system constituted by the palate, nasal cavity, nasopharynx, eustachian tube, middle ear, and mastoid air cells. Within this system, the eustachian tube has at least three physiologic functions with respect to the middle ear: *equilibration of air*

pressure (ventilation) in the middle ear with atmospheric pressure, *protection* from nasopharyngeal sound pressure and secretions, and *clearance* into the nasopharynx of secretions produced within the middle ear.

The protective and clearance functions of the eustachian tube have been assessed by radiographic techniques. Radiopaque material was instilled through the nose of the children in order to assess the protective function by observing the retrograde flow of the medium from the nasopharynx into the eustachian tube. Clearance function was assessed following instillation or insufflation of radiopaque material into the middle ear and observing the flow of the medium down the eustachian tube into the nasopharynx.

The findings of these radiographic studies can be understood if a model of the system is constructed. The eustachian tube, middle ear, and mastoid can be likened to a flask with a long, narrow neck, the mouth of the flask representing the nasopharyngeal end, the narrow neck the isthmus of the eustachian tube, and the bulbous portion the middle ear and mastoid air chamber. When a small amount of liquid is instilled into the mouth of the flask, liquid flow will stop somewhere in the narrow neck, owing to capillarity and to the cushion of air pressure that develops in the chamber of the flask. This basic geometric design is considered to be critical for the protective function of the eustachian tube and middle ear system. Reflux of the liquid into the vessel occurs if a hole is made in the bulbous portion of the flask, since the pressure in the bottom of the flask remains unchanged. This is analogous to the condition in which a perforation of the tympanic membrane or the presence of a tympanostomy tube could allow reflux of nasopharyngeal secretions as a result of loss of the middle ear-mastoid air cushion. The effect of the application of a negative pressure to the bottom of the flask: the liquid is aspirated into the vessel. In the clinical situation represented by the model, high negative middle ear air pressure could lead to the aspiration of nasopharyngeal secretions into the middle ear. The effect of applied positive pressure to the mouth of the flask is also shown: the liquid is insufflated into the vessel. Noseblowing, crying, or closed-nose swallowing could create a high positive nasopharyngeal pressure and result in a similar condition in the human system.

However, one of the major differences between a flask with a rigid neck and a biological tube such as the eustachian tube is that the isthmus (neck) of the human tube is compliant. Application of positive pressure at the mouth of a flask with a compliant neck would distent the neck, enhancing fluid flow into the vessel. The effect of applied negative pressure in a flask with a compliant neck is shown. Liquid flow through the neck would not occur until a negative pressure was slowly applied to the bottom of the flask. In this case, fluid flow would occur even if the neck were collapsed, but if the negative pressure were applied suddenly, temporary locking of the compliant neck would prevent flow of the liquid. Therefore, the speed of application of the negative pressure, as well as the compliance in such a system, would appear to be critical factors in the results obtained. Clinically, aspiration of gas into the middle ear would be possible, since negative middle ear pressure would develop slowly, as the gas was absorbed by the middle ear mucous membrane. However, sudden application of negative middle ear pressure, such as would occur with rapid alterations in atmospheric pressure (as in an airplane during descent, when diving, or when attempting to test the ventilatory function of the eustachian tube), could lock the tube, thus preventing the flow of air. Likewise, sudden application of negative pressure at the nasopharyngeal portion of a highly compliant tube could also result in locking. Negative pressure in the middle ear would be less likely to functionally obstruct the eustachian tube than negative pressure at the nasopharyngeal end, since the tympanic portion of the tube is a bony funnel. This geometric difference is probably important in the mechanics of passive tubal opening, when a gradient (negative middle ear pressure) is required to open the tube or assist the active opening mechanism.

Certain aspects of the clearance function of fluid from the middle ear into the nasopharynx can be demonstrated by inverting the flask of the model. The liquid trapped in the bulbous portion of the flask does not flow out of the vessel as a result of the relative negative pressure that develops inside the chamber. However, if a hole is made in the vessel, the liquid drains out of the flask, since the suction is broken. Clinically, these conditions occur in cases of middle ear effusion in which pressure is relieved by myringotomy. Insufflation of air into the flask achieves a release of pressure, which may explain the frequent success of politzerization or the Valsalva maneuver in clearing middle ear effusion.

The foregoing description of fluid flow through a flask only presents some of the mechanical aspects of the physiology of the human middle ear system. Other factors that probably affect the flow of liquid and air through the middle ear in the physiologic state would include: (1) the mucociliary transport system of the eustachian tube and middle ear, (2) contraction of the tensor tympani muscle and tympanic membrane movement, (3) active tubal opening mechanisms, and (4) surface tension factors.

