

Chapter 7: Infections of the Ear

Common Pathogens Found in Infections of the Ear

Acute Otitis Media

1. Bacterial pathogens isolated from middle ear fluids (MEF) of children with acute otitis media (AOM) are seen in Table 7-1.

Table 7-1. Pathogens Found in the MEF of AOM

Microorganism	Children with Pathogen (%)
<i>Streptococcus pneumoniae</i>	35
<i>Haemophilus influenzae</i>	20
Streptococcus, group A	8
<i>Branhamella catarrhalis</i>	3
<i>Staphylococcus aureus</i>	2
Gram-negative enteric bacilli	1
Mixed	2
None or nonpathogens	29

2. *Streptococcus pneumoniae* and *H. influenzae* are the most frequently isolated bacteria from middle ear effusion of AOM.

3. The eight most common types of *S. pneumoniae* in order of decreasing frequency are 19, 3, 6, 23, 14, 18, 4, and 7. All are included in a pneumococcal vaccine (Pneumovax) introduced in the USA in February 1978.

4. Otitis media due to *H. influenzae* is associated with non-typeable strains in almost all patients. In approximately 10% the otitis is due to type B. Some children infected with *H. influenzae* type B appear to be very toxic and may have meningitis.

5. Until recently *H. influenzae* was believed to be limited in importance to preschool age children under 5 years of age, but several studies indicate that this organism is a significant cause of otitis media until the age of 10 years and older. Acute otitis media in children through adolescence should be treated with antibiotics known to be effective against *H. influenzae*.

6. Gram-negative enteric bacilli are isolated from the MEF of approximately 20% of infants up to 6 weeks of age, but are rarely present in the MEF of older children.

7. Group A beta-hemolytic streptococci and *S. aureus* are infrequent causes of otitis.

8. Preliminary results suggest that anaerobic bacteria are responsible for some episodes

of OM.

9. Although epidemiologic data suggest that virus infection is associated with OM, the results of ten studies indicate that these agents are infrequently isolated from MEF of children with OM. Viruses were isolated from 29 of 663 patients (4.4%). Respiratory syncytial virus and influenza virus were isolated most frequently.

10. The results of seven studies of mycoplasma infection in 771 patients with OM included only one isolation of *Mycoplasma pneumoniae* from MEF.

11. *Staphylococcus sp.* and *Pseudomonas aeruginosa* isolated in acute otitis media probably represented an overgrowth or contamination of the culture by infection of the external auditory canal.

12. *Chlamydia trachomatis* is the etiologic agent of a mild, but persistent pneumonia in infants that may be accompanied by otitis media.

13. Recent studies of asymptomatic children with persistent MEF indicate that bacterial pathogens are present in some of these fluids. Investigators in Columbus, Boston, and Pittsburgh obtained MEF for culture at the time of myringotomy or placement of tympanostomy tubes. Bacteria were isolated from MEF of 50% of these children; *S. pneumoniae*, *H. influenzae*, or group A streptococci were isolated from 10-20% of the patients. There were only minimal differences in the rates of isolation of bacteria from serous, mucoid, or purulent fluids. The significance of these results is uncertain, but they suggest that persistent effusion may be a result of asymptomatic but prolonged infection, or may be an immune response to a persisting antigen.

14. *Acute necrotizing otitis media* is a virulent form of acute otitis media, nearly always caused by a beta-hemolytic streptococcus, and seen in children who are acutely ill from a systemic infectious disease such as scarlet fever, measles, pneumonia, or influenza. The pathologic process is true necrosis of the soft tissues and bones of the middle ear and mastoid.

The disease is characterized by a profuse, purulent, foul-smelling otorrhea, a large tympanic membrane perforation (in contrast to a small perforation seen in the usual acute suppurative otitis media), sloughing of the annulus tympanicus, portions of ossicles, the scutum, and the mastoid air cells. Occasionally, naked white bone of the promontory and the antrum may be observed.

15. *Acute mastoiditis* is most likely caused by *S. pneumoniae*, but *Streptococcus pyogenes* and *Staphylococcus aureus* are almost as frequent pathogens. Surprisingly, mastoiditis is rarely due to *H. influenzae*, which causes mucous membrane infections, but may be less invasive to bone.

16. Most culture studies utilize *tympanocentesis* as the method of sampling infected material. However, Schwartz et al showed a high correlation between culture results of tympanocentesis and deep nasopharyngeal samplings (77% for a haemophilus and 84% for

pneumococcus) when: (a) the culture was taken deep and vigorously in the nasopharyngeal-adenoidal area, and (b) the material was promptly plated on sheep blood and chocolate agar plates without its being passed through a transport broth (which decreases accuracy by allowing overgrowth of nonpathogens).

17. *Indications for myringotomy in AOM* include: (a) severe pain, (b) failure of initial antibiotic therapy, (c) an immunologically compromised child, and (d) complicated AOM.

Otitis Media with Effusion (Serous Otitis Media)

Otitis media with effusion (OME) continues to be the leading cause of hearing loss in childhood. Recent studies have shown that more than 30% of all children have had three or more episodes of the disorder by their second birthday.

Synonyms for this condition include *secretory otitis*, *glue ear*, *chronic catarrh otitis*, and *nonsuppurative otitis*.

