Paparella: Volume II: Otology and Neuro-Otology

Section 3: Diseases of the Ear

Part 3: Middle Ear and Mastoid

Chapter 26: Diseases and Disorders of the Eustachian Tube-Middle Ear

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Diseases and disorders that affect the eustachian tube can result in middle ear disease. Thus, this chapter also includes the author's method of management and prevention of middle ear disease that is related to dysfunction of the eustachian tube. The reader is referred to Chapter 6 (Physiology of the Eustachian Tube and Middle Ear) in Volume I as a background to the content of this chapter and for descriptions of methods used to test the function of the eustachian tube. The reader is also advised to refer to two publications that contain state-of-knowledge information on eustachian tube dysfunction (Bluestone and Doyle, 1985; Sadé, 1987).

The eustachian tube has at least three physiologic functions related to the ear: ventilation, protection, and drainage and clearance. Interference with one or more of these functions can potentially impact on the function of the ear and adjacent structures.

Abnormal function of the eustachian tube appears to be one of the most important factors in the pathogenesis of ear disease. This hypothesis was first suggested more than 100 years ago by Politzer (1862). However, later studies by Zollner (1942), Suehs (1952), Senturia and colleagues (1958), and Sadé (1966) suggested that otitis media is a disease primarily of the ear mucous membrane - that is, due to infection or allergic reactions in this tissue, rather than related to dysfunction of the eustachian tube. Related to this hypothesis is the concept that nasopharyngeal infection spreads up the mucosa of the eustachian tube to the ear.

The vast majority of patients with otitis media and related conditions have (or have had in the past) abnormal function of the eustachian tube that may have caused secondary mucosal disease of the ear - that is, inflammation (Bluestone et al, 1972a). A much smaller number of patients may have primary mucosal disease as a result of infection, allergy, or, more rarely, an abnormality of the mucociliary transport system, such as Kartagener's syndrome (Fischer et al, 1978; Ernstson et al, 1984). Hematogenous spread of bacteria to the ear may also result in otitis media. However, an abnormality of the mucous membrane can affect eustachian tube function, and abnormalities of eustachian tube function can cause otitis media and certain related conditions.

The major types of abnormal function of the eustachian tube that can cause otitis media appear to be obstruction, abnormal patency, or both. Eustachian tube obstruction can be functional or mechanical or both. Functional obstruction results from persistent collapse of the eustachian tube due to increased tubal compliance, an abnormal 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 (Holborow, 1975; Bluestone et al, 1972a; Bylander, 1984a).

In addition, there appear to be marked age differences in the craniofacial base that may render the tensor veli palatini muscle less efficient prior to puberty (Bylander and Tjernström, 1983). Mechanical obstruction of the eustachian tube may be intrinsic or extrinsic. Intrinsic obstruction could be the result of abnormal geometry or intraluminal or mural factors that could compromise the lumen of the eustachian tube; the most common of these is inflammation due to infection or possibly to allergy (Bluestone et al, 1977a; Ackerman et al, 1984). Extrinsic obstruction could be the result of increased extramural pressure, such as occurs when the subject is supine or when there is peritubal compression secondary to a tumor (Myers et al, 1984) or possibly an adenoid mass (Bluestone et al, 1972c, 1975a).

In extreme cases of abnormal patency of the eustachian tube, the tube is open even at rest (ie, patulous). Lesser degrees of abnormal patency result in a semipatulous eustachian tube that is closed at rest but has low resistance in comparison with the normal tube. Increased patency of the tube may be due to abnormal tube geometry or to a decrease in the extramural pressure, such as occurs as a result of weight loss or possibly as a result of mural or intraluminal factors.

Eustachian Tube Dysfunction Related to Pathogenesis of Otitis Media and Atelectasis

Functional Obstruction

Functional obstruction of the eustachian tube may result in persistent high negative ear pressure; when associated with marked collapse or retraction of the tympanic membrane, it has been termed atelectasis. If ventilation occurs when there is high-negative ear pressure, nasopharyngeal secretions can be aspirated into the ear and result in acute bacterial otitis media.

If ventilation does not occur, persistent functional eustachian tube obstruction could result in sterile otitis media with effusion. Development of otitis media with effusion at this stage might be dependent upon the degree as well as ear hypoxia or hypercapnia. Since tubal opening is possible in an 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. Infants with unrepaired palatal clefts and many children with repaired cleft palates have otitis media as a result of functional obstruction of the eustachian tube (Bluestone, 1971).

Functional obstruction has been demonstrated in an experimental animal model (Cantekin et al, 1977). Following transection of the tensor veli palatini muscle posterior to the hamulus of the pterygoid bone in the Rhesus monkey, temporary high negative ear pressure and severe retraction of the tympanic membrane were noted to occur and persisted until the muscle healed. To test the hypothesis that if ventilation occurs when there is high negative ear pressure, nasopharyngeal secretions can be aspirated into the ear and can result in an acute bacterial otitis media with effusion, Cantekin and coworkers (1977) unilaterally transected the tensor muscle in the Rhesus monkey. The result was persistent high negative ear pressure without effusion, whereas in the unoperated side, ear pressure remained normal. Forty-eight hours after instillation of *Streptococcus pneumoniae* into the nasopharynx of the monkey, acute otitis media with effusion developed in the ear with the high negative ear

pressure, but not in the unoperated side. Cantekin and colleagues (1977) also reproduced the condition of persistent functional eustachian tube obstruction, resulting in sterile otitis media with effusion in the Rhesus monkey, by excision of the tensor muscle, which resulted in severe functional eustachian tube obstruction and the development of sterile otitis media with effusion shortly after the procedure.

Intrinsic Mechanical Obstruction

Intrinsic mechanical obstruction of the eustachian tube most commonly is the result of inflammation. Obstruction within the bony or ear portion of the tube is usually caused by acute or chronic inflammation of the mucosal lining, which also may be due to polyps or a cholesteatoma. Total obstruction may be present at the ear end of the tube. Stenosis of the eustachian tube has also been described but is a rare finding.

Most ears at risk for developing atelectasis or otitis media when inflammation is present probably have a significant degree of functional obstruction. An upper respiratory tract infection in children with this condition has been shown to decrease eustachian tube function significantly (Bluestone et al, 1977a; Bylander, 1984b). Periods of upper respiratory tract infection may then result in atelectasis of the tympanic membrane - middle ear, acute otitis media, or otitis media with effusion due to swelling of the eustachian tube lumen. The mechanisms are similar to those described for functional eustachian tube obstruction. Allergy as a cause of intrinsic mechanical eustachian tube obstruction has not been demonstrated in children. However, in adult volunteers, eustachian tube obstruction has been produced by a challenge with antigen inhaled into the nasal cavity (Ackerman et al, 1984).

Extrinsic Mechanical Obstruction

Functional mechanical obstruction of the eustachian tube may be the result of extrinsic compression by adenoids or, more rarely, by tumor. In an attempt to improve criteria for the preoperative selection of children for adenoidectomy to prevent otitis media with effusion, Bluestone and associates (1972c) made radiographic studies of the nasopharynx and eustachian tube prior to and following adenoidectomy. The ventilatory function of the eustachian tube has also been studied by the inflation-deflation manometric technique both before and after adenoidectomy in a group of children with recurrent or chronic otitis media with effusion in whom tympanostomy tubes had been inserted (Bluestone et al, 1975). The results of these studies indicated that, following adenoidectomy, eustachian tube function improved in some, remained the same in others, and worsened in a few children. Improvement was related to a reduction of extrinsic mechanical obstruction of the eustachian tube.

Partial tube obstruction may result only in atelectasis of the tympanic membrane - middle ear or bacterial otitis media with effusion; however, more severe obstruction could result in a sterile otitis media with effusion. Otitis media with effusion has been produced in animal models when the eustachian tube was mechanically obstructed (Paparella et al, 1970).

Abnormally Patent or Patulous Eustachian Tube

An abnormally patent or patulous eustachian tube usually permits air to flow readily from the nasopharynx into the ear, which thus remains well ventilated; however, unwanted nasopharyngeal secretions can also 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 ear may have negative pressure or an effusion or both. Since the tubal walls are abnormally distensible, nasopharyngeal secretions may be readily insufflated into the ear even with modest positive nasopharyngeal pressures, such as the results of nose-blowing, 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 is also likely.

Of 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 may enter the ear more readily than would air. Certain American Indians have been shown to have tubal resistances that are lower than those in the average white population (Beery et al, 1980). They seem to have an increased incidence of reflux of nasopharyngeal secretion into the ear and frequently suffer from recurrent acute otitis media that is often associated with perforation and discharge. However, American Indians have a low incidence of cholesteatoma. This type of eustachian tube function and ear disease is different from the types of disease seen in individuals who have a cleft palate.

Some patients with abnormally patent eustachian tubes have a habit of sniffing, which can result in a relative negative pressure in the nasopharynx and probably acts to temporarily close the proximal end of the tube. However, sniffing has been postulated to be the cause of high negative ear pressure (Magnusson, 1981). This condition has been considered by some investigators to be due to a failure of the eustachian tube to completely close at rest (Falk, 1984). However, other investigators, including the opinion and experience of the author, consider sniffing to be the cause of negative pressure in the ear in a small number of patients with eustachian tube dysfunction.

Nasal Obstruction Related to Eustachian Tube Function

Nasal obstruction may also be involved in the pathogenesis of ear dysfunction and otitis media. Swallowing when the nose is obstructed (owing to inflammation or possibly obstructed adenoids) results in initial positive nasopharyngeal air pressure followed by a negative pressure phase. When the tube is dysfunctional, positive nasopharyngeal pressure might conceivably insufflate infected secretions into the ear, especially when the ear has a high negative pressure; with negative nasopharyngeal pressue, a dysfunctional tube could be prevented from opening and could be obstructed functionally (Toynbee phenomenon) (Bluestone et al, 1974, 1975a; Jorgensen and Holmquist, 1984).

Allergy and Eustachian Tube Function

The role of allergy in the etiology and pathogenesis of acute otitis media and otitis media with effusion may be related to one or more of the following mechanisms: (1) ear functioning as a target or shock organ; (2) inflammatory swelling of the eustachian tube; (3)

inflammatory obstruction of the nose; and (4) reflux, insufflation, or aspiration of bacteria-laden nasopharyngeal secretions in the ear cavity (Bluestone, 1983). The last three mechanisms would be associated with abnormal function of the eustachian tube.