The function of the eustachian tube to equilibrate air pressure can be assessed by a modification of the manometric technique. A perforation of the tympanic membrane or a tympanostomy tube must be present, so that middle ear pressure can be directly inflated or deflated employing a pump-manometer system. Figure is a simplified explanation of the combined passive and active function test, when positive pressure is applied tothe middle ear - inflation. This test is similar to ascending in an airplane until the eustachian tube passively opens. The test consists of the application of enough positive pressure into the middle ear to force open the eustachian tube. The pressure remaining in the middle ear after passive opening and closing is termed the closing pressure. Further equilibration of pressure is by swallowing - active function - which is the result of contraction of the tensor veli palatini muscle. When the muscle contracts, the lumen of the eustachian tube is opened and air flows down the tube. The pressure can be monitored on a strip chart recorder; the pressure remaining in the middle ear after passive and active function is termed the residual positive pressure.

Figure shows the deflation phase of the study, which is similar to descent in an airplane. Low negative pressure is applied to the middle ear, which is then equilibrated by active function. The pressure remaining in the middle ear after swallowing is termed the residual negative pressure. In certain instances, active function to applied low positive pressure is also assessed. Figure shows that the test is similar to ascent in an airplane, but to an altitude lower than a pressure that would force open the eustachian tube. The patient is asked to swallow in an attempt to equilibrate the pressure by active function.

Figure shows the symbols employed and examples of results obtained in ventilation studies. Example A describes a typical study of a patient with normal function. Following passive opening and closing of the eustachian tube during the inflation phase of the study, the

patient is able to completely equilibrate the remaining positive pressure. Active swallowing also completely equilibrates applied negative pressure - deflation. Example B describes a typical study of a child who had had otitis media with effusion. The eustachian tube passively opens and closes following inflation, but subsequent swallowing fails to equilibrate the residual positive pressure. In the deflation phase of the study, the child is unable to equilibrate negative pressure. Inflation to a pressure below the opening pressure but above the closing pressure cannot be equilibrated by the active swallowing function.

From these studies of fluid flow through a vessel and studies in children and adults, the following physiology of the eustachian tube and stages in the pathogenesis of otitis media with effusion have been postulated. Figure shows that the normal eustachian tube is functionally obstructed or collapsed at rest, with probably a slight negative pressure existing in the middle ear. In ideal tubal function, intermittent active opening of the eustachian tube maintains near-ambient pressures in the middle ear.

It is suspected that, in cases in which active function is inefficient in opening the eustachian tube, functional collapse of the tube persists. The interval between openings would then depend on the establishment of a pressure gradient between the middle ear cavity and the nasopharynx, which passively assists tubal function. Physiologically, this gradient is achieved by the absorption of middle ear gas, which results in progressive negative middle ear pressure. This type of middle ear pressure regulation appears to be quite common in children, as moderate-to-high negative middle ear pressures have been identified by tympanometry in many who are apparently normal.

There appear to be two types of eustachian tube obstruction which would result in an otitis media with effusion: functional and mechanical. Functional obstruction could result from persistent collapse of the eustachian tube owing to increased tubal compliance, or an inadequate active opening mechanism, or both. Functional eustachian tube obstruction is common in infants and younger children, since the amount and stiffness of the cartilage support of the eustachian tube are less than in older children and adults, and since there appear to be marked age differences in the craniofacial base which render the tensor veli palatini muscle less efficient prior to puberty. The other major type of dysfunction of the eustachian tube is abnormal patency. In its extreme form the tube is open even at rest, ie, patulous. Lesser degrees of abnormal patency would result in a semipatulous eustachian tube which would be closed at rest but have low resistance in comparison to the normal tube. Increased patency of the tube may be caused by abnormal tube geometry or to a decrease in the extramural pressure (such as occurs with weight loss, or possibly due to mural or intraluminal factors).

Figure depicts the pathogenesis of otitis media with effusion resulting from eustachian tube dysfunction. Functional obstruction may result in persistent high negative middle ear pressure, and when associated with collapse of the tympanic membrane has been termed *atelectasis*. If a large bolus of air enters the middle ear from the nasopharynx when there is high negative middle ear pressure, nasopharyngeal secretions could be aspirated into the middle ear and result in an acute bacterial otitis media with effusion.

If middle ear pressure equilibration does not occur, persistent functional eustachian tube obstruction could result in a sterile otitis media with effusion. The occurrence of an otitis

media with effsion at this stage might depend on the degree and duration of the negative pressure, as well as middle ear hypoxia or hypercarbia. Since tubal opening could be possible in a middle ear with an effusion, aspiration of nasopharyngeal secretions might occur, thus creating the clinical condition in which persistent otitis media with effusion and recurrent acute bacterial otitis media with effusion occur together. All infants with unrepaired palatal clefts and many children with repaired palates have otitis media with effusion as a result of functional obstruction of the eustachian tube.