1. A careful and precise otoscopic examination with the use of the pneumatic otoscope is essential for correct diagnosis. The use of tuning forks, tympanometry, audiometry, and the operating microscope will aid and confirm the diagnosis. Recently the use of brain-stem evoked response audiometry has been proposed to differentiate conductive from sensorineural hearing loss. It appears that there is an increase in the latency of wave I in patients with middle ear effusion.

2. *Nonoperative therapy* includes the use of (a) antihistamines and decongestants, (b) corticosteroids, (c) antibiotics, (d) autoinflation (Valsalva's maneuver), (e) politzerization and (f) control of etiologic factors (nasal infection, sinusitis, allergy, etc).

3. The use of *pressure equalizing (PE)* or *ventilating tubes* has become the most widely accepted therapeutic modality for the treatment of otitis media with effusion. Ever since the reintroduction of this treatment in 1954 by Armstrong, the placement of a PE tube through the tympanic membrane has gained worldwide acceptance and is rapidly becoming the most commonly performed surgical procedure in the United States.

4. Approximately 80% of intubated patients respond after one insertion and require no further therapy. Approximately 20% of the patients with OME will require reinsertion of PE tubes on two or more occasions.

5. The overall *complication* rate after PE tube intubation is about 11%. These include:

- a. Persistent otorrhea (5-15%).
- b. Persistent perforation after tube extrusion.
- c. Scarring or atrophic membrane formation.
- d. Granuloma of TM or EAC.

- e. Tympanosclerosis.
- f. Cholesteatoma (by ingrowth of the surface epithelium).
- g. Ossicular disruption.
- h. Sensorineural hearing loss.

6. *Indications* for insertion of PE tubes for OME are:

- a. Persistent effusion for more than 12 weeks.
- b. Failure to respond to 4-week antibiotic-antihistamine-decongestant therapy.
- c. Severe otitis media with considerable atelectasis or retraction pocket.
- d. Severe conductive deafness.
- e. Impending cholesteatoma.
- f. "Otitis media prone" child.
- g. Cleft palate.

7. Placement of PE tubes significantly decreased the number of episodes of acute purulent OM and was shown to be an effective method of prophylaxis in the otitis prone child.

Immunoglobulin levels in the middle ear in this group of patients were found to be similar to those levels in patients with otitis media with effusion. This supports the hypothesis that a dynamic relationship between the different clinical entities of otitis media exists.

8. *Adenoidectomy* with or without tonsillectomy: Advocates for adenoidectomy argue that hypertrophic adenoids produce a mechanical obstruction of the eustachian tube orifice leading to diminished middle ear ventilation. Others believe that the adenoids and occasionally the tonsils may harbor microorganisms that ascend through the eustachian tube, establishing an inflammatory process and a middle ear effusion. Unfortunately the controlled studies necessary to prove or disprove such hypotheses are lacking.

According to recent studies, adenoidectomy had no additional beneficial effect on the cure rate obtained by tympanostomy. Data thus far collected by Paradise et al are not sufficient to reach a conclusion for or against the efficacy of adenoidectomy for otitis media, but it is apparent that adenoidectomy by no means eliminates the problem.

9. The successful use of *simple mastoidectomy* has been reported by Holmquist et al. In most ears with middle ear effusion the mastoid air cells may be involved as well. The mucosal changes and the secretion extends into all cavities of the ear including the mastoid air cells. In most ears insertion of a ventilating tube through the tympanic membrane is

adequate for proper aeration of the middle ear as well as of the mastoid air cell system. In a small percentage of cases, the ear will continue to drain and the mastoid will not clear up. Antibiotics and decongestants fail to cure the ear. In such an event, simple mastoidectomy is indicated. Cholesterol granuloma and/or cholesteatoma may be found.

10. Children with conductive hearing loss due to OME may be at risk for language delays that have been suspected of causing later language problems. After a 3-months duration of serous otitis, a language evaluation should be given the infant or child. Any language delay should be promptly treated with a home language stimulation program and, in more extreme cases, there should be a consideration of hearing aid use.

11. Draf reported that 61% of x-ray controlled patients with OME had a pathologic condition of the paranasal sinuses. In persistent cases of OME, the nose and sinuses should be evaluated.

Chronic Otitis Media

1. The bacterial flora found in chronic otitis media varies considerably. The predominating organisms are usually gram-negative bacilli.

2. Chronic suppurative otitis media with tympanic membrane perforation or cholesteatoma, is usually attributed to the aerobic *P. aeruginosa* and *S. aureus* organisms, but *proteus sp.* and *Escherichia coli* are also frequently isolated.

Draining ears, especially if cholesteatoma (keratoma) is present, often produce a foul-smelling pus which is characteristic of anaerobic streptococcal infections. From two-thirds of infected cholesteatomas various anaerobes can be recovered. Most chronic ear drainage results from mixed infections with both aerobic and anaerobic pathogens. 13% of anaerobes are *Bacteroides fragilis*.