Even though there is a lack of convincing evidence that allergy plays a significant role in the etiology of otitis media, there may be a relation between upper respiratory tract allergy and eustachian tube dysfunction. In prospective study of children with recurrent or chronic ear disease who also had evidence of functional obstruction of the eustachian tube, these children had more severe obstruction (mechanical) of the tube when an upper respiratory tract infection developed (Bluestone et al, 1977a). A similar relationship has been reported between upper respiratory tract allergy and eustachian tube function. In a study of adult volunteers who had normal tympanic membranes and a negative otologic history and had normal eustachian tube function prior to an intranasal challenge with an antigen to which they were sensitive, all developed partial eustachian tube obstruction following the challenge (Ackerman et al, 1984). However, otitis media did not develop in these subjects. This study was subsequently reproduced using histamine in another laboratory (Walker et al, 1985). Recently, animals that had been passively sensitized had eustachian tube dysfunction following intranasal challenge (Doyle et al, 1984, 1985).

From these studies, the following sequence of events is postulated in patients who have allergy and otitis media. Most likely, a basic eustachian tube dysfunction is present that becomes clinically evident in the presence of upper respiratory tract allergy in a manner similar to eustachian tube obstruction caused by an upper respiratory tract infection. Upper respiratory tract allergy may cause some intrinsic mechanical obstruction in individuals who have normal eustachian tube function, but their normal active opening mechanism (ie, tensor veli palatini muscle pull) is able to overcome the obstruction. Therefore, individuals who have functional obstruction due to poor muscular opening would be at highest risk for developing sufficient mechanical obstruction to develop ear disease. Since many children have difficulty actively opening their eustachian tubes, they are a high-risk population. If the eustachian tube obstruction is minimal, the patient will have only signs and symptoms of *eustachian tube dysfunction* such as otalgia ("popping" and "snapping" sounds in the ear), mild hearing loss, and tinnitus or even vertigo. Often these symptoms will be fluctuating and will be present only during the worst periods of the patient's allergic rhinitis.

Even though there is no evidence that allergic nasopharyngeal secretions can be *aspirated* into the ear, this concept seems reasonable. Acute otitis media resulting from the aspiration of bacteria from the nasopharynx has been documented in animal models, which lends credence to this pathogenic mechanism. Likewise, it is possible that nasopharyngeal secretions containing bacteria could be *refluxed* into the ear if the eustachian tube is abnormally patent, which could result in "reflux otitis media".

Insufflation into the ear of bacteria-laden nasopharyngeal secretions secondary to allergy is also possible during nose-blowing, jumping or diving into water when swimming, in the infant who is crying, or during closed-nose swallowing (ie, the Toynbee phenomenon).

Allergic rhinitis that is severe enough to cause nasal obstruction may cause ear disease due to the Toynbee phenomenon. If nasal obstruction exists, the nasopharyngeal pressure during closed-nose swallowing is at first positive due to the elevation of the soft palate and

the closing off of the velopharyngeal space, followed by negative pressure when the soft palate is in a lower position. These pressures are similar to pressure changes that occur in the lower pharynx during physiologic swallowing. If the eustachian tube opens during the positive phase of closed-nose-swallowing, allergic nasopharyngeal secretions may be insufflated in the ear, resulting in otitis media. It is more likely, however, that the eustachian tube is prevented from opening due to the high negative pressure developed in the nasopharynx during the second phase of closed-nose swallowing. This may cause eustachian tube obstruction and negative ear pressure that would be manifested by signs and symptoms of eustachian tube dysfunction (fluctuating or sustained otalgia, hearing loss, tinnitus, and vertigo) or atelectasis of the tympanic membrane - middle ear; also, otitis media may develop. "Locking" of the eustachian tube could also be possible, in which it would be difficult for the patient to open the tube even after swallowing. Aspiration of allergic nasopharyngeal secretions in which bacteria are present may also be possible with this type of mechanism.

At present, however, there is no scientific proof in animals or humans that the Toynbee phenomenon is involved in the pathogenesis of otitis media, but the mechanism seems reasonable. Likewise, there is no conclusive evidence that upper respiratory tract allergy can cause otitis media when there is an associated eustachian tube dysfunction; however, the study of adult volunteers who had partial eustachian tube obstruction when they were challenged with antigen showed that the eustachian tube certainly may be involved by allergy. Even though conclusive proof of this relationship is lacking, it appears that, in the individual with upper respiratory allergy and eustachian tube dysfunction, otitis media may be a result of this combination. Future studies are needed to define the role of allergy in the pathophysiology of the eustachian tube and the pathogenesis of otitis media. Randomized clinical trials will be required to determine the efficacy of the currently popular forms of immunotherapy and allergy control in the prevention of otitis media.

Eustachian Tube Function Related to Cleft Palate

Otitis media with effusion is universally present in infants with an unrepaired cleft to the palate (Stool and Randall, 1967; Paradise et al, 1969). Palate repair appears to improve ear status, but ear disease nonetheless often continues or recurs even after palate repair (Paradise and Bluestone, 1974; Doyle et al, 1986). Radiographic assessment has shown that infants and children with both unrepaired and repaired cleft palates have abnormal eustachian tube function, which suggests an abnormal opening mechanism in infants with an unrepaired cleft palate (Bluestone, 1971; Bluestone et al, 1972a) and either a persistent failure of the eustachian tube to open actively or increased distensibility of the eustachian tube, or both, after repair of the soft palate (Bluestone et al, 1972b). However, Falk and Magnusson (1984) postulated that the tube in patients with cleft palate may have "closing failure". Following certain palatal repairs, the tube may be too open in some cases.

Inflation-deflation manometric eustachian tube function tests have shown that infants with unrepaired cleft palates have variable degrees of difficulty equilibrating increased ear pressure and are unable to equilibrate negative pressure by active function (swallowing) (Bluestone et al, 1975b). Children with repaired cleft palates had either the same type of test results as those with unrepaired palates or had lower opening pressures. Doyle and colleagues (1980a), employing the forced-response test, found that the eustachian tube of infants and children with cleft palates constricted instead of dilating during swallowing. Animal models

in which the palate was surgically split have developed otitis media with effusion (Odoi et al, 1971; Doyle et al, 1980b).

All these studies indicate that the eustachian tube is functionally obstructed in children with cleft palates, which results in ear disease characterized by persistent or recurrent high negative ear pressure, effusion, or both. Cholesteatoma is a frequent sequel in such children; this is not the case in American Indians, in whom the eustachian tube has been shown to be abnormally patent, that is, to have low tubal resistance (Beery et al, 1980).

Patients with a submucous cleft of the palate appear to have the same risk of developing ear disease as those with an overt cleft. In addition, the presence of a bifid uvula has also been associated with a high incidence of otitis media (Taylor, 1972). However, Fischer and associates (1987) found no such association in a population of American Indians. Both these conditions are probably associated with the same pathogenic mechanism for otitis media that is found in patients with overt cleft palates - that is, functional obstruction of the eustachian tube.

Other Causes of Eustachian Tube Dysfunction

There are many etiologic factors responsible for abnormal function of the eustachian tube. Inflammation of the nose-nasopharynx-eustachian tube - middle ear system has been presented as a major factor in the pathogenesis of otitis media; however, congenital, traumatic, neoplastic, degenerative, metabolic, and idiopathic conditions also can result in tubal abnormalities.

Since a cleft palate results in functional obstruction of the eustachian tube, any child with a craniofacial malformation that has an associated cleft palate will have otitis media or a related condition, one of the more common examples being the Pierre Robin syndrome. However, children with craniofacial anomalies that do not include an overt cleft palate also have an increased incidence of ear disease. These anomalies include, among others, syndromes such as Down's, Crouzon's, Apert's, and Turner's. White and coworkers (1984) performed eustachian tube function tests on 14 children who had Down's syndrome and who had had tympanostomy tubes previously inserted for recurrent or chronic otitis media with effusion. These children were described as having eustachian tubes that had low resistance (ie, too open) and that failed to dilate during swallowing. Although there have been no other reports of formal eustachian tube function studies in individuals with these and other anomalies, dysfunction of the eustachian tube is the most likely cause of such ear disease. Presumably, a defect related to the abnormal craniofacial complex influences the relation between the eustachian tube and the tensor veli palatini muscle.

Even in the absence of an obvious craniofacial malformation that is associated with otitis media, there is some evidence that children and adults with ear disease have a congenital defect that results in a dysfunction of the tube. Such a dysfunction could be abnormal patency or functional obstruction of the tube which is the result of an abnormal relation between the eustachian tube and the tensor veli palatini muscle. Such an assumption is supported by apparent racial differences in the prevalence and incidence of otitis media: Eskimos and American Indians have a higher incidence of otitis media than do whites, whereas blacks have an incidence of otitis media that is half that in whites (Doyle, 1977).

There is also some evidence that otitis media is more prevalent in certain families. In the Boston Collaborative Study (Doyle, 1979), a familial tendency to otitis media has been found.

It has also been observed that patients with dentofacial abnormalities may have otitis media or may develop ear disease as a result of these abnormalities. Correction of the defect to relieve the eustachian tube dysfunction would appear to be indicated.

In certain patients with a deviated nasal septum, impaired eustachian tube function has been reported. This dysfunction is especially apparent during attempts to equilibrate ear pressure by the Valsalva maneuver during periods of wide fluctuations in barometric pressure, such as flying in an airplane or diving. In such cases, successful inflation of the ear by the Valsalva maneuver has been reported following repair of the deviated nasal septum (McNicoll and Scanlan, 1979).

Trauma to the palate, the pterygoid bone, the tensor veli palatini muscle, or the eustachian tube itself can result in abnormal eustachian tube function (Myers et al, 1984). Injury to the trigeminal nerve, or more specifically, to the mandibular branch of this nerve, can result in either functional obstruction of the eustachian tube or a patulous tube, since the innervation of the tensor veli palatini is from this nerve (Perlman, 1951; Cantekin et al, 1979a). The trauma may be associated with surgical procedures such as palate or maxillary resection for tumor.

Neoplastic disease, either benign or malignant, that invades the palate and pterygoid bone can interfere with tensor veli palatini muscle function and can result in functional obstruction of the tube (Takahara et al, 1986). Functional obstruction or abnormal patency of the tube can also occur as a result of involvement of the innervation of the tensor veli palatini muscle. Mechanical obstruction of the eustachian tube can result from direct invasion by neoplasm. Degenerative and metabolic diseases such as myasthenia gravis can alter the eustachian tube by affecting the tubal musculature or by changing the extramural or mural pressures, as would occur with major shifts of extracellular fluids.