Intrinsic mechanical obstruction of the eustachian tube may be caused by inflammation. Most ears at risk for the development of atelectasis or otitis media with effusion when inflammation is present probably have a significant degree of functional obstruction. An upper respiratory tract infection in such children has been shown to significantly decrease eustachian tube function. Periods of upper respiratory tract infection may then result in either atelectasis of the middle ear, bacterial otitis media with effusion, or a sterile otitis media with effusion owing to swelling of the eustachian tube lumen. The mechanisms are similar to those described for functional obstruction of the eustachian tube. Allergy a a cause of intrinsic mechanical obstruction of the eustachian tube has not been demonstrated.

Extrinsic mechanical obstruction of the eustachian tube may be the result of extrinsic compression by nasopharyngeal tumors or adenoids. Partial obstruction of the eustachian tube may result only in atelectasis of the middle ear or a bacterial otitis media with effusion, but more severe obstruction could result in a sterile otitis media with effusion.

Abnormal Patency of the Eustachian Tube

A patulous eustachian tube usually permits air to flow readily from the nasopharynx into the middle ear, which thus remains well ventilated; but also, unwanted nasopharyngeal secretions can traverse the tube and result in "reflux otitis media". A semipatulous eustachian tube may be obstructed functionally as the result of increased tubal compliance and the middle ear may even have negative pressure, or an effusion, or both. Since the tubal walls are abnormally distensible, nasopharyngeal secretions may readily be insufflated into the middle ear even with modest positive nasopharyngeal pressures, for example, as a result of noseblowing, sneezing, crying, or closed-nose swallowing. If active tubal opening (tensor veli palatini contraction) occurs, resulting in an abnormally patent tube, reflux or insufflation of nasopharyngeal secretions would also be likely. If the eustachian tube has lower resistance than normal but remains functionally obstructed even during attempts at active tubal opening, it is conceivable that nasopharyngeal secretions, rather than air, would more readily enter the middle ear. Native Americans (American Indians) have been shown to have tubal resistances that are lower than those of the average Caucasian. They seem to have an increased incidence of reflux of nasopharyngeal secretion into the middle ear, and frequently suffer from recurrent acute otitis media, which is often associated with perforation and discharge. However, American Indians have a low incidence of cholesteatoma. This type of eustachian tube function and middle ear disease is different from those in individuals who have a cleft palate.

Nasal Obstruction

Nasal obstruction may also be involved in the pathogenesis of otitis media with effusion. Swallowing when the nose is obstructed (due to inflammation or obstructed adenoids) results in an initial positive nasopharyngeal air pressure, followed by a negative pressure phase. When the tube is pliant, positive nasopharyngeal pressure might insufflate infected secretions into the middle ear, especially when the middle ear has a high negative middle ear pressure; or, with negative nasopharyngeal pressure, such a tube could be prevented from opening and be further obstructed functionally (the "Toynbee phenomenon").

Diagnostic Methods

Next to the patient's medical history, the most important diagnostic tool is the otoscopic examination. However, before adequate visualization of the external canal and tympanic membrane can be obtained, obstructing cerumen must be removed from the canal. This can be accomplished with either an otoscope with a surgical head and a wire loop or a blunt cerumen curette, or by gently irrigating the canal with warm water. In the newborn infant the external canal is filled with vernix caseosa which disappears shortly after birth.

Proper assessment of the tympanic membrane and its mobility is accomplished by the use of the *pneumatic otoscope* in which the diagnostic head has an adequate seal. Physical findings noted during inspection of the tympanic membrane should include position, color, degree of translucency, and mobility. Evaluation of the light reflex is of limited value. The normal tympanic membrane should be in the neutral position, in contrast to a drum that is bulging. The latter condition may be caused by increased middle ear air pressure, or an effusion within the middle ear, or both; the malleus handle and short process are obscured by the bulging drum. Retraction of the tympanic membrane usually indicates the presence of middle ear negative pressure; however, the tympanic membrane may even be severely retracted (presumably because of previous high negative middle ear pressure or inflammation with subsequent fixation of the ossicles and ligaments) without the presence of high negative middle ear pressure. When retraction of the tympanic membrane is present, the short process of the malleus is prominent and the long process is foreshortened. The normal tympanic membrane has a ground-glass appearance; a blue or yellow color usually indicates a middle ear effusion. A red tympanic membrane alone may not be indicative of pathology, since the blood vessels of the drum head may be engorged as the result of crying, sneezing, or blowing the nose. The distinction between translucency and opacification is critical in identification of middle ear disease. The normal tympanic membrane should be translucent, the observer being able to look through the drum and visualize the middle ear landmarks: incudostapedial joint, promontory, round window niche, and frequently the chorda tympani nerve. If a middle ear effusion is present medial to a translucent drum, an air-fluid level or bubbles of air mixed with the fluid may be visible. Inability to visualize the middle ear structures indicates opacification of the drum, which is usually the result of thickening of the tympanic membrane, or a middle ear effusion, or both.