3. *Tuberculous otitis media* is a rare type of infection caused by acid-fast tuberculous bacilli (*Mycobacterium tuberculosis*), characterized by an insidious and painless onset; scanty thin odorless discharge; progressive enlargement of the perforation in the pars tensa; *multiple perforations* with pale granulations; and hearing loss out of proportion to other symptoms. It is usually secondary to pulmonary tuberculosis. Early investigators believed that the portal of entry was the eustachian tube, while others felt that the infection was spread by the hematogenous route. Any caseous focus may be a source from which tubercle bacilli enter the bloodstream to reach the temporal bone. Histologically, it is characterized by (a) extensive edema and infiltration of the mucosa and tympanic membrane by round cells and giant cells, (b) formation of numerous tubercles consisting of epithelioid and lymphoid cells and containing characteristic giant cells of the Langerhans type, (c) caseation and ulceration.

Diagnosis may be made from direct smears, cultures, and histologic examination of granulation tissue removed from the middle ear or mastoid. Tuberculosis should be suspected when otitis media does not respond readily to ordinary methods of therapy.

Isoniazid and PAS (para-aminosalicylic acid) are commonly used for initial treatment. Streptomycin may be added in more severe cases.

4. *Syphilitic otitis media* (rare today) is caused by *Treponema pallidum*. The usual aural manifestations of syphilis is a meningoneurolabyrinthitis, but occasionally the middle ear cleft is involved by a *gummatous osteitis* or osteoperiosteitis. The gummatous change may lead to a foul discharge and extensive destruction of the mastoid. Secondary pyogenic infection develops. Diagnosis may be suspected from foul painless otorrhea in the presence of sensorineural deafness and confirmed by specific tests for syphilis: (a) detection of *T. pallidum* by direct darkfield examination of material obtained from a primary and secondary lesion, (b) serologic tests (Wassermann, VDRL, Kahn and Kline, Kolmer, rapid plasma reagin tests). (c) *Treponema pallidum* immobilization test (TPI), and (d) the more sensitive *fluorescent treponemal antibody absorption test (FTA-ABS)*. *Inductomy* may help the diagnosis. Treatment should be both local (removal of sequestra may be necessary) and general (systemic penicillin and steroid therapy). There is some evidence that penicillin alone will not eradicate *T. pallidum* from the human temporal bone, and that ampicillin may reach higher levels than penicillin in perilymph. However, ampicillin and penicillin, when each is combined with a high dosage of steroids, seem equally effective in the treatment of syphilitic hearing loss.

5. *Late congenital syphilis* is characterized by the following: A rather abrupt onset of deafness in apparently healthy young adults; bilateral involvement, which initially may be asymmetric; family history of lues; periods of exacerbations, i.e. pregnancy, colds; rapid progression even to complete bilateral loss of cochlear and vestibular function in some cases; vestibular symptoms occasionally resembling those seen in Ménière's disease; *Tullio's sign* (vertigo and nystagmus on stimulation with loud noise); *Hennebert's sign*: despite an intact drum, a positive fistula test, especially with negative pressure; preponderance in females; negative or equivocal blood serology (Wassermann, VDRL, Kahn and Kline, Kolmer tests), with negative spinal fluid serology; positive FTA-ABS; and a long interval between eye and ear involvement.

One should remember that some diseases may produce false-positive reactions to serologic tests. These include malaria, infectious mononucleosis, systemic lupus erythematosus, and leprosy. Syphilis, both congenital and acquired, can cause sensorineural hearing loss. The incidence of such loss among various forms of syphilis has been estimated at 18% for late congenital, 17% for early congenital, 25% for late latent, 29% for asymptomatic neurosyphilis, and 80% for symptomatic syphilis.

The histopathology of syphilitic infection is primarily two-fold. First, syphilis may cause a meningoneurolabyrinthitis as the predominant lesion in early (infantile) congenital syphilis and in the acute meningitides of secondary and tertiary syphilis. Second, syphilis may cause an osteitis of the temporal bone as the predominant lesion with secondary involvement of the membranous labyrinth in late (tardive) congenital, late latent, and tertiary syphilis. Pathologically, the lesions of congenital and acquired syphilis cannot be differentiated and similarly the hearing loss may be sudden or progressive, with or without vestibular involvement in both congenital and acquired syphilis.

The basic histologic feature of bone involvement is an inflammatory rarefying osteitis featured by round cell infiltration, multinucleated giant cells, and endarteritis leading to varying degrees of destruction of the bony labyrinth. Mononuclear leukocytic infiltration and

obliterative endarteritis are common to all syphilitic lesions, whatever the organ affected. Mild reactions promote proliferation of fibrous tissue leading to an inflammatory fibrosis. Severe reactions result in gummatous lesions which are characterized by lymphocytic infiltration, vascular occlusion, and central necrosis.

Other sites commonly involved in congenital syphilis are (a) the nasal cartilaginous and bony framework (snuffles), (b) periosteitis of the cranial bones (bossing of the skull), (c) periosteitis of the tibia (saber shins), (d) injury to odontogenous tissue (Hutchinson's teeth), (e) involvement of epiphyseal cartilages (reduction in stature), and (f) interstitial keratitis (cloudy cornea).