Whenever eustachian tube dysfunction is diagnosed and the etiology is obscure, the dysfunction is usually considered to be idiopathic. Most patients with otitis media have been found to have functional obstruction of the eustachian tube with an idiopathic etiology. It should not be forgotten, however, that the cause may be a congenital defect in the anatomy of the base of the skull.

Management of Eustachian Tube Dysfunction

Obstruction

Otitis media and atelectasis are usually the result of functional or mechanical dysfunction of the eustachian tube. However, abnormal function of the eustachian tube may cause otologic symptoms without an apparent effusion or severe atelectasis. The tympanic membrane may even have a normal appearance and mobility may be unimpaired when tested by pneumatic otoscopy or by tympanometry. Two types of eustachian tube dysfunction can be present: obstruction or abnormal patency. When the eustachian tube is obstructed but no effusion is present, the tube periodically opens to ventilate the ear cavity but at less frequent

intervals than normal; in this case, high negative intratympanic pressure may be present for relatively long periods. This type of intermittent ear ventilation may cause periods of otalgia, a feeling of fullness or pressure, hearing loss, popping and snapping noises, tinnitus, and even vertigo.

Medical treatment should be directed toward relief of the nasal congestion, if the condition is present only during an episode of acute upper respiratory tract infection. However, if the symptoms are of a chronic nature, a search for an underlying cause should be attempted, as described previously in this chapter, and if found, appropriate management instituted. If no underlying cause is uncovered, then a trial with a decnogestant or antihistamine or both may be helpful, or eustachian tube-middle ear inflation may be tried. If nonsurgical methods are not successful, myringotomy, and possibly insertion of a tympanostomy tube, may be necessary.

Abnormally Patent Eustachian Tube

At the other end of the spectrum of eustachian tube dysfunction is abnormal patency. In its extreme form, the hyperpatent eustachian tube is open even at rest (ie, patulous). Lesser degrees of abnormal patency result in a semipatulous eustachian tube that is closed at rest but has low tubal resistance to airflow in comparison to the normal tube. A patulous eustachian tube may be due to abnormal tube geometry or to a decrease in extramural pressure, such as occurs as a result of weight loss or possibly as a result of mural or intraluminal changes, which may occur when the extracellular fluid is altered by medical treatment of another, unrelated condition. Interruption of the innervation of the tensor veli palatini muscle has also been shown to be a cause of a hyperpatent eustachian tube (Perlman, 1939).

Palatal myoclonus may also cause a patulous eustachian tube due to rhythmic contractions of the tensor veli palatini muscle (Pulec and Simonton, 1961). The cause of palatal myoclonus remains obscure, but intracranial and extracranial vascular lesions, multiple sclerosis and tumor must be considered.

Clinically, a patulous Eustachian tube may be present in adolescents and adults but is rarely seen in young children. The patient frequently complains of hearing his/her own breathing in the ear or of autophony. Otoscopic examination reveals a tympanic membrane that moves medially on inspiration and laterally on expiration; the movement can be exaggerated with forced respiration. The condition is relieved when the patient is recumbent, since Eustachian tube extramural pressure is increased by paratubal venous engorgement in this position. The patient should therefore be examined in the sitting position and is best visualized with the aid of otomicroscope. The diagnosis can also be made by measuring the impedance of the ear (Bluestone, 1980). A tympanogram should be obtained while the patient is breathing normally and a second one obtained while the patient holds his breath. Fluctuation in the tympanometric line should coincide with breathing. The fluctuation can be exaggerated by asking the patient to occlude one nostril and close the mouth during forced inspiration and expiration, or by performing the Toynbee or Valsalva maneuver.

Management of a patulous eustachian tube depends on first determining the etiology of the problem. If the symptoms are of relatively short duration, the condition may subside without any active treatment. In children and teenagers, this condition is usually self-limited

and probably related to changes in the structure and function of the eustachian tube and adjacent areas secondary to rapid growth and development. When a medication can be identified as the responsible agent, cessation of the medication usually alleviates the problem. In most instances, however, the condition is idiopathic. When the symptoms are disturbing and the condition is chronic, active treatment is indicated. A myringotomy with insertion of a tympanostomy tube may be performed but usually does not alter the symptoms in most cases and occasionally will result in increasing the patient's discomfort. Insufflation of powders into the eustachian tube (the Bezold treatment - insufflation of boric and salicylic acid powder) and instillation of 2 per cent iodine or 5 per cent trichloroacetic acid solution has also been advocated (Mawson, 1974). Infusion of an absorbable gelatin sponge solution has also been suggested (Ogawa et al, 1976), as has injection of polytetrafluoroethylene (Teflon) into the paratubal area (Pulec, 1967); however, all these methods have major disadvantages. Changes produced by these methods are, for the most part, irreversible and may not improve the condition or may provide only temporary relief. Total obstruction of the eustachian tube can also be a complication. Stroud and coworkers (1974) have suggested the transposition of the tensor veli palatini through a palatal incision, but the procedure has not been shown by other investigators to be safe and effective in a large number of patients.

At presengt, the most logical choice for relief when the discomfort becomes severe is a procedure that would alleviate the symptoms simply, reversibly, and without untoward reactions. The technique described below has been found to fulfill these criteria and has been successful in relieving the symptoms of patulous eustachian tube (Bluestone and Cantekin, 1981). An anterior tympanotomy approach is used to insert an indwelling, intravenous catheter with a flared tip (Medicut) into the protympanic, or bony, portion of the eustachian tube. Prior to insertion, the lumen of the catheter is filled with methyl methacrylate to prevent the passage of air through the catheter; thus the catheter occludes the eustachian tube. Following insertion of the catheter, a tympanostomy tube is inserted into the tympanic membrane to aerate the ear through the membrane. Even though spontaneous extrusion of the tympanostomy tube may occur, the ear may remain aerated with relief of symptoms and without development of high negative pressure or effusion. The catheter most likely does not totally obstruct the eustachian tube, and adequate ventilation of the ear is provided around the catheter. Less than 20 patients have had the procedure, but the results have been gratifying. In the few patients who have not been helped, the reason for failure was inability to properly seat the catheter in the middle ear end of the eustachian tube. However, the indwelling eustachian tube catheter can be removed at any time, especially if and when the etiology of this most perplexing otologic problem is uncovered and a nonsurgical or surgical method of management is shown to be more efficacious. In the meantime, this method to partially obstruct the eustachian tube appears to be effective in providing relief of symptoms of a patulous eustachian tube.

Management of Atelectasis of the Tympanic Membrane, High Negative Middle Ear Pressure, and Retraction Pockets

Atelectasis of the tympanic membrane can be acute or chronic, localized or generalized, and mild or severe and may or may not be associated with abnormal negative pressure. The condition is caused by obstruction of the eustachian tube, functional, mechanical, or both, but it is not due to abnormal patency. Retraction of the tympanic membrane may be secondary to the presence of high negative pressure. However, a flaccid,

atelectatic tympanic membrane may not be associated with high negative intratympanic pressure: the abnormal negative pressure may have been the original cause of such a condition of the membrane but may no longer be present. Localized atelectasis or a retraction pocket may be seen in the area of a healed perforation or at the site where a tympanostomy tube had been inserted ("atrophic scar" or dimeric membrane). A retraction pocket in the posterosuperior portion of the pars tensa or a pars flaccida retraction pocket is more frequently associated with the development of more serious sequelae (ossicular discontinuity or cholesteatoma) than is a retraction pocket in other areas of the tympanic membrane. These variations should be kept in mind when deciding how to manage atelectasis. In all patients who have chronic atelectasis/retraction pocket of the tympanic membrane - middle ear, a search for an underlying cause of eustachian tube obstruction should be conducted, as described earlier in this chapter.

If chronic ear effusion is present concurrently with atelectasis, then the patient should be treated similar to patients with otitis media with effusion. However, whether or not a middle ear effusion is present, if a chronic severe retraction pocket of the posterosuperior area of the pars tensa or of the pars flaccida, or both, is present, myringotomy and insertion of a tympanostomy tube should be performed to prevent possibly irreversible changes in the ear. After insertion of a tympanostomy tube, the tympanic membrane in the area of the retraction pocket should return to a more neutral position within several weeks or months; however, if the retraction area remains adherent to the ossicles or ear or to both, then adhesive otitis media is present, and tympanoplasty should be considered to prevent further progression of the disease process (such as ossicular discontinuity or cholesteatoma formation or both). Even though this method of management has not been tested in appropriately controlled clinical trials, and the natural history of retraction pockets in these areas has not been studied adequately, this method of management appears to be reasonable at present (Bluestone et al, 1977b).

For less severe cases in which the atelectasis of the tympanic membrane apparently is not associated with an ear effusion and in which a retraction pocket is not present in the posterosuperior portion or pars flaccida, the management options become less obvious and more controversial. Generalized atelectasis, or even a localized area that is retracted for only a short time (acute retraction), is usually secondary to transient high negative end pressure associated with an acute upper respiratory tract infection (and occasionally due to barotrauma). This condition is quite common in children and usually is self-limited. No specific treatment should be directed toward the ear unless the child complains of severe otalgia, hearing loss, tinnitus, or vertigo. The atelectasis (and high negative intratympanic pressure) and associated symptoms, if present, will usually subside when the acute upper respiratory tract infection disappears. Treatment at this time should be directed toward relief of the nasal symptoms. Topical or systemic nasal decongestants may provide relief of these symptoms and may also relieve congestion of the eustachian tube, although their effectiveness in this latter area has not yet been shown. If the symptoms become severe enough, myringotomy may be necessary to provide relief by returning ear pressure to ambient levels. Inflation of the eustachian tube - middle ear system by employing the methods of Valsalva or Politzer has been advocated, but studies in animals indicate that these methods will not return the ear pressure to normal for a sustained period when the eustachian tube is obstructed (Cantekin et al, 1980a). Even though controlled trials in children have not been reported to

demonstrate the efficacy of these methods, it is the opinion of the author that repeated attempts to inflate the ear with air, in such cases, is ineffective.