Abnormal middle ear pressure is reflected in the pattern of tympanic membrane mobility when first positive and then negative pressure is applied to the external canal. Pressure is applied by first obtaining an adequate seal between the external auditory canal and the ear speculum, and then by applying slight pressure on the rubber bulb (positive pressure) followed by release of the bulb (negative pressure). The presence of a liquid, or abnormal pressure (positive or negative), or both, within the middle ear can markedly dampen the movement of the eardrum. When the middle ear has a high negative pressure, the tympanic membrane will not move to applied positive pressure but will move outward with applied negative pressure if the applied pressure exceeds the middle ear negative pressure. Conversely, when high middle ear positive pressure is present, the drum will not move to applied negative pressure but will move to applied positive pressure as long as there is air within the middle ear. The tympanic membrane will not move to applied positive or negative pressure when the middle ear-mastoid cavity is completely filled with a liquid. When available, the otomicroscope may be helpful in the evaluation of the difficult-to-assess tympanic membrane.

The most significant advance in the identification of middle ear disease is the use of the *electroacoustic impedance bridge* with which a *tympanogram* can be obtained. To perform tympanometry, a small probe is inserted into the external auditory canal. A tone of fixed characteristics is presented via the probe, and the compliance (mobility) of the tympanic membrane is measured electronically while the external canal pressure is artificially varied. The patterns obtained are highly diagnostic in distinguishing a normal ear from one in which a middle ear effusion is present. It is especially beneficial for the following: as an aid in diagnosis when otoscopy is equivocal or difficult to perform; for objective determination of the middle ear pressure; as an aid in teaching otoscopy; and for screening for ear disease. Tympanometry also may be helpful in identifying middle ear disease other than effusion, such as disarticulation or fixation of the ossicular chain.

However, tympanometry does not assess hearing; *audiometry* measures this function. Usually, in patients over the age of two years behavioral audiometry, which is a subjective assessment of hearing, is possible. In the young infant or in children who are difficult to test, objective audiometry is necessary (auditory brainstem response audiometry or the acoustic reflex obtained with an electroacoustic impedance bridge).

Aspiration of the middle ear is the most definitive method of verifying the presence and type of a middle ear effusion. Diagnostic tympanocentesis may be performed by inserting, through the inferior portion of the tympanic membrane, an 18-gauge spinal needle attached to a syringe. Culturing the ear canal and cleansing of the canal with alcohol should precede the procedure; the canal culture is helpful in determining whether organisms cultured are contaminants from the canal or pathogens from the middle ear. *Microbiologic studies* of the aspirate constitute an invaluable diagnostic aid to the clinician. Indications for tympanocentesis include the following:

- 1. Otitis media in children who are critically ill.
- 2. Unsatisfactory response to antimicrobial therapy.
- 3. Onset of otitis media in a child who is receiving an antimicrobial agent.
- 4. Presence of suppurative complications.

5. Otitis media in the neonate, the very young infant, or in the immunologically deficient patient, in each of whom an unusual organism may be suspected.

When therapeutic drainage is required, a myringotomy should be performed and the incision should be large enough to allow for adequately drainage and aeration of the middle ear. Myringotomy should always be performed when a suppurative complication is present and may also be helpful following tympanocentesis for the indications listed above. In addition, a myringotomy frequently will be beneficial for a child who has unusually severe otalgia, either when initially examined or at any time during the course of the disease.

Roentgenographic assessment of the ear and temporal bone is frequently helpful in diagnosis and management; polytomography is more helpful in certain instances than conventional radiographs.

When the tympanic membrane is not intact (as a result of perforation or insertion of a tympanostomy tube), *assessment of the ventilatory function of the eustachian tube* by pressure-flow studies may be an additional aid in the diagnostic workup of a child with middle ear disease.

Acute Otitis Media With Effusion

In the classic description of this condition, a child who has an upper respiratory infection for several days suddenly develops otalgia, fever, and hearing loss. Examination with the pneumatic otoscope reveals a hyperemic, opaque, bulging tympanic membrane that has poor mobility. Purulent otorrhea may be present. However, earache and fever are not invariable concomitants of infection. Because of the variability of symptoms, an otoscopic examination should always be included in the evaluation of infants and children; those who have diminished or absent mobility and opacification of the tympanic membrane should be suspected of having a bacterial otitis media with effusion. Middle ear infection must be ruled out in any child with a "fever of undetermined origin". When the diagnosis of acute otitis media with effusion is in doubt, or when determination of the causative agent is desirable, aspiration of the middle ear should be performed.