Otitis Externa

1. The usual infecting organism found in localized external otitis (furunculosis) is *S. aureus*.

2. The most common organisms found in diffuse external otitis (swimmer's ear) are *Pseudomonas aeruginosa* (*B. pyocyaneus*) and *Staphylococcus*. Less commonly found are *Streptococcus* and *Proteus vulgaris*.

3. The most common organism found in perichondritis is *P. aeruginosa* (*B. pyocyaneus*).

4. The most common fungi affecting the external ear are *Candida albicans*, *Aspergillus niger*, and yeastlike fungi.

5. The most common organism found in impetigo contagiosa of the external ear is *S. aureus*.

6. *Bullous myringitis* is caused by a virus and generally associated with an acute upper respiratory infection (most commonly influenza). The *Mycoplasma pneumoniae* organism was implicated but its role in isolated tympanic membrane infection is not proved. In children the same organisms of acute otitis media may be found in bullous myringitis.

Temporary sensorineural hearing loss may be associated with this condition.

The serous or hemorrhagic blebs on the tympanic membrane and adjacent meatal wall may produce severe pain without fever and hearing loss. Treatment is supportive. The blebs may be opened in the presence of severe pain.

7. *Herpes zoster oticum* (Ramsay Hunt syndrome) is a viral infection affecting the geniculate ganglion characterized by facial paralysis; herpetic vesicles in the external auditory canal and cavum conchae; severe ear pain; and impairment of lacrimation, salivation, and taste; often with vertigo and a sensorineural hearing loss. Treatment is symptomatic. Facial nerve decompression including the region of the geniculate ganglion may be indicated when electrical excitability is lost or markedly impaired.

8. *Malignant external otitis* (necrotizing external otitis) is a serious infection of high mortality which occurs in the elderly diabetic. Other predisposing conditions include arteriosclerosis, immunosuppression by chemotherapy, steroid administration, or hypogammaglobulinemia. The responsible organism is uniformly *P. aeruginosa*.

It begins insidiously, frequently with a history of minor trauma, and is characterized by progressive pain and purulent discharge from the external canal. The infection begins in the external auditory canal and extends inferiorly into the soft tissues at the junction of the cartilaginous and osseous portions of the external auditory canal or through the fissures of Santorini. The infection thus involves the parotid gland, cartilage, bone, nerves, and blood vessels.

The pathognomonic sign is the *presence of active granulation tissue in the external auditory canal* at the junction of its osseous and cartilaginous portion. There is pain on movement of the temporomandibular joint and marked tenderness on palpation beneath the external auditory canal. Facial palsy is an ominous prognostic sign and is due to involvement of the facial nerve at its exit from the stylomastoid foramen.

The infection is resistant to ordinary methods of treatment and, if not arrested, progresses to chondritis; osteitis and osteomyelitis of the temporal bone and base of the skull; facial nerve paralysis and other multiple cranial nerve palsies; sigmoid sinus thrombosis; meningitis; brain abscess; and death.

Treatment should consist of local debridement and systemic administration of carbenicillin and gentamicin for 4-6 weeks. Wide surgical debridement, including radical mastoidectomy, total parotidectomy, excision of the condyle and ascending ramus of the mandible, and occasionally sacrifice of the facial nerve, may be necessary for its control. Subtotal temporal bone resection to gain access to the primary focus of infection and provide adequate drainage may become necessary.

9. The most widely used otic preparations contain antibiotics such as neomycin and/or polymixin and also a steroid to relieve inflammation. The inclusion of both antibiotics is recommended in treating acute external otitis and chronic suppurative otitis media. Chloramphenicol drops are available to treat *B. fragilis* infection. Both amphotericin B and nystatin are available in topical preparations to treat candidiasis, but they have no activity against *Aspergillus*.

Cholesterol Granuloma

The cholesterol granuloma does not represent an independent clinical or pathologic entity. Rather, it is a term used for the description of a tissue response of the temporal bone to the presence of a particular foreign body, i.e. cholesterol crystals.

Three factors are considered to play an important role in its development: interference with drainage, hemorrhage, and obstruction of ventilation. The cause of the initial hemorrhage may be a hemorrhagic inflammation or diathesis, a trauma, or some other form of vascular disorder. Interference with air exchange and clearance can be caused by: tubal blockage, persistent mesenchyme, polypoid changes, scar formations, tympanosclerosis, cholesteatoma,

etc.

The cholesterol granuloma may develop in any portion of the pneumatic system of the temporal bone, and it can be associated with a variety of middle ear disorders. Its principal precursor is the chronic middle ear effusion or serous otitis media. Its clinical expression and hallmark is the "idiopathic hematotympanum", the dark bluish discoloration of the tympanic membrane.

Osteitis and bone erosion are manifestations of an unusual more advanced stage. Resorption of bone, in a rare instance, may lead to extensive destruction of the temporal bone.

Definition of Tympanoplasty and Mastoidectomy

Definitions

Simple Mastoidectomy (Cortical Mastoidectomy)

A complete mastoidectomy with anatomic dissection of all accessible pneumatic cells is indicated for acute mastoiditis with impending or existing complications, or acute mastoiditis which does not resolve after appropriate antibiotic therapy and myringotomy drainage.

Myringoplasty

An operation in which the reconstructive procedure is limited to the repair of tympanic membrane perforation.