When the atelectasis is chronic and there is no evidence of a deep retraction pocket in the posterosuperior quadrant or pars flaccida, a thorough search should be made for an underlying origin, as described previously. If none is found, then the management options include only watchful waiting and active treatment. The decision for or against treatment should rest on the presence or absence of other, associated symptoms, and whether there is abnormal negative pressure within the ear. The presence of persistent or transient otalgia, hearing loss, vertigo, or tinnitus that is troublesome to the patient warrants active treatment. For chronic atelectasis in this case, a trial with a topical or systemic nasal decongestant with or without an antihistamine may be helpful; however, this type of treatment is often disappointing. Inflation of the eustachian tube and ear may provide temporary relief but usually must be repeated for permanent control of the symptoms and to maintain the tympanic membrane in a more normal position. For most patients, especially children, myringotomy with insertion of a tympanostomy tube will usually be necessary to provide long-term relief. The procedure will prevent the sustained or transient high negative pressure secondary to eustachian tube obstruction that is responsible for the active retraction of the tympanic membrane. If the severely atelectatic tympanic membrane does not return to a more normal position after the insertion of the tympanostomy tube, or if the tube cannot be inserted owing to a lack of suitable aerated space within the ear, a tympanoplasty should be considered.

When a flaccid tympanic membrane is passively collapsed upon the ossicles and ear and high negative ear pressure is not present, the nonsurgical and surgical management options described above may not be effective in restoring the tympanic membrane to a more normal position. Fortunately, symptoms of high negative ear pressure and eustachian tube obstruction are frequently absent, so no treatment may be necessary. Even myringotomy and tympanostomy tube insertion may not be beneficial, since the tympanic membrane is no longer actively being retracted by high negative ear pressure. In addition, at this stage, adhesive otitis media may also be present, and portions of the tympanic membrane may be adherent to the ear. The posterior or epitympanic (attic) portions of the ear may become separated from the anterior portion by adhesions, and subsequently ventilation from the eustachian tube or a tympanostomy tube does not aerate the affected area. In such cases there are two management options: tympanoplasty or periodic (once or twice a year) observation.

Otitis Media With Effusion

The presence of a relatively asymptomatic middle ear effusion has many synonyms, such as "secretory", "nonsuppurative" or "serous" otitis media, but the most acceptable term is *otitis media with effusion.* The duration (not the severity) of the effusion can be divided into acute (less than 3 weeks), subacute (3 weeks to 2 to 3 months) and chronic (greater than 2 to 3 months). The most important distinction between this type of disease and acute otitis media (acute "suppurative" otitis media) is that the signs and symptoms of acute infection are lacking in otitis media with effusion (eg, otalgia and fever); hearing loss is usually present in both conditions. The disease is usually due to eustachian tube dysfunction, as described earlier in this chapter.

Otitis media with effusion has been assumed to be sterile, since several reports describe unsuccessful attempts to culture bacteria from them. However, a study was conducted in Pittsburgh in 1977 by Riding and coworkers (1978) of 179 children aged 1 to 16 years who had chronic middle ear effusions. Of 179 ears, bacteria were cultured from 84 (48 per cent) chronic middle ear effusions. Bacteria were present in serous and mucoid effusions as well as in the purulent type. Other investigators have reported similar findings (Healy and Teele, 1977; Liu et al, 1976; Senturia et al, 1958; Stanievich et al, 1981).

More recent findings from the Pittsburgh Otitis Media Research Center show that in approximately 4500 middle ear aspirates from ears with chronic otitis media with effusion, about two-thirds of the aspirates had bacteria isolated; of the one-third that were considered to be pathogens, the most common bacteria were *Haemophilus influenzae, Branhamella catarrhalis,* and *S. pneumoniae,* the most common pathogens found in middle ear aspirates from children with acute otitis media. In addition, *Staphylococcus epidermidis* was cultured from many ears when this organism was not cultured from the external canal of the same ear (a culture preceded sterilization and tympanocentesis). Beta-lactamase activity was similar to that reported for isolates from ears with acute otitis media; 20 to 30 per cent of *H. influenzae* and 70 to 85 per cent of B. catarrhalis produce beta-lactamase. Anaerobic bacteria were also isolated, but of low magnitude.

Management

Infants and children who have otitis media with effusion most likely have a condition that is an extension of an upper respiratory tract infection, which should resolve spontaneously without active treatment (Casselbrant et al, 1985). However, treatment may be indicated in some children, since possibly there are complications and sequelae associated with this condition. Since little information is presently available regarding the incidence of these complications and sequelae, some thoughtful clinicians take a watch-and-wait position and do not actively treat such a child. However, hearing loss of some degree usually accompanies a middle ear effusion. Although the significance of this hearing loss is still uncertain, such a loss may impair cognitive and language function and result in disturbances in psychosocial adjustment.

With these uncertainties in mind, the clinician should decide whether to treat or to watch and, if treatment is decided upon, which treatment option or options appear to be most appropriate in eliminating the middle ear effusion in the individual child. Many factors should be considered in this decision-making process. A child with a unilateral, asymptomatic otitis media with effusion of recent onset, in whom there is only a mild hearing loss and in whom there are no serious secondary changes in the tympanic membrane, may be a candidate for watchful waiting. Conversely, a child with bilateral chronic middle ear effusions who has an associated marked hearing loss is a more likely candidate for active treatment.

When deciding to treat or not to treat (and which treatment), important factors that should be considered in addition to hearing loss include one or more of the following:

1. Occurrence in young infants, since they are unable to communicate about their symptoms and may have suppurative disease.

2. An associated acute purulent upper respiratory tract infection.

3. Concurrent permanent conductive/sensorineural hearing loss.

4. Vertigo or tinnitus.

5. Alterations of the tympanic membrane, such as severe atelectasis, especially a deep retraction pocket in the posterosuperior quadrant or the pars flaccida or both.

6. Middle ear changes, such as adhesive otitis or ossicular involvement.

7. Persistence of the effusion 2 to 3 months or longer (ie, chronic otitis media with effusion).

8. Frequent recurrence of the episodes, resulting in an accumulation of an excessive duration of middle ear effusion during a given period of time, such as 6 out of 12 months.

Before embarking on a nonsurgical or surgical method of management of children with frequently recurrent or chronic effusions, a thorough search for an underlying etiology (ie, paranasal sinusitis, adenoiditis, and/or obstructing adenoids, upper respiratory allergy, submucous cleft palate, nasopharyngeal tumor) should be attempted.

Of the many methods of management that are available for otitis media with effusion, few have been reported to be effective in acceptable clinical trials. However, the clinician is forced to make a decision to treat actively or not to treat (watchful waiting), and, if treatment is decided on, which of the surgical or nonsurgical treatment options would be reasonable and most appropriate for the individual child. The most rational approach initially should be a trial of one or more of the nonsurgical methods; if the effusion is still persistent, then periodic observation or surgical intervention should be considered: The decision between these latter options should be based on the signs and symptoms present and should consider the potential complications and sequelae of *both.*

Probably the most popular method of management, a trial with an orally administered combination of a decongestant and antihistamine has recently been shown to be ineffective in infants and children with acute, subacute, and chronic otitis media with effusion (Cantekin et al, 1983). Their use for this disease in children is not recommended; however, but they may be effective in adolescents and adults or patients of all ages in whom there is evidence of upper respiratory allergy, since the study did not test the efficacy of this drug combination in these populations.

The efficacy of topical intranasal and systemic corticosteroid therapy has been tested, but convincing clinical trials have not been reported. In addition, some thoughtful clinicans believe that the risks of corticosteroid therapy for otitis media with effusion in children outweigh its possible benefits (Macknin and Jones, 1985). Even though clinical trials have not been reported which have tested the efficacy of immunotherapy and control of allergy in children with evidence of upper respiratory allergy, this method of management seems reasonable in children who have frequently recurrent or chronic otitis media with effusion. Likewise, inflation of the eustachian tube - middle ear using the method of Politzer or

employing the Valsalva maneuver may have merit. Inflation that achieves a positive middle ear pressure should enhance drainage of a thin (serous) middle ear effusion down the eustachian tube into the nasopharynx. Unfortunately, no randomized controlled trials have been reported to establish the efficacy of such procedures; therefore, it is seldom recommended, especially in children. However, middle ear inflation may be helpful in adults who have serous effusions.

Of all the medical treatments that have been advocated, a trial of an antimicrobial agent would appear to be most appropriate in those children who have not received an antibiotic recently. Bacteria similar to those found in acute otitis media have been isolated from a significant proportion of middle ear aspirates in children with chronic otitis media with effusion. In a study recently reported by Mandel and colleagues (1987), amoxicillin (40 mg/kg/day) in three divided doses) given for 14 days was twice as effective as placebo; therefore, amoxicillin would be a reasonable agent. However, if the effusion is chronic and unresponsive to amoxicillin therapy, a trial with an antimicrobial agent effective against ampicillin-resistant bacteria might by of benefit prior to consideration for surgery. Appropriate agents would be amoxicillin-clavulanate, erythromycin and sulfisoxazole, trimethoprim-sulfamethoxazole, or cefaclor; however, clinical trials have not been reported that have shown these antimicrobials to be superior to amoxicillin for otitis media with effusion.

If nonsurgical methods of management fail, then surgical intervention should be considered. Myringotomy with aspiration of the middle ear effusions appears to be appropriate in those children in whom the procedure can be performed *without* the aid of a general anesthetic, since a second myringotomy with or without the insertion of a tympanostomy tube would be indicated if the effusion is present soon after the myringotomy incision heals (ie, if the disease is persistent). It is desirable to avoid the risk of administering a second general anesthetic; if a myringotomy is elected and general anesthesia is required, a tympanostomy tube should be inserted at the time of the initial myringotomy to preclude, if possible, the necessity for performing a second procedure under general anesthesia should a tube later be required (Mandel et al, 1984). This mwethod of management appears at present to be the most reasonable. The efficacy of adenoidectomy with or without tonsillectomy for chronic otitis media with effusion may benefit about one-third of these children, but one-third spontaneously improve and one-third have persistent disease despite the adenoid/tonsil surgery (Maw, 1985). Following spontaneous extubation of the tympanostomy tube, reinsertion for recurrence of effusion would be indicated only after antimicrobial therapy has failed and the effusion has persisted for 2 to 3 months.

In some children the procedure must be repeated for several years until the child grows older. For children who have had chronic otitis media with effusion that appears to be resistant to the methods or management described above, mastoidectomy has been advocated (Porud and Duff, 1976), but this procedure is rarely indicated and should be reserved for those children in whom mastoid osteitis or a cholesteatoma is suspected, since almost all chronic effusions are at least temporarily eliminated following tympanostomy tube insertion. (See also Chapter 27, "Otitis Media with Effusion.")