Rational therapy for acute otitis media with effusion depends upon knowledge of the bacterial cause of the disease. The bacteria that have been cultured from middle ear effusions in children with acute otitis media have been shown to be the same found in the nasopharynx. *Streptococcus pneumoniae* has been cultured from approximately 40 per cent of the effusions and is the most common causative agent in all age groups. *Haemophilus influenzae* causes about 20 per cent of cases. This proportion declines with increasing age but *H. influenzae* is still significant in *all* age groups. Recently, there has been an increasing percentage of *H. influenzae* strains, 15 to 30 per cent, that have been beta-lactamase producing and, therefore, ampicillin-resistant. Group A beta-hemolytic streptococcus and *Staphylococcus aureus* account for 7 and 2 per cent, respectively. In about 25 per cent of effusions, no bacteria are cultured. In neonates, approximately 20 per cent of effusions may contain gram-negative enteric bacilli.

In patients with classic signs and symptoms of acute otitis media, antimicrobial therapy is the treatment of choice. Since the clinician rarely is certain of the causative organism before starting therapy for otitis media, ampicillin is the single most useful drug, and will usually be effective against the four most commonly encountered bacteria. Oral ampicillin, 50 to 100 mg/kg/24 hr, in four divided doses for 10 to 14 days, is recommended. Amoxicillin,

20 to 40 mg/kg/24 hr, is probably equally effective and can be given in three divided doses. If the patient is allergic to the penicillins, then a combination of oral erythromycin, 50 mg/kg/24 hr, and triple sulfonamides, 100 mg/kg/24 hr (or sulfisoxazole, 150 mg/kg/24 hr), in four divided doses, is a suitable alternative. The combination of trimethoprim and sulfamethoxazole, 8 to 40 mg/kg/24 hr in two divided doses, also can initially be given to penicillin-sensitive individuals, but its effectiveness in the treatment of acute otitis media caused by *Streptococcus pyogenes* is uncertain. A new cephalosporin, cefaclor, 40 mg/kg/24 hr, in three divided doses, appears to be a promising new antimicrobial agent for otitis media since it is effective against the common pathogens causing acute otitis media.

Additional supportive therapy, including analgesics, antipyretics, and local heat, will usually be helpful. In some instances, meperidine hydrochloride may also be required for sedation. The efficacy of antihistamines and decongestants in the treatment of acute otitis media has not been proven.

If the patient continues to have appreciable pain or persistent fever, or both, after 24 to 48 hours, tympanocentesis/myringotomy should be performed as a diagnostic and therapeutic procedure. At this stage the presence of an effusion alone does not constitute a clinical failure. In patients with unusually severe earache, myringotomy may be performed initially in order to provide immediate relief.

When an unusual organism is cultured from a middle ear aspirate, sensitivity testing will help in the choice of antimicrobial agents. An example of this situation is an acute otitis media caused by *H. influenzae* that is resistant to ampicillin. When this occurs or when the patient fails to improve clinically after the initial treatment with ampicillin or amoxicillin and a tympanocentesis/myringotomy is not performed, the initial antimicrobial should be changed since an ampicillin-resistant *H. influenzae* should be suspected. Erythromycin in combination with a sulfonamide, or trimethoprim-sulfamethoxazole, or cefaclor are appropriate choices at present.

All patients should be reevaluated approximately two weeks after the institution of treatment. At this time some patients will have had complete resolution of the middle ear effusion but in others complete clearing of the effusion may take six weeks or longer. Within two to three months the tympanic membrane should be entirely normal. If complete resolution has occurred and the episode represents the only known attack, the patient may be discharged. However, periodic follow-up is indicated for patients who have had recurrent episodes.

Recurrent Acute Otitis Media

It is not uncommon for children, especially infants, to have recurrent bouts of acute otitis media. Some children have an acute episode with almost every respiratory tract infection, have more or less dramatic symptoms, respond well to therapy, and improve with advancing age. Others are more difficult, in that they have persistent middle ear effusion and suffer recurrent episodes of acute otitis media with effusion superimposed on the chronic disorder. The child with recurrent acute otitis media with effusion that completely clears between episodes may be managed as previously outlined. However, if the bouts are frequent and close together, further treatment, similar to that described for patients with chronic otitis media with effusion, is indicated. In many of these children, the underlying cause is not

evident but myringotomy with insertion of middle ear ventilation tubes is frequently helpful. Prophylactic antimicrobials (a daily dose of ampicillin or sulfonamides) have been advocated as an alternative to myringotomy and ventilating tubes in children with recurrent acute otitis media with effusion who are free of effusion between attacks. The efficacy of myringotomy with tympanostomy tube insertion and of chemoprophylaxis is as poorly established as is the usefulness of other forms of prevention, such as hyposensitization and adenoidectomy.