Tympanoplasty Without Mastoidectomy

An operation to eradicate disease in the middle ear and to reconstruct the hearing mechanism, without mastoid surgery.

Tympanoplasty With Mastoidectomy

An operation to eradicate disease in both the mastoid process and middle ear cavity, and to reconstruct the hearing mechanism.

Modified Radical Mastoidectomy

An operation to eradicate disease of the epitympanum and mastoid in which the mastoid and epitympanic spaces are converted into an easily accessible common cavity by removal of the posterior and superior external bony canal walls. In this operation the tympanic membrane and functioning ossicles are left intact. Thus infection is eradicated and hearing preserved.

Radical Mastoidectomy

An operation to eradicate disease of the middle ear and mastoid in which the mastoid, antrum, and the middle ear are exteriorized so that they form a common cavity with the external auditory canal. In this operation, the tympanic membrane, malleus, incus, chorda tympani, and the mucoperiosteal lining are all removed.

Classifications of Tympanoplasty by Wullstein (Table 7-2)

Table 7-2. Classification of Tympanoplasty by Wullstein

Type

--> Damage to Middle Ear
- Method of Repair

I

--> Perforated tympanic membrane with normal ossicular chain.
- Closure of perforation, type I same as myringoplasty.

II

--> Perforation of tympanic membrane with erosion of malleus.
- Closure with graft against incus or remains of malleus.

III

--> Destruction of tympanic membrane and ossicular chain but with intact and mobile stapes.
- Graft contacts normal stapes. Also gives sound protection for round window.

IV

--> Similar to type III, but head, neck, and crura of stapes missing; footplate mobile.
- Mobile footplate left exposed or graft attaches to mobile footplate; air pocket between round window and graft provides sound protection for round window.

V

--> Similar to type IV plus fixed footplate.
- Fenestra in horizontal semicircular canal; graft seals off middle ear to give sound protection for round window.

Mastoid Obliteration Operation

An operation to eradicate disease when present and to obliterate mastoid or fenestration cavities.

Paparella modified type V tympanoplasty, subdividing it into type Va (fenestration of the horizontal semicircular canal) and type Vb (stapedectomy in cases of tympanoplasty type IV with stapes fixation) (Fig. 7-1).

Classification of Tympanoplasty by Farrior

Farrior proposed the types of tympanoplasties according to the basic pathologic anatomy at the completion of surgery, rather than classifying them according to the method of reconstruction. He also advocated the grouping of cases according to the preoperative status of the middle ear mucosa, eustachian tube function, and associated diseases of the nose and nasopharynx.

Classification

1. Type I: The reconstruction of a new eardrum, intact malleus, incus and stapes.
2. Type II: The reconstruction of a new eardrum in its natural position.
3. Type III: The reconstruction of a new eardrum on top of an upright, freely mobile stapes.
4. Type IV: The reconstruction of a new eardrum and columella on the stapedial footplate.
5. Type V: The reconstruction of an eardrum either over a fistula in the horizontal semicircular canal or a new eardrum with a secondary fenestration of the horizontal semicircular canal.

Tympanoplasty III (Farrior)

1. Type III: drum on stapes.
2. Type III IG: incus graft.
3. Type III IGM: incus graft to malleus.
4. Type III MR: malleus repositioned.
5. Type III MG: malleus graft.
6. Type III BG: bone graft.
7. Type III SS MS: stainless steel malleus to stapes, etc.

The best results in tympanoplasty are obtained when the stapes is upright and freely movable, regardless of the type of reconstruction used. In classifying tympanoplasty according to basic pathologic anatomy all cases with intact stapedial superstructures are classified under type III with indication of the type of superstructure by initials, as IG incus graft.

Tympanoplasty IV (Farrior)

1. Type IV: no columella.
2. Type IV IG: incus graft.
3. Type IV MG: malleus graft.
4. Type IV BG: bone graft.
5. Type IV C SS: cartilage graft with stainless steel.
6. Type IV HG MIS: homograft drum with malleus, incus, and stapes.

In classifying tympanoplasty according to the basic pathologic anatomy all cases with absent stapedial superstructures are subclassified under type IV with indication of the type of superstructure reconstructed as MG (malleus graft).

Grouping (Bellucci)

1. Group I: A good prognosis being those cases who are relatively free of any associated middle ear or eustachian tube disease.
2. Group II: A fair prognosis, has a period of quiescence.
3. Group III: With a poor prognosis, has no period of quiescence.
4. Group IV: With a very poor prognosis, has persistent disease with associated deformity of the nasopharynx as cleft palate.

Systematic Approach to Evaluate and Treat Chronic Otitis Media

Systematic *preoperative evaluation* of patients with chronic otitis media and a brief description of *surgical procedures* commonly performed for chronic otitis media will be described.

Preoperative Evaluation

Preoperative evaluation should include:

1. Careful analysis of symptoms and signs.
2. Otologic examination.
3. Examination of the upper respiratory tract.
4. Audiologic evaluation.

5. Preoperative preparation.

Careful Analysis of Symptoms and Signs

Careful analysis of the symptoms and findings allows the otologist to determine the need for surgery, its urgency (if any), and the anticipated results.