Otitic Barotrauma and Eustachian Tube Dysfunction

Since one of the functions of the eustachian tube is to ventilate the middle ear and maintain approximately the same gas pressure within the middle ear as the surrounding atmospheric pressure, individuals with eustachian tube dysfunction may have significant otologic problems during activities, such as flying or sports diving, which can exceed the physiologic characteristics of the human tubal system. People who have patulous eustachian tubes, whether or not they are symptomatic, rarely complain of ear symptoms during these activities, since gas can pass freely into and out of the middle ear. However, patients who have eustachian tube obstruction (functional or mechanical) may have complaints referrable to the ear, depending on the type and severity of the obstruction and the degree of stress placed on the tubal system. Even individuals who have excellent eustachian tube function and a negative history of symptomatic ear disease may experience some middle ear discomfort during rapid descent in an airplane or when diving. (See also Chaptrer 34, "Underwater Medicine," and Chapter 35, "Aerospace Medicine.")

The Normal Eustachian Tube During Abnormal Conditions

Under physiologic conditions, individuals who have normal tubal function can equilibrate minor variations in atmospheric pressure that are gradual, such as occurs when climbing up and down a mountain, by contraction of the tensor veli tympani muscle during swallowing. Likewise, when the normal eustachian tube is healthy, ascent and descent in an airplane is usually not a problem. During ascent, the relative positive pressure that develops in the middle ear, due to the increase in gas volume within the cavity, is equilibrated by periodic swallowing; even in the absence of swallowing, the tube will be passively opened and middle ear gas will pass down the eustachian tube when the pressure reaches the forced opening pressure of the tube. However, during descent, when a relative negative middle ear pressure develops due to decrease in gas volume in the cavity, the eustachian tube must actively open by swallowing, yawning, or movement of the jaw to permit gas to enter the middle ear from the nasopharynx. When the normal eustachian tube system is subjected to extreme unphysiologic conditions, ear pathology can result. For example, if a diver does not actively equilibrate middle ear pressures during descent at a depth of 1.17 meters (3.9 feet), a 90 mm Hg pressure differential is developed that can lock the eustachian tube (Ivarsson et al, 1977). Locking it the condition in which the eustachian tube is closed so tightly, due to sudden development of negative pressure within the middle ear, that swallowing and other attempts (eg, Valsalva's maneuver) at dilating the tube cannot open it (Ingelstedt et al, 1967). If a diver does not equilibrate the relative negative pressure that develops within the middle ear frequently during a gradual descent, otitic barotrauma can result (Farmerr, 1985). A similar outcome can occur in individuals with normally functioning eustachian tubes, following extremely rapid descent in a plane.

When changes in environmental pressures are not great or do not develop as rapidly as described above during diving or flying, but an upper respiratory tract inflammation (ie, infection or allergy) is present, middle ear problems can develop even in individuals who are otherwise otologically healthy and who, during similar periods of inflammation have few if any ear complaints when not engaging in such unphysiologic activity. Viral infections can cause mechanical eustachian tube obstruction in adults with normal tympanic membranes and a negative otologic history (McBrdie et al, unpublished 1987). Likewise, upper respiratory

allergy has been shown to compromise tubal function in individuals who had had excellent tubal function when not exposed to the antigens they were sensitive to (Friedman et al, 1983). When upper respiratory inflammation is present, a partially obstructed tube can usually open passively during ascent due to the compliance of the tubal walls; however, on descent locking may occur, which can result in ear pathology. Since most, if not all, infants and young otologically healthy children appear to have difficulty in dilating (by swallowing) the eustachian tube when even a low negative pressure is applied to the ear (ie, functional obstruction) (Bluestone et al, 1972a; Bylander and Tjernström, 1983), they can develop significant middle ear discomfort during descent in a plane, even though the cabin is pressurized and the rate of descent is gradual; in most commercial aircraft, the cabin is pressurized to a level that is between 6000 and 8000 feet. Infants who cry during descent are probably compensating for the negative pressure in the middle ear by insufflating gas from the nasopharynx into the middle ear cavity. Surprisingly, infants and children who have middle ear effusion present when they fly experience little ear discomfort compared with those who are effusion free. This effect probably is related to the volume gas in the middle ear and the distensibility of the tympanic membrane.

The Abnormal Eustachian Tube During Abnormal Condition

Patients who have chronic eustachian tube obstruction are at particular risk for developing middle ear discomfort or pathology, even when mild-to-moderate stress is placed on the system. Most patients, especially children, who have recurrent/chronic middle ear disease have eustachian tubes that are functionally obstructed, in that they have difficulty actively dilating the tube by swallowing; the tube is not mechanically obstructed. In the absence of upper respiratory inflammation, such patients will have no ear problems in a plane during ascent, since the tube will be forced open; but on descent, ear discomfort develops due to the middle ear negative pressure. They may have difficulty equilibrating middle ear negative pressure even during gradual descent, which can lead to a recurrence of their ear disease, or even inner ear barotrauma. The presence of an upper respiratory tract infection or allergy can further compromise a functionally obstructed tube by superimposing partial or complete mechanical obstruction. When partial inflammatory obstruction is present, the tube may still be forced open during ascent, but the tubal opening pressure will be higher and on descent equilibration may not be possible. If mechanical obstruction is severe, otalgia may be present on ascent as well as descent. Though much less common than functional obstruction as a cause of eustachian tube dysfunction, some patients have tubes which are pathologically obstructed to some degree, all or most of the time, such as when tubal stenosis is present. These patients will complain of ear discomfort on ascent as well as descent.

Otitic Barotrauma

Middle Ear Barotrauma. Also called aerotitis, this is inflammation of the middle ear (and mastoid air-cell system) secondary to rapid development of negative (or less commonly positive) middle ear pressure. It is the most common medical disorders experienced by divers and is also prevalent among flight personnel as well as passengers in airplanes. When the middle ear cleft is under rapidly induced high negative pressure, the tympanic membrane becomes retracted, the mucosal lining becomes edematous, and capillaries are engorged, resulting in transudation of middle ear effusion. In mild cases the patient may complain of some hearing loss, mild otalgia, tinnitus, and even vertigo. In some patients, the tympanic

membrane may only be retracted, due to negative middle ear pressure, but no effusion is present; in more severe cases, hemotympanum can occur or even rupture of the eardrum. During descent when diving, the tympanic membrane may rupture at a pressure differential ranging from 100 to 400 mm Hg, which is equivalent to depths of 1.29 to 5.22 meters (4.3 to 17.4 feet) (Famer, 1982).

Inner Ear Barotrauma. This condition most frequently is a complication of middle ear barotrauma that develops during descent; such injuries to the inner ear are not uncommon, especially during diving (Edmunds et al, 1973). The postulated pathogenesis involves the association of high negative middle ear pressure and increased cerebral spinal fluid pressure, which occurs during attempts to inflate the middle ear by Valsalva's maneuver that results in rupture of the round or oval windows and leak of perilymph into the middle-ear cavity (Freedman and Edmunds, 1972; Goodhill, 1972). A perilymphatic fistula can also occur during descent in a plane, probably through a similar mechanism and has been reported to occur in children who have pre-existing congenital malformations of the middle/inner ear (Grundfast and Bluestone, 1978; Supance and Bluestone, 1983). Patients may develop sensorineural hearing loss of sudden onset or vertigo, or both.

Alternobaric Vertigo. This is the term used to describe the vertigo that can develop during diving due to asymmetric middle ear pressure equilibration during descent; the vestibular end-organ is stimulated by the pressures (Lundgren, 1965). Transient facial paralysis has also been described following failure to equilibrate middle ear pressures while flying and diving (Becker, 1983).

Prevention and Management

The most effective method to avoid otitic barotrauma is to advise patients in whom eustachian tube function is chronically poor not to participate in sports diving or flying; a thorough search for underlying pathologic conditions of the eustachian tube system should also be conducted. The same advice should be given to patients who have marginal eustachian tube function, or even normal function, during periods when an upper respiratory tract infection or when allergy is present. However, many patients will not or cannot adhere to these recommendations; thus the physician should counsel them as to the most effective methods to prevent barotrauma when flying or diving. The patient should be taught to use the Valsalva method of autoinflation of the middle ear during descent, or one of the modifications of the procedure, such as to swallow after building up positive nasopharyngeal pressure, as described in the following section. Effective treatment of superimposed upper respiratory inflammation, if present, should also be a goal. Antihistamines, which have a sedative effect, should be withheld if the patient is planning to pilot his plane or if he is going to be involved in sports diving. Divers should be advised to equilibrate middle ear pressure more frequently during descent, which should be at a more gradual rate than usual. Divers should also be cautioned that if they descend to a level at which they have great difficulty in equilibrating pressure, the dive should be aborted. The use of systemic decongestants prior to flying, or the application of a nasal spray prior to descent in a plane or before diving, can often aid equilibration by swallowing or by Valsalva's maneuver; patients who cannot perform the latter even after using a nasal decongestant, should be advised against participating in these activities. For the patient who has transient eustachian tube obstruction and for whom flying is necessary, a preflight myringotomy is the most effective means of preventing barotrauma;

myringotomy and insertion of a tympanostomy tube may be required for patients who have chronic tubal obstruction and who must frequently fly.

Management of middle ear barotrauma is aimed at reestablishing normal middle ear pressure. Following flying, systemic or topical decongestants may be helpful if the condition is mild. Eustachian tube catheterization or Politzer's technique may be required as an office procedure if the patient has a middle ear effusion. However, the most effective method to alleviate the symptoms and allow the eustachian tube to return to normal function is by myringotomy; an occasional patient may require a tympanostomy tube to be inserted if the course becomes protracted. For divers who develop middle ear barotrauma, rest, cessation of diving until resolution, and decongestant and antimicrobial therapy if infection is suspected may be sufficient (Edmunds et al, 1973). Management of a perilymphatic fistula that occurs during flying or diving consists of immediate exploratory tympanotomy and repair of the leak with a muscle graft. Following such an event, further diving should be prohibited. When flying is the cause, this activity should be done only in aircraft with pressurized cabins; a tympanostomy tube should be inserted in those who must continue to fly.

Inflation of the Eustachian Tube

Procedures that force air through the eustachian tube and into the middle ear and mastoid cavities have been employed for over one hundred years in an effort to normalize negative tympanic pressure and eliminate middle ear effusion. The methods of Valsalva and Politzer are the most commonly used in children (Politzer, 1909; Valsalva, 1949). Catheterization of the eustachian tube has also been utilized but is of limited usefulness in children because the procedure can be frightening and is technically difficult to perform in young patients. All three of these methods are also crude tests of eustachian tube patency and have been described in detail in Chapter 6.