Chronic Otitis Media With Effusion

Chronic middle ear effusions may be thin (serous), thick (mucoid), or purulent. Pneumatic otoscopy will frequently reveal either a retracted or convex tympanic membrane. The membrane is usually opaque, but when it is translucent an air-fluid level or air bubbles may be seen and an amber or sometimes bluish fluid may be apparent in the middle ear. The mobility of the eardrum is almost always impaired. Occasionally, even when the middle ear is free of effusion, the tympanic membrane will be retracted and its mobility impaired. This finding usually indicates the presence of negative middle ear air pressure, which, when extreme, is termed "atelectasis of the tympanic membrane"; it may be accompanied by the same symptoms usually associated with otitis media with effusion. In both conditions, auditory acuity is usually decreased, and although systemic symptoms are usually absent, there may be behavioural disturbances owing to the child's inability to communicate adequately. A feeling of fullness in the ear, tinnitus, and even vertigo may be present. Audiometry may be helpful in establishing the diagnosis but is not a reliable indicator, because some patients, even with thick middle ear effusions, have fairly good hearing. Tympanometry is a more reliable diagnostic tool. A patient with chronic otitis media with effusion who has not received prior antimicrobial therapy should be treated initially as a case of acute otitis media with effusion, since bacteria are frequently present.

A study was conducted of 274 children who had recurrent acute or chronic otitis media with effusion. Of the ears with effusion 45 per cent were found to contain bacteria, and 11 per cent bacteria what were "probable pathogens" (*S. pneumoniae, H. influenzae, and S. pyogenes*). Bacteria were also found in 40 per cent of the ears without effusions. The type of organism found did not vary with the age of the patient studied or the season of the year. Pathogens have also been aspirated from young infants with chronic effusions. However, the efficacy of antimicrobials, corticosteroids, decongestants, and antihistamines for chronic otitis media with effusion has not been proven. Occasionally, attempts at middle ear inflation by Valsalva's or Politzer's method are successful.

If the effusion persists for eight weeks or longer, or if there have been frequent recurrences of episodes of acute otitis media with effusion, the patient requires further evaluation. Several avenues of investigation are open: a search for respiratory allergy may prove fruitful; a lateral roentgenogram of the nasopharynx may reveal adenoid tissue obstructing the nose and nasopharynx; immunologic studies may be of value if other organs are involved (the lung, for example). More thorough physical examination may reveal adonormalities, such as submucous cleft palate or a tumour of the nasopharynx, that require definitive management.

For those patients in whom medical management has failed, myringotomy with aspiration of the middle ear fluid is indicated. Frequently, insertion of a ventilation tube may

be necessary to allow the middle ear mucous membrane to return to normal and to prevent subsequent accumulation of effusion. Myringotomy and insertion of ventilation tubes may also be helpful in patients with atelectasis of the middle ear when significant symptoms - pain, hearing loss, vertigo, or tinnitus - are present. Ventilation tubes should be used to prevent permanent structural damage and cholesteatoma if a deep retraction pocket develops in the posterosuperior quadrant or in the attic (pars flaccida) portion of the tympanic membrane. Occasionally, troublesome otorrhea develops after the insertion of tympanostomy tubes. This can usually be treated successfully with ear drops containing neomycin, polymyxin, or colisting with hydrocortisone. Since these medications may be ototoxic, some physicians advocate the use of systemic antibiotics without the aural drops. In most children otitis media with effusion is usually self-limiting and will improve with advancing age, but in selected cases allergic hyposensitization and adenoidectomy may be beneficial; however, the efficacy of these methods of management has not been established. Tonsillectomy (in the absence of documented recurrent tonsillitis) does not seem to alter the course of otitis of any type and should not be performed alone or in conjunction with adenoidectomy for these conditions.

Since otitis media with effusion is universal in the infant with an unrepaired cleft palate and very common following surgical repair of the palate, tympanostomy tubes should be inserted when a chronic effusion is present to prevent the complications and sequelae of otitis media.

Complications and Sequelae of Otitis Media

Today, the intracranial suppurative complications of otitis media with effusion are relatively uncommon except in neglected cases. However, the complications and sequelae that occur within the aural cavity and adjacent structures of the temporal bone are more common, and awareness of their possible existence is essential in management of children with otitis media with effusion. Even though many of these less serious conditions may not threated life (as when there is an intracranial extension of the disease), the quality of life may be severely affected, making prevention imperative.

Nonsuppurative Complications and Sequelae

The nonsuppurative complications and sequelae of otitis media with effusion are hearing loss, adhesive otitis media, tympanosclerosis, and ossicular discontinuity.