Otorrhea

1. Discharge from the ear is the most common manifestation of chronic otitis media. Note should be made of its duration, frequency, character, and odor.

2. Malodorous discharge, at times bloody, of a constant or frequently recurring nature usually indicates significant middle ear and mastoid disease.

3. A central perforation without significant disease is usually accompanied by episodes of mucoid discharge of short duration. This discharge may be initiated by an upper respiratory infection or by introduction of water into the ear.

Hearing Loss

1. The extent of hearing impairment in chronic otitis media is dependent primarily on the degree of ossicular disruption.

2. In the absence of cholesteatoma, a conductive loss of 20 dB or less usually indicates that the ossicular chain is intact.

3. Disruption or fixation of the chain results in an impairment of 30 dB or more.

4. It is not unusual to find normal hearing in an ear with an attic perforation and cholesteatoma. This may be an indication of an intact ossicular chain. However, this may indicate that sound transmission is accomplished through a mass of cholesteatoma that has replaced ossicular tissue ("cholesteatoma hearer" or "silent cholesteatoma").

5. A progressive conductive impairment in the absence of active disease suggests ossicular fixation. This may be due to tympanosclerosis or otosclerosis. This is significant because surgery may have to be performed in two stages: one a graft of the tympanic membrane to eliminate disease, the other a revision to perform stapedectomy or type Vb tympanoplasty. The patient should be advised of this possibility preoperatively.

Pain (Otalgia)

1. Pain is not a frequent complaint in chronic otitis media unless there is secondary otitis externa.

2. Dull pain, in the absence of otitis externa, particularly when it is severe enough to disturb sleep, is usually an indication of an expanding mass of cholesteatoma or empyema in the antrum. Surgery should not be delayed.

3. Pain may indicate acute exacerbation of infection by upper respiratory infection.
4. Pain may indicate development of complications of chronic otitis media such as petrositis, subperiosteal abscess, or lateral sinus thrombosis.

Vertigo

1. Minor degrees of postural vertigo are seen frequently in patients with chronic otitis media.
2. Continuous vertigo or postural vertigo of recent onset in a patient with cholesteatoma is usually an indication for immediate surgery. It indicates labyrinthine irritation or a semicircular canal fistula.

Facial Nerve Paralysis

Facial nerve paralysis occasionally develops in the course of chronic otitis media with cholesteatoma. If and when it occurs, surgery should be undertaken without delay to relieve pressure on the nerve. It is not necessary to "decompress" the nerve in most cases. Elimination of the disease is sufficient.

Examination of the Upper Respiratory Tract

1. A sound review of the history and careful examination of the upper respiratory tract is mandatory.
2. Gross abnormalities and chronic suppurative sinus disease should be identified and corrected before reconstructive surgery is performed.
3. Patients with histories of "repeated colds" in winter months are not good candidates for tympanoplasty.

Otologic Examination

1. Careful inspection of the ear should be the first part of the systematic evaluation. The inspection should include examination under magnification, with an otoscope or preferably with a surgical microscope.
2. *Pneumatic otoscopy* should be routine to examine the mobility of the tympanic membrane and malleus and to rule out coexisting chronic serous otitis media. Care must be taken to avoid forceful insufflation. Lethal intracranial complications following air insufflation have been reported.
3. Careful examination of the attic area should be done to identify hidden retraction pockets, perforation, and/or cholesteatoma. It is often necessary to freely change the position of the patient's head and the angulation of the otoscope or microscope.
4. The fistula test should be performed whenever a marginal perforation is present or

when there is a history of dizziness.

5. Specific notes should be made regarding the type of perforation, the character of the discharge, the status of the mucosa, and the presence or absence of ossicular tissue.

Discharge (Otorrhea)

1. The character of the discharge, whether mucoid or purulent, with or without odor, is noted.

2. A mucoid discharge without odor is usually an indication of mucosal disease and/or eustachian tube malfunction, often of a temporary nature.

3. Purulent discharge is an indication of infection. This may be a transient mucous membrane infection by opportunistic organisms, in which case it should clear rapidly with local treatment. Purulent discharge that does not subside on local treatment is an indication of a resistant organism, irreversible mucous membrane disease, cholesteatoma, or all of these.

4. The presence of odor suggests tissue necrosis. A malodorous discharge usually is found in active cholesteatoma.

Perforation

1. Perforations of the tympanic membrane are generally divided into two types: central and marginal.

2. A *central* perforation usually is not associated with cholesteatoma although there are exceptions, especially in children. Intermittent discharge of mucoid material, responding quickly to local treatment, is the rule.

3. A *marginal* perforation usually is associated with a cholesteatoma. Continuous or frequently recurring malodorous discharge is the rule. This may respond only temporarily, if at all, to local treatment.

4. There are two types of marginal perforations: attic and posterosuperior marginal. *Attic* perforations involve the area of the pars flaccida. The pars tensa may at times appear quite normal. As a result, perforations in this area occasionally are overlooked. A small perforation may be covered by dried crusts. A polyp of granulation tissue may be seen to extrude through the perforation and tends to block discharge. The hearing impairment is negligible at times because the cholesteatoma develops external to the ossicles and even may extend into the antrum without producing significant ossicular necrosis.