From a physiologic standpoint, inflation of the eustachian tube, middle ear, and mastoid has merit. Figure shows the flask model of the nasopharynx-eustachian tube-middle ear system. Liquid is shown in the body and narrow neck of an inverted flask. Relative negative pressure inside the body of the flask prevents the flow of the liquid out of the flask. This is analogous to an effusion in a middle ear that has abnormally high negative pressure. If air is insufflated up into the liquid, through the neck and into the body of the flask, the negative pressure is converted to ambient or positive pressure and the liquid will flow out of the flask. However, if the liquid is of high viscosity, the likelihood of air being forced through the liquid into the body of the flask is remote, especially if the thick liquid completely fills the chamber. Therefore, in the human system a thin, or serous, effusion would be more likely to flow out of the middle ear and down the eustachian tube than would a thick, mucoid effusion that fills the middle ear and mastoid cavities. The method probably is not effective in maintaining normal middle ear pressure in children who have atelectasis caused by eustachian tube obstruction (ie, high negative pressure), since experiments in animals have shown inflation of the middle ear not to be effective (Cantekin et al, 1980b).

Theoretically, then, inflation of the eustachian tube and middle ear should be an effective treatment option for children with certain types of otitis media with effusion or atelectasis or both; however, in reality, there are several problems with this method of management. The self-inflation method of Valsalva is somewhat difficult for children to learn,

since it is a technique involving forced nasal expiration with the nose and lips closed. Cantekin and associates (1976) tested 66 children between the ages of 2 and 6 years who had chronic or recurrent otitis media with effusion and who had functioning tympanostomy tubes in place. They asked each subject to try to blow his or her nose with the glottis closed. None of these children could passively open their eustachian tubes and force air into the middle ear by the Valsalva method, even though they developed a maximum nasopharyngeal pressure of 538.8 ± 237.0 mm H_2O. It was concluded that the Valsalva method of opening the eustachian tube in this age group was not successful, owing to possible tubal compliance problems. Unfortunately, children in this age group have a high incidence of otitis media; for infants, who have the highest incidence of otitis media, the procedure cannot be used at all.

Politzer's method of opening the eustachian tube involves inserting the tip of a rubber air bulb into one nostril while the other nostril is compressed by finger pressure and then asking the child to swallow while the rubber bulb is compressed. Some children complain of a sudden "pop" in the ear as the positive pressure is forced up the eustachian tube and have discomfort with the procedure. This method also is extremely difficult to perform in infants.

The major difficulty with both methods is determining whether the middle ear is actually inflated by the procedure. If a child hears a "pop" or has a pressure sensation in the ear, there is only presumptive evidence of passage of air into the middle ear. Auscultation of the ear (listening for the sound of air entering the middle ear during the procedure) is helpful in determining whether or not the procedure is successful, but a sound may be heard even when air does not enter the middle ear. Objective otoscopic evidence that the middle ear is actually inflated would be constituted by the presence of bubbles or a fluid level behind the tympanic membrane when these findings were not present prior to inflation. Another excellent method for determining objectively if the inflation is successful is to obtain a tympanogram before and after the procedure: the compliance peak should shift toward or be in the positive pressure zone after inflation. Another method to inflate the eustachian tube is to catheterize from the nasopharyngeal end. This technique can be useful in adults but is difficult to perform in children. If none of the results of these presumptive or objective methods of determining the success of inflation is definitive, then the clinician cannot be certain that the procedure has been therapeutic. Failure to achieve a successful result may be related to (1) inability of the patient to learn the method, (2) insufficient nasopharyngeal overpressure to open the eustachian tube passively, (3) eustachian tube abnormality, or (4) a middle ear filled with a very thick, mucoid effusion.

Unfortunately, the beneficial effect of the Valsalva and Politzer methods of inflation for treatment of otitis media with effusion or atelectasis has not been subjected to any acceptable randomized, controlled trials, and most of the evidence has been anecdotal. Gottschalk (1966, 1980) described remarkable success with a modification of the Politzer method in over 12.000 patients; the average course of treatment was a minimum of 12 inflations in the office on 3 separate days. Schwartz and colleagues (1978) have shown that it is possible to inflate the middle ears of children at home by the Politzer method; they documented the results of the method by tympanometry but did not test its efficacy. The only controlled trial of this method was reported by Fraser and coworkers (1977), and they were not able to demonstrate that it was efficacious.

Until well-controlled clinical trials are reported, it would appear reasonable to use the Politzer method of inflating the middle ear for the following conditions. Barotrauma (following flying or swimming) should respond ideally to the Politzer procedure if atelectasis with high negative pressure or otitis media with effusion or both are present. Inflation of the middle ear should be helpful under these circumstances, since this condition is usually not due to chronic eustachian tube dysfunction, and inflation may resolve the acute, subacute, or chronic disorder rapidly. When a middle ear effusion not due to barotrauma is found in a patient who only occasionally has a problem and in whom frequently recurrent or chronic disease is not suspected, then the procedure may also be successful, especially if a small amount of serous effusion is visible behind a translucent tympanic membrane. However, it is unlikely that a mucoid or purulent effusion could be evacuated by this technique, and if it could be, it would probably recur immediately after the procedure. Atelectasis of the tympanic membrane and middle ear, with or without high negative pressure, can also be treated by repeated autoinflation (Valsalva) or the Politzer method; however, even if the middle ear is successfully inflated, the benefit is usually only of short duration and the procedure must be repeated frequently. Therefore, it is unlikely that inflation will be successul in alleviating for any length of time frequently recurrent or chronic eustachian tube dysfunction. There is also a remote possibility that bacteria can be forced into the middle ear from the nasopharynx during this procedure.

In conclusion, these procedures may be worthwhile for children with barotitis and for children who have an occasional episode of otitis media with effusion or atelectasis, but they are probably not helpful in children who have chronic or frequently recurrent middle ear effusion or atelectasis, or both.

Acute Otitis Media

The rapid and short onset of signs and symptoms of inflammation in the middle ear is termed acute otitis media. Synonyms such as acute suppurative or purulent otitis media are acceptable. One or more of the following are present: otalgia (or pulling of the ear in the young infant), fever, and the recent onset of irritability. The tympanic membrane is full or bulging, is opaque, and has limited or no mobility to pneumatic otoscopy - indicative of a middle ear effusion. Erythema of the eardrum is an inconsistent finding. The acute onset of ear pain, fever, and a purulent discharge (otorrhea) through a perforation of the tympanic membrane (or tympanostomy tube) also would be evidence of acute otitis media. As previously described, bacteria that commonly cause acute otitis media can gain access to the middle ear from the nasopharynx through the eustachian tube by reflux, aspiration, or insufflation of the organisms. *S. pneumoniae* was cultured from approximately 30 per cent of the effusions and is the most common causative agent in all age groups. *H. influenzae* caused about 20 per cent of the ear infections. In the past, the incidence of *B. catarrhalis* has been about 5 per cent but the incidence is now 12 per cent. However, in a separate report by Kovatch and colleagues (1983), this incidence was found to be 22 per cent of 146 infants and children from one private practice pediatric group in suburban Pittsburgh. Shurin and coworkers (1983) also reported the incidence of this bacterium to be 27 per cent in Cleveland between 1980 and 1982, which was significantly greater than the 6 per cent recovered the year before. We found the incidence of Group A beta-hemolytic streptococcus to account for 3 per cent; *S. aureus* was present in less than 2 per cent. Anaerobic bacteria and viruses have been cultured infrequently from middle ear aspirates of children who have acute otitis media.

The percentage of *H. influenzae* that were beta-lactamase producing was about 30 per cent in the Pittsburgh study; this percentage has increased over the last several years. About 80 per cent of *B. catarrhalis* strains produced beta-lactamase. Shurin and colleagues (1983) reported that 77 per cent of their strains produced beta-lactamase. This change in the incidence of the pathogens and the emergence of beta-lactamase-producing organisms has an important impact on management of acute otitis media today.

Management

Most experts agree that infangts and children who have signs and symptoms of acute otitis media should receive antimicrobial therapy; however, several investigators has questioned the need for antimicrobial agents in all cases (Laxdal et al, 1970; Diamant and Diamant, 1974; Mygind et al, 1981; Van Buchem et al, 1981). Since the rate of suppurative complications has decreased in the antibiotic era (Sorenson, 1977), antimicrobial therapy is still the treatment of choice (Howie and Ploussard, 1969).

Amoxicillin and ampicillin are the currently preferred drugs for initial treatment of acute otitis media because they are active both in vitro and in vivo against *S. pneumoniae* and most strains of *H. influenzae* and they are relatively inexpensive in the USA (Bluestone, 1987). Amoxicillin is the most commonly prescribed drug, since it can be given in three divided doses and has fewer side effects such as diarrhea. Treatment for 10 days is recommended; this duration of therapy has not been determined by clinical trials but has been reasoned from clinical trials involving children with pharyngitis. If a child is allergic to the penicillins, then trimethoprim-sulfamethoxazole or a combination of oral erythromycin and sulfisoxazole is advocated. If beta-lactamase producing *H. influenzae* or *B. catarrhalis* organisms are isolated by tympanocentesis or from otorrhea, then the choices also would be these combinations, cefaclor, the combination of amoxicillin-clavulanate, cefuroxime axetil, or cefixime.

With appropriate antimicrobial therapy, most children with acute bacterial otitis media are significantly improved within 48 to 72 hours. Persistent or recurrent pain or fever during treatment would signal the need for tymanocentesis/myringotomy or selection of another antimicrobial agent or both. If a middle ear aspirate is not obtained for culture and susceptibility testing, the agent chosen should be effective against whatever resistant bacteria have been found to be in the community that have been associated with symptomatic treatment failures. This rate has been almost 30 per cent in our community, but it appears to vary widely with age, season, and geographic location.

The new antimicrobial agent should be effective against beta-lactamase-producing bacteria such as *H. influenzae* and *B. catarrhalis.* If ampicillin or amoxicillin was initially given, then the combination of erythromycin-sulfisoxazole or trimethoprim-sulfamethoxazole should be effective when these organisms are present. However, trimethoprim-sulfamethoxazole apparently is not effective when *Streptococcus pyogenes* is the causative organism (this drug combination is not indicated if there is an associated streptococcal pharyngitis). In addition, both these drug combinations contain a sulfonamide, which has a high rate of adverse side effects such as vasculitis. Cefaclor is not effective against some strains of beta-lactamase-producing *B. catarrhalis;* it has been associated with a serum-

sickness-like reaction. Amoxillin-clavulanate, cefuroxime axetil, or cefixime would be other choices at this stage.