Hearing loss is by far the most prevalent complication and morbid outcome of otitis media with effusion, and may be caused by one or more of the intra-aural complications or sequelae. To a varying degree, fluctuating or persistent loss of hearing is always associated with acute or chronic otitis media with effusion. The presence of high negative pressure within the middle ear (atelectasis), in the absence of an effusion, can also be associated with a significant hearing loss. The audiogram usually reveals a mild to moderate conductive loss. However, there may be a sensorineural component, generally attributed to the effect of increased tesnion and stiffness of the round window membrane. This hearing loss is usually reversible with resolution of the effusion, but permanent conductive hearing loss can result from irreversible changes secondary to recurrent acute or chronic inflammation, such as adhesive otitis, tympanosclerosis, or ossicular discontinuity. Irreparable sensorineural loss may also occur, presumably as the result of spread of infection through the round or oval window

membrane. Audiometry can be reliably performed in children over three years of age, but children under three years are the group at highest risk for effusions and associated hearing loss and in these patients standard audiometric assessment is difficult to perform reliably. Whenever an otitis media with effusion is diagnosed clinically or by tympanometry, there is a concurrent hearing loss. The relation between persistent or episodic hearing loss and impairment in the cognitive, language, and emotional development of children has been reported. However, the degree and duration of the hearing loss required to produce such deficits have not been defined.

Adhesive otitis media is the result of a healing reaction following inflammation of the middle ear. The mucous membrane is thickened by proliferation of fibrous tissue, which frequently impairs the movement of the ossicles, resulting in a conductive hearing loss. The condition is irreversible, making prevention of middle ear inflammation the most effective means of reducing the incidence of this condition, since medical and surgical treatment after adhesive otitis media is present has been disappointing.

Tympanosclerosis is characterized by the presence of whitish plaques in the tympanic membrane and nodular deposits in the submucosal layers of the middle ear. Pathologically there is hyalinization with deposition of calcium and phosphate crystals. Conductive hearing loss may result from embedment of the ossicles in the deposits. As in adhesive otitis media, a prerequisite seems to be chronic middle ear inflammation. Prevention has been the only successful means of controlling this disease.

Ossicular discontinuity is the result of rarefying osteitis secondary to chronic middle ear inflammation. The most common ossicle involved is the long process of the incus but the crural arch of the stapes, body of the incus, or manubrium of the malleus may also be eroded. The conductive hearing loss resulting from this complication frequently can be corrected surgically.

Suppurative Complications and Sequelae

The *intratemporal* suppurative complications and sequelae secondary to otitis media are perforation of the tympanic membrane, acquired cholesteatoma, mastoiditis, facial paralysis, and suppurative labyrinthitis.

Perforation of the tympanic membrane most frequently occurs with spontaneous rupture of the central portion of the eardrum during a bout of acute otitis media with effusion. Persistent purulent otorrhea may follow, in which instance a culture should be obtained, if possible, from the middle ear, and appropriate antimicrobials administered accordinly. In addition, antibiotic-cortisone otic medication may be helpful. Healing of the tympanic membrane frequently follows cessation of the suppurative process. A central perforation that fails to heal spontaneously and which is associated with a dry middle ear and good eustachian tube function may be amenable to surgical closure of the perforation with a graft - tympanoplasty. However, if the otorrhea persists, or if the drainage seems to be coming from an apparent posterosuperior or attic (pars flaccida) perforation, then a cholesteatoma should be suspected. *Chronic suppurative otitis media with mastoiditis* is represented by a perforation of the tympanic membrane or a cholesteatoma in which there is a persistent or episodic

purulent discharge. The most common pathogenic organisms are the gram-negative bacilli, such as *Bacillus proteus* and *Pseudomonas aeruginosa*. Aural polyps, which appear as red friable masses, may protrude through one of these perforations and may indicate the presence of a cholesteatoma. Treatment consists of active medical or surgical management, or both.

Acquired cholesteatoma is a sac-like structure lined by keratinized, stratified, squamous epithelium with accumulation of desquamating epithelium or keratin within the middle ear. Clinically, white, shiny greasy debris accompanied by a foul-smelling discharge can be observed in either the posterosuperior or an attic portion of the tympanic membrane and is a sequela of otitis media or atelectasis, or both. Tympanomastoid surgery is indicated, and if it is delayed the disease can invade and destroy other structures of the temporal bone and spread to the intracranial cavity.