5. A posterosuperior marginal perforation below the malleolar ligament may or may not be associated with cholesteatoma. When cholesteatoma is present, the hearing impairment tends to be more severe than with attic perforation. The incus, and at times the stapes, is destroyed early in the development of the disease.

Status of the Middle Ear Mucosa

1. Much may be learned regarding the possible outcome of surgery by careful evaluation of the middle ear as seen through the perforation.

2. The character of the mucosa, the status of the ossicles, and the presence or absence of tympanosclerosis are noted.

3. The presence of normal or near normal mucous membrane is a favorable prognostic sign. When squamous epithelium is observed in the middle ear, the status of the tubotympanic recess should be checked.

4. When ossicular necrosis occurs, usually the incus is the first involved. Of prime importance is the status of the stapes. When the stapes superstructure is absent the prognosis for restoration of hearing is usually less favorable. If the malleus handle is also absent, a two-stage operation may be required for hearing improvement.

5. *Tympanosclerosis* is the term used to describe a sclerotic or hyaline change of the submucosal tissue of the middle ear. It appears to be an end product of recurrent acute or chronic ear infection. Hyalinized connective tissue develops under the mucous membrane superficial to the bone. Calcification and ossification may occur. Its presence may affect the ultimate prognosis for hearing improvement. If there is a progressive hearing impairment, there may be ossicular fixation by tympanosclerosis. A second operation for stapedectomy may be required at times: the patient should be informed of this possibility.

Myringosclerosis (Doyle) is the term applied to describe deposits of hyaline masses with fibrous layer of the tympanic membrane, but it also is referred generally to as tympanosclerosis of the tympanic membrane. It is often necessary to remove these hyaline masses for a successful myringoplasty.

Fistula Test

This is a production of vertigo and deviation of the eyes on the application of pressure to the affected ear. This is elicited by increasing the pressure within the ear canal by means of a pneumatic otoscope or Politzer's bag with an olive tip, or pressing sharply on the tragus with the thumb. Suction with the Politzer bag may cause the reversal of the labyrinthine symptoms. The significance of this test is that there is a fistula of the labyrinth due to destructive cholesteatoma or infection. A positive fistula test is present in two-thirds of the cases with a labyrinthine fistula.

A positive fistula test despite an intact tympanic membrane may indicate an abnormally mobile footplate of the stapes, and suggests congenital syphilis (Hennebert's sign).

Patch Test

When some hearing loss accompanies a central perforation, it is possible to assess the damage of the ossicular chain by placing a small patch of cigarette paper or a Teflon sheet over the perforation. Should the hearing improve with this maneuver, it is assumed that the

ossicular chain is intact, and that myringoplasty is likely to succeed in improving the hearing.

Eustachian Tube Function

1. The function of the eustachian tube is to protect, aerate, and drain the middle ear and mastoid. The muscle responsible for the opening of the eustachian tube is a part of the *tensor veli palatini* known as the *dilator tubae*.

2. Methods of estimating the pressure required to open the eustachian tube have been devised. Unfortunately, the methods of testing the eustachian tube are not entirely satisfactory and it may be difficult to derive a quantitative measurement in a clinical situation.

3. Miller developed a method of eustachian tube function test by applying the pressure differential through a catheter sealed in the external auditory canal. The effect of swallowing on this pressure may be recorded easily on a paper writer such as an ECG machine. The necessity of a perforated tympanic membrane has made the determination of normal values difficult, but this is not a problem in clinical practice since most surgical patients have a preexisting perforation. The application of this method in patients with secretory otitis media requires placing a tube through the intact membrane. In practice the test is carried out by causing a negative pressure of 250 mmH₂O within the external auditory canal and observing the equalization of pressure as the patient swallows. With normal function, the pressure difference is eliminated after several swallows. Four gradations of abnormality have been described, the most severe being in those patients in whom no airflow occurs even with the application of a positive pressure of 250 mmH₂O, indicating complete functional obstruction of the tube.

4. The tympanic cavity clearance test with use of a dye through the intact tympanic membrane or through the tympanic perforation also has been used as a measure of function.

5. Inflation (Valsalva), although not always accurate, remains the simplest and a most satisfactory method. For practical purposes, if the eustachian tube can be inflated easily by the Valsalva's or Politzer's method the prognosis for successful tympanoplasty is excellent. If inflation is difficult, requiring repeated attempts or use of the catheter, there may be development of secretory otitis after tympanoplasty, requiring insertion of a polyethylene tube through the tympanic membrane.

6. Tympanometry has become a useful method of eustachian tube function test (see *Tympanometry* below). A blocked eustachian tube is usually associated with type B or C tympanogram.

Audiologic Evaluation

Careful audiometric tests should be performed routinely. The findings are confirmed by the otologist using the tuning fork with a Barany noisemaker to mask the opposite ear.

The *minimum* audiometric test requirements are pure tone, bone-air conduction thresholds, speech reception levels, and speech discrimination scores. The audiometric test results should *always* coincide with those of the tuning fork test.

Tympanometry

The most significant advance in the identification of middle ear disease is the use of a new instrument. The electroacoustic impedance bridge with which a tympanogram can be obtained.