All children should be reexamined at the end of the course of antibiotic therapy (ie, after 10 to 14 days). At this time, some children will have a persistent middle ear effusion; this proportion is approximately 50 per cent of those treated with antibiotics. Since the presence of a persistent middle ear effusion after a 10-day trial of an antimicrobial agent is common, this finding alone is not sufficient grounds for performing surgery such as a myringotomy and tympanostomy tube insertion.

Additional supportive therapy, including analgesics, antipyretics, and local heat, will usually be helpful. An oral decongestant, such as pseudoephedrine hydrochloride, may relieve nasal congestion, and antihistamines may help patients with known or suspected nasal allergy. However, the efficacy of antihistamines and decongestants in the treatment of acute otitis media has not been proven. Olson and coworkers (1978) failed to show efficacy of an oral decongestant when administered in conjunction with an antibiotic for children with acute otitis media. 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.

If the middle ear fluid is persistent after the initial 10 days of antimicrobial therapy, one or more of the following treatment options have been advocated to hasten the resolution of the effusion during the next, subacute phase:

1. A course of an antimicrobial agent different from the initial one based on the assumption that if a resistant organism is present the new antimicrobial agent may be effective.

2. A topical or systemic nasal decongestant or antihistamine, or a combination of these drugs.

3. Eustachian tube-middle ear inflation employing the method of Valsalva or Politzer.

4. Tympanocentesis or myringotomy or both for culture of the aspirate and draining of the middle ear.

Unfortunately, none of these commonly employed methods has been shown to be effective in randomized, controlled trials of children with subacute otitis media with effusion. In fact, Cantekin and colleagues (1983) showed a lack of efficacy of a combination of an oral decongestant and antihistamine in eliminating persistent middle-ear effusion in a study that involved 553 infants and children. At present, many clinicians do not treat children who have asymptomatic (except for hearing loss) otitis media with effusion still present after 2 weeks, and they reexamine the child 6 weeks later (ie, 2 months after the initial visit). At this time, most patients usually have a middle ear that is effusion-free. However, treatment with another antimicrobial, such as cefaclor, trimethoprim-sulfamethoxazole, erythromycin-sulfisoxazole, or amoxicillin-clavulanate K, which are effective against possible resistant bacteria, is becoming increasingly popular, especially if the child has any signs or symptoms of persistent

infection, such as otalgia, or if such organisms have been isolated from subacute effusions in the community.

Schwartz and associates (1981) studied children whose clinical signs did not resolve after initial therapy of a 10-day course of ampicillin, amoxicillin, or erythromycin-sulfonamide combination. Middle ear effusion was aspirated and cultured for bacteria; ampicillin-resistant *H. influenzae* was found in 31 per cent, ampicillin-susceptible strains of *S. pneumoniae* or *H. influenzae* were identified in 51 per cent, and no bacterial growth was found in the other effusions. Teele and colleagues (1981) also studied children who failed to respond to therapy and noted the following results: 19 per cent had organisms resistant to initial therapy, and 57 per cent had no bacteria isolated from the middle ear effusions. Thus, although some children who fail clinically do so because of a bacterial pathogen resistant to initial therapy, many children have bacteria that are susceptible to the drug and some have negative bacterial cultures and presumably have a nonbacterial microorganism as the cause of otitis media or some other reason for the persistent fever, such as persistent sinusitis.

If the child still has otitis media with effusion after 2 to 3 months, the effusion is chronic and should be treated as described previously under the heading "Otitis Media with Effusion".

The method of management of acute otitis media may vary with the age of the patient. Acute otitis media during the neonatal period may warrant more aggressive management than such a condition in an older child. Bland (1972) reported that otitis media in neonates was frequently caused by an unusual organism compared with those that usually cause such problems in older infants and in children (ie, gram-negative bacilli or *Staphylococcus aurueus*). Following this report, many authorities advocated treating these babies in the hospital according to protocols for neonatal sepsis, since the infection could be life-threatening. However, since then other investigators (Shurin et al, 1978; Tetzlaff et al, 1977) have shown that the incidence of these unusual organisms is relatively low, especially in neonates who were apparently well when discharged from the hospital following birth and then developed an acute otitis media while at home. For these neonates, the acute otitis media should be treated as described above for older infants and children. However, if the neonate appears to be severely ill and toxic, hospitalization and tympanocentesis and possibly a myringotomy are indicated. If the baby is still hospitalized following delivery and develops an otitis media, tympanocentesis and myringotomy are indicated, since infection at this time can be life-threatening. Culture of the middle ear aspirate may reveal an unusual organism that necessitates treatment with an antimicrobial agent different from the antibiotics recommended for treatment of acute otitis media in older children.

The management of acute otitis media may be different in certain infants and children whose underlying condition is known to be associated with otitis caused by an unusual organism. Such children are primarily those who are immunologically compromised. Tympanocentesis possibly followed by a myringotomy is indicated in an effort to identify the causative organisms and to promote drainage.

Recurrent Acute Otitis Media

It is not uncommon for an infant to have recurrent bouts of acute otitis media. Some children develop 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 may have persistent middle ear effusion and suffer recurrent episodes of acute otitis media superimposed on the chronic disorder. The child with recurrent acute otitis media who completely clears between episodes may be managed as previously outlined. The bacteriology of middle ear infection in children who have recurrent episodes of acute otitis media is similar to that found in first episodes; the predominant organisms are *S. pneumoniae* (though of different serotypes) and nontypable strains of *H. influenzae.* Thus, the child with a recurrent episode of otitis media should be treated initially with the same antimicrobial regimens as the child with a first episode of middle ear infection.

However, if the bouts are frequent and close together, prevention of further attacks is desirable. The patient requires further evaluation. Several avenues of investigation are open: a search for respiratory allergy may prove fruitful; roentgenograms of the paranasal sinuses may reveal sinusitis; immunologic studies may be of value if other organs are involved (the lung, for example). In addition, more thorough physical examination may reveal abnormalities, such as submucous cleft palate or a tumor of the nasopharynx, that require definitive management. If none of the above conditions is present, then one or more of the popular methods of prevention may be attempted; however, the efficacy of these various modalities has yet to be proven in acceptable clinical trials. For infants and children who have frequent episodes (such as three or more episodes within the preceding 6 months) of acute otitis media without middle ear effusion in between the bouts, the most common nonsurgical and surgical methods currently employed for prevention are (1) chemoprophylaxis with an antimicrobial agent(s), (2) myringotomy with insertion of tympanostomy tube, and (3) adenoidectomy with or without tonsillectomy. Administration of polyvalent pneumococcal vaccine is not currently recommended as a preventive measure but may be helpful in children above the age of 2 years.

Chemoprophylaxis implies use of drugs in anticipation of infection, whereas treatment implies use of drugs after infection has taken place or signs of infectious disease are evident. Although indiscriminate use of antimicrobial agents of prophylaxis is to be avoided, many forms of chemoprophylaxis have been extensively tested and are of proven value. Recent studies suggest that chemoprophylaxis may be effective in children with recurrent episodes of acute otitis media.

An antimicrobial agent of value for prophylaxis should be effective for the bacteria most likely to cause disease, should be of limited toxicity and have few side effects and should be unlikely to decrease in efficacy with prolonged usage (the bacteria should not develop resistance to the drug). The potential liabilities of chemoprophylaxis include alteration of the patient's microflora, allergic or toxic reactions, and relaxation of the physician's watchfulness for the occurrence of disease in the patient.

Children are at risk for recurrent episodes of acute otitis media during a relatively short period of life; most episodes occur between 6 and 36 months of age, and usually only during the winter and early spring. If the child who is susceptible to recurrent otitis media

could be protected from infection during this period, the morbidity of middle ear disease might be avoided. Thus, the concept of prophylaxis is a worthy one and may be helpful when used appropriately by physicians who care for children.

Two double-blind controlled trials of chemoprophylaxis in children with acute otitis media have been reported: Maynard and colleagues (1972) studied Alaskan Eskimo children, and Perrin and coworkers (1974) studied children in Rochester, New York. In the first study, ampicillin or a placebo was administered for 1 year to children under 7 years of age living in Alaskan Eskimo villages. The children received a daily dose of oral ampicillin - 125 mg for those up to 2 and one-half years of age, and 150 mg for older children. Otitis media was defined as a new episode of otorrhea by history or observation made by a research nurse who visited the villages monthly. The incidence of otorrhea was reduced by approximately 50 per cent in the 173 children receiving ampicillin, compared with the 191 children who received a placebo.

In the study by Perrin and colleagues (1974), sulfisoxazole or a placebo was administered to 54 children in Rochester, New York, who were 11 months to 8 years of age and had histories of recurrent episodes of acute otitis media (three or more episodes in the previous 18 months, or more than five episodes total). The children received a placebo or 500 mg of sulfisoxazole twice a day for 3 months. They were then switched to the alternate regimen for another 3-month period. No specific criteria for otitis media were used by the participating pediatricians (a four-physician group practice in suburban Rochester). A significant decrease in new episodes of acute otitis media occurred in the group of children receiving the antimicrobial agent. The older children, 6 to 8 years of age, showed a minimal or insignificant decrease in incidence of otitis media when on the prophylactic regimen. A similar study was reported by Liston and associates (1983) in which the results confirmed those of Perrin.

Biedel (1978) evaluated the effectiveness of sulfonamides (sulfisoxazole or trisulfpyrimidine in a dosage of 100 to 130 mg/kg/24 hours, or sulfamethoxazole in a dosage of 55 mg/kg/24 hours) used at the onset of signs of infection of the upper respiratory tract. Children were enrolled in the program after receovery from a recent acute episode of otitis media. They were placed alternately into treatment (sulfonamide) or control (decongestant) groups when parents called the physician and reported any sign of a new upper respiratory tract infection. Treatment was prescribed for a minimum of 6 days and any new episodes of otitis media that occurred during the 8 weeks following recovery from the original episode of otitis media were recorded. Otitis media was found to recur with a new upper respiratory infection more frequently in children receiving decongestants than in children receiving sulfonamide.

Although the data from these trials are persuasive, additional studies of more prolonged courses of antimicrobial prophylaxis are needed. Future studies must determine whether the antimicrobial agents decrease the duration of effusion in the middle ear as well as decrease the signs of otitis media and whether prolonged courses will result in significant side effects or development of resistant bacteria.