Mastoiditis or inflammation of the mastoid air cell system frequently accompanies acute and chronic otitis media with effusion. Radiographic examination of this condition reveals a cloudy mastoid. The process is usually reversible as the effusion resolves with appropriate medical management. Occasionally, a severe acute otitis media with effusion is accompanied by mastoiditis in which there is pain, tenderness, edema, and erythema of the postauricular area. The pinna is displaced inferiorly and anteriorly, and swelling or sagging of the posterior-superior canal wall may also be present; this is the stage of mastoid periosteitis. It is treated with immediate tympanocentesis / myringotomy and systemic ampicillin, with possible later adjustment of medication according to the antibiotic susceptibility of the organism. If the condition progresses to the stage of rarefying osteitis, the infectious process may break thrugh the cortex of the mastoid to form a subperiosteal abscess. The infection may also break through the mastoid tip into the nexk (Bezold's abscess) or fistulize into the external ear canal. At this stage, when osteitis is present, mastoid surgery is required to prevent further intratemporal or intracranial complications. Petrositis may result from acute or chronic infections of the pneumatized apical and perilabyrinthine cells of the temporal bone. The triad consisting of otitis media with effusion, paralysis of the homolateral external rectus muscle, and pain in the homolateral orbit or retro-orbital area, constitutes Gradenigo syndrome.

Facial paralysis secondary to involvement of facial nerve may occur during an episode of acute otitis media with effusion. Exposure of facial nerve may occur during an episode of acute otitis media with effusion. Exposure of the nerve caused by a congenital bony dehiscence within the middle ear is thought to be pathogenic mechanism. When it occurs as an isolated complication, a myringotomy should be performed and parenteral antibiotics administered. The paralysis will usually improve rapidly without requiring further surgery (ie, facial nerve decompression). Mastoidectomy is not indicated unless mastoid osteitis is present as demonstrated by the clinical and roentgenographic appearance of the mastoid. However, immediate surgical intervention is indicated when a facial paralysis develops in a child who had chronic suppurative otitis media with or without cholesteatoma.

Suppurative labyrinthitis may occur during an episode of acute otitis media with effusion from the direct invasion of bacteria through the round or oval windows. When chronic suppurative otitis media is present the infection may penetrate the windows or enter through a pathologic fistula of the bony horizontal semicircular canal. Signs and symptoms

include vertigo, nystagmus, tinnitus, hearing loss, nausea, and vomiting. Treatment consists of intense parenteral antimicrobials; however, surgical labyrinthectomy may be indicated to prevent spread to the intracranial cavity.

Intracranial suppurative complications of acute and chronic otitis media are meningitis, focal encephalitis, brain abscess, sinus thrombophlebitis, extradural abscess, subdural abscess, and otic hydrocephalus. Today, these complications occur more often in association with chronic suppurative otitis and mastoiditis, with or without cholesteatoma, than with acute otitis media with effusion. Infection can spread from the middle ear and mastoid to the intracranial structures by one of the following routes: vascular channels (osteothrombophlebitis), direct extension (osteitis), or preformed pathways, for instance, round window, previous skull fracture, and congenital or surgically acquired bony dehiscences. Clinically, any child who has an acute or chronic otitis media and who develops one or more of the following signs or symptoms, especially while receiving medical treatment, should be suspected of having a suppurative intracranial complication: persistent headache, severe otalgia, onset of fever, nausea, or vomiting. The following signs and symptoms demand an intensive search for an intracranial complication: stiff neck, focal seizures, ataxia, blurred vision, hemiplegia, intention tremor, papilledema, diplopia, past-pointing, dysdiadochokinesia, aphasia, or hemianopsia. Conversely, children with intracranial infection (recurrent meningitis or brain abscess) must have middle ear - mastoid disease ruled out as the origin of the disease. The life-threatening complications of middle ear disease in children are relatively uncommon. Our goal should be to reduce these complications still further by effective management of acute and chronic otitis media with effusion.

Summary

1. Otitis media is one of the most common diseases of childhood.

2. Pathogenesis is related to eustachian tube dysfunction.

3. Etiology is primarily bacterial (*S. pneumoniae*, 40 per cent; *H. influenzae*, 20 per cent). Bacteria are also present in chronic otitis media with effusion ("secretory otitis").

4. *H. influenzae* is present in all age groups, and 15 to 30 per cent are ampicillin-resistant.

5. Diagnosis is by pneumatic otoscopy, or tympanometry, or both.

6. Tympanocentesis and/or myringotomy is important diagnostic-therapeutic procedure in selected patients.

7. Ampicillin (or amoxicillin) is initial therapy of choice.

8. Erythromycin and sulfonamide, trimethoprim-sulfamethoxazole, or cefaclor is recommended for those who have poor clinical response to initial antimicrobial therapy.

9. Efficacy is yet to be shown for antimicrobial prophylaxis, decongestants, antihistamines, myringotomy and tympanostomy tubes, and adenoidectomy with or without tonsillectomy.

10. Attendant conductive hearing loss is probably related to abnormalities in cognition, language, and learning.