Although the studies are inadequate to provide conclusive evidence of the value of chemoprophylaxis, the results are impressive. It seems reasonable, at present, to use

amoxicillin, 20 mg/kg in one dose (given at bedtime) or, if the child is allergic to the penicillins, a daily dose of sulfonamide, 50 mg/kg, for the child who has repeated episodes of otitis media, such as three episodes in 6 months or four to five in 12 months with at least one episode being present during the preceding 6 months; this prophylactic regimen should be continued for about 6 months. (Note: trimethoprim-sulfamethoxazole is not indicated for prophylaxis of otitis media in children.) The child should be examined at frequent and regular intervals (every 1 or 2 months) to be certain that inapparent middle ear effusion does not occur. If a decrease in signs of acute disease occurs but there is persistence of middle ear effusion, surgical intervention should be considered, such as myringotomy and tympanostomy tube insertion. Parental and patient compliance in giving and taking the medication daily is a consideration, as are the potential risks of drug toxicity and possible emergence of resistant organisms while on the medication. Since the studies reported in the past have shown that some children still have recurrent episodes of acute otitis media despite the preventive dose of the antibiotic, myringotomy and tympanostomy tube placement should be considered for patients who are prophylaxis failures. In addition, surgery also may be required if signs and symptoms of eustachian tube dysfunction persist, such as pain, fluctuating hearing loss, and vertigo, or if severe atelectasis develops.

At present, there is no evidence that a topical or systemic nasal decongestant or antihistamine, either alone or in combination, administered daily or at the onset of an upper respiratory tract infection, prevents recurrent acute otitis media. Therefore, the use of such medications for prophylaxis is not recommended until their efficacy is proved.

It has been suggested that a myringotomy performed at the initial onset of an attack of acute otitis media would reduce the recurrence rate of this problem; however, this was not shown in a study by Lorentzen and Haugsten (1977). At present, myringotomy as the initial treatment for acute otitis media remains optional and should only be considered in selected patients. Nevertheless, myringotomy with insertion of tympanostomy tubes is commonly performed to prevent recurrent episodes of acute otitis media. The procedure is usually performed after the signs and symptoms of the acute otitis media have resolved but may be performed during an acute episode if persistent otalgia or fever or both are present in a child who has had frequently recurrent episodes. A prospective randomized study by Gebhart (1981) was conducted in 95 children who had multiple episodes of acute otitis media. Comparison of infection rates was made between patients treated with conventional antibiotic therapy for each episode and patients who had tympanostomy tubes placed. Placement of tympanostomy tubes significantly decreased the number of episodes of acute purulent otitis media during a 6-month observation period. However, children with and without middle ear effusions were entered into the study.

In some children, insertion of tympanostomy tubes will prevent the severe symptoms of acute otitis media but recurrent episodes of otorrhea will occur. Systemic antimicrobial agents or ototopical antibiotic-cortisone medication, or both, will usually be effective in resolving the otitis media. However, caution is advised in prescribing ototopical agents, due to potential ototoxicity.

Adenoidectomy with or without tonsillectomy is frequently advocated for the prevention of recurrent acute otitis media, but the randomized, controlled studies reported in the past have not proved the efficacy of these procedures for this condition. However,

adenoidectomy with or without tonsillectomy may reduce recurrent otitis media in some children; candidates would include, among others, those who have had frequently recurrent tonsillitis/adenoiditis or upper airway obstruction or both.

In summary, the parents of a child who has frequently recurrent episodes of acute otitis media in whom the effusion appears to clear between bouts should be offered the following management options: (1) antimicrobial treatment of each episode, (2) antimicrobial prophylaxis, or (3) myringotomy and tympanostomy tube insertion. The treatment option selected should involve the parents and possibly the child (if old enough) in the decision-making process. A few parents choose to watch and wait, usually if the episodes have been mild or relatively infrequent. At present, the decision should be between administering an antibiotic in a prophylactic dose or a myringotomy and insertion of a tympanostomy tube. Since neither of these two procedures has been shown to be superior to the other, or even to watchful waiting, the decision should be based upon the parents' (and child's) willingness to have the child take daily medication as a preventive measure or to have surgery performed on the child's ear, which involves the administration of general anesthetic. The possibility of an adverse reaction occurring with either method should be discussed fully with the family. Usually a decision in favor of one of the treatment options is arrived at by this method, since some parents are unwilling to give a daily antibiotic or are concerned about the possible side effects of long-term antibiotic treatment; oter parents are concerned about the possible complications and sequelae of tympanostomy tube insertion or complications of a general anesthetic. If the parents are undecided, then a trial of antimicrobial prophylaxis can be offered with the option to perform a myringotomy and tympanostomy tube insertion if the chemoprophylaxis fails to prevent recurrent otitis media or the signs and symptoms of eustachian tube dysfunction persists.

For the rarely encountered child in whom tympanostomy tubes fail to prevent frequently recurrent acute otitis media (ie, otorrhea through the tube), the combination of both antimicrobial prophylaxis and tympanostomy tubes is usually effective in preventing recurrent episodes.

The above management options should only be offered to those children in whom chronic middle ear effusion is not present between episodes. If recurrent bouts of acute otitis media are superimposed on the chronic condition, the child should be treated as described below for management of chronic otitis media with effusion. (See also Chapter 28, "Acute Otitis Media and Mastoiditis.")

Chronic Suppurative Otitis Media

Chronic suppurative otitis media is a state of ear disease in which there is chronic infection of the middle ear and mastoid and in which a "central" perforation of the tympanic membrane (or a patent tympanostomy tube) and discharge (otorrhea) are present. Mastoiditis is invariably a part of the pathologic process. The condition has been called chronic otitis media, but this term can be confused with chronic otitis media with effusion, in which no perforation is present. It is also called chronic suppurative otitis media and mastoiditis, chronic purulent otitis media, and chronic otomastoiditis. The most descriptive term is chronic otitis media with perforation, discharge, and mastoiditis, but this is not common usage. When a cholesteatoma is also present, the term chronic suppurative otitis media with cholesteatoma

is used; however, an acquired aural cholesteatoma does not have to be associated with chronic suppurative otitis media.

Chronic suppurative otitis media develops from a chronic bacterial infection. The middle ear and mastoid air-cells no longer have a protective air cushion to prevent nasopharyngeal secretions from entering the ear, which can result in reflux otitis media. In addition, the open tympanic membrane can permit contaminated water to enter the middle ear during bathing and swimming. The bacteria that caused the initial episode of acute otitis media with perforation, or through a tympanostomy tube, are usually not those that are isolated from the chronic discharge when there is chronic infection in the middle ear and mastoid. Thus, the antimicrobial therapy recommended for acute otitis media will not be effective in most cases of chronic suppurative otitis media. The microbiology of chronic suppurative otitis media without cholesteatoma has been reported by Kenna and coworkers (1986). Of the 51 ear cultures obtained from the 36 children, 23 microbiologic species were isolated. One organism was isolated from 18 ears, two from 20 ears, three from three ears, four from four ears, and five from two ears. The most common bacterial species isolated was *Pseumonas aeruginosa,* which was present in 34 ears (67 per cent) and the only isolate in 16 ears (31 per cent). Of the 15 children who had bilateral otorrhea, 11 (73 per cent) had the same organism(s) identified in both middle ears: seven children had *P. aeruginosa* isolated, four had *S. aureus,* and one each had *S. epidermidis, Candida albicans,* and diphtheroids.

The bacteriology of chronic suppurative otitis media with cholesteatoma has been reported (Harker and Koontz, 1977; Brook, 1981). The most common aerobic microbiologic organisms isolated were *P. aeruginosa* and *S. aureus,* and the most frequent anaerobic organisms were *Bacteroides, Peptostreptococcus,* and *Peptococcus* species. It is important to make the distinction between chronic suppurative otitis media with and without cholesteatoma, since tympanomastoid surgery is indicated when cholesteatoma is present, whereas medical management *may* be effective when cholesteatoma is absent.

Management

Medical management of chronic suppurative otitis media without cholesteatoma is directed toward eliminating the infection from the middle ear and mastoid. Since the bacteria most frequently cultured are gram-negative, antimicrobial agents should be selected to be effective against these organisms. A suspension that contains polymyxin B, neomycin and hydrocortisone (Cortisporine) and one that combines neomycin, polymixin E, and hydrocortisone (Coly-Mycin) have been advocated; however, because of concern about the potential ototoxicity of these agents, caution is advised (Brummett et al, 1976; Meyerhoff et al, 1983). Orally administered antibiotics are usually not effective unless an organism which will be susceptible, such as *S. aureus,* is seen on Gram stain or is cultured from the discharge. Oral antibiotics are also effective against the organisms that commonly cause acute otitis media, such as pneumococcus or *H. influenzae.*

If topical antibiotic medication is elected, the child should return to the outpatient facility daily so that the discharge can be thoroughly aspirated. Frequently, the discharge will rapidly improve with this type of treatment within a week or two.

Because of concern over toxicity of the ototopical agents, parents should be informed of this potential danger if these agents are used. As an alternative, we hospitalize our patients and administer a parenteral betalactam antipseudomonal drug such as ticarcillin. The middle ear is aspirated daily. In most children, the middle ear will be free of discharge, and the signs of otitis media will be greatly improved or absent within 7 to 10 days.

When the discharge fails to respond to intensive medical therapy, surgery on the middle ear and mastoid is indicated. In the study by Kenna and coworkers (1986) of 36 pediatric patients with chronic suppurative otitis media, in which all received parenteral antimicrobial therapy and daily aural toilet, 32 (89 per cent) had resolution of their infection with medical therapy alone; four children required tympanomastoidectomy.

If the infection can be eliminated using the methods described previously, prevention of recurrence can be achieved by the following options: (1) prophylactic antimicrobial therapy, (2) removal of the tympanostomy tube, and (3) surgical repair of the tympanic membrane defect. The choice of these options will depend on the age of the child and the status of the function of the eustachian tube.

When chronic suppurative otitis media is present with cholesteatoma, tympanomastoid surgery is indicated. Preoperative antimicrobial therapy and possibly perioperative prophylaxis may be helpful in reducing postoperative infection and will promote better healing.

In some patients, in whom a radical mastoidectomy was performed for extensive cholesteatoma, troublesome otorrhea occurs following surgery. This complication can usually be prevented by closing the middle end of the eustachian tube. The most effective way to obliterate the bony portion of the tube is with bone paté. (See also Chapter 29, "Chronic Otitis Media and Mastoiditis.")