Tympanometry is a reliable, simple procedure, easily carried out in a short time. To perform tympanometry, a small probe is inserted in the external auditory canal. A tone of fixed characteristics is presented via the probe, and the compliance of the tympanic membrane is measured electronically while the external canal pressure is artificially varied.

As is true for eardrum mobility observed visually with a pneumatic otoscope, acoustic compliance is greatest when pressures are equal on both sides of the tympanic membrane. Thus a peak is present when the middle ear pressure is normal (type A). A peak is present in the negative range when middle ear pressure is reduced (type C). Middle ear effusions are present in most cases in which no impedance peak can be determined (type B) (see Fig. 7-2).

For evaluation of chronic otitis media, tympanometry is useful to detect or confirm the following:

1. Otitis media: type B (43%) or C (47%).
2. Cholesteatoma: type B (54%) or C (42%).
3. Middle ear fluid: type B (44%) or C (45%).
4. Scarred or thickened TM: type A (45%) or C (40%).
5. Ossicular fixation: "shallow" type A (reduced peak).
6. Ossicular discontinuity: "deep" type A (100%) (open high peak) (high compliance).

Tympanometry also is useful (1) as an aid in diagnosis when otoscopy is equivocal or difficult, particularly in children, (2) in conforming otoscopic diagnosis, and (3) as a screening test of ear diseases.

Radiographic Examination

Mastoid x-rays which are useful for evaluation of chronic otitis media include Law, Schüller, Stenvers, and submentovertical. Polytomography and/or CT scan may clearly demonstrate bony destruction by cholesteatoma, presence or absence of ossicles, and, in some cases, fistula of the horizontal semicircular canal (see Chap 24).

The decision whether or not to operate is rarely based on the x-ray findings alone. They are helpful, however, in detecting disease not otherwise suspected but which requires mastoid investigation. Occasionally one detects an anatomic variation, i.e. a far forward lateral sinus, which allows a better planned approach to the mastoid disease.

Preoperative Treatment

Systemic antibiotics are of minimal value in chronic otitis media. Before any drugs are applied to the ear, the ear should be thoroughly cleared of debris and discharge. This can be done in several different ways. Whenever possible, suction should be employed. The ear can be gently cleaned with cotton applicators. Insufflation of boric acid powder may assist in drying the ear. If fungal infection is suspected, an antifungal agent should be used, usually in powder form.

In patients with chronic otitis media with mucoid discharge treatment of coexisting upper respiratory infections must always be a part of the management of otitis media. Exacerbations of the chronic otitis media commonly accompany and are dependent upon infections in the nose and nasopharynx. Therefore, any conditions such as sinusitis, nasal obstruction, or any other recurrent nasal infection, must be treated before reconstructive surgery is attempted. This also includes allergic evaluation when indicated.

Surgical Treatment

Any patient with a perforated tympanic membrane, chronic ear infection, or a defect in the ossicular chain is a potential candidate for surgical treatment. The primary purpose of surgery may be one or all of the following:

1. Elimination of infection.
2. Improvement of hearing.
3. Closure of a perforation (prevention of infection).

It should be understood first that the complete eradication of disease is a prerequisite of all surgical treatment, and preservation and restoration of hearing are secondary.

Indications for immediate surgery include:

1. Persistent ear pain.
2. Vertigo (continuous).
3. Facial paralysis.
4. Threatened intracranial complications.

The following are the common operations performed in chronic ear surgery. With rare exceptions, an operation for chronic ear disease can be classified as one of the following. Technical surgical variations peculiar to one or another surgeon do not alter the fundamental classifications (Standard Classification for Surgery of Chronic Ear Infection).

1. Modified radical mastoidectomy.

2. Radical mastoidectomy.
3. Mastoid obliteration operation.
4. Myringoplasty.
5. Tympanoplasty without mastoidectomy.
6. Tympanoplasty with mastoidectomy.

Modified Radical Mastoidectomy

This commonly performed operation (Bondy's operation) remains the basic operation in most mastoidectomies and tympanoplasties, particularly in the sclerotic mastoid, when the disease extends medial to the facial nerve and into the posterior tympanic recesses. This frequently is used for acquired cholesteatoma and exteriorizes the mastoid antrum to form a common cavity with the external auditory canal. Unlike the radical mastoidectomy the tympanic membrane and functioning ossicles are left intact.

Radical Mastoidectomy

This operation is performed for cases of chronic otitis media with cholesteatoma which have developed secondary to the marginal tympanic perforation; for those beyond the scope of the modified radical mastoidectomy; for chronic otitis media with extension into the labyrinth or petrous portion of the temporal bone; for chronic otitis media with osteitis or osteomyelitis; and for certain neoplasms.

In this operation, the mastoid antrum, mastoid air cells, and middle ear space are exteriorized so that they form a common cavity with the external auditory canal. Meatoplasty is always performed. This permits inspection and periodic cleaning in the postoperative period.,

Radical mastoidectomy is primarily for eradication of infection and not intended to restore the hearing.

Mastoid Obliteration Operation

This is an operation to obliterate the mastoidectomy cavity, which is created following the radical or modified radical mastoidectomy, using a muscle or other tissue pedicle graft. The purpose of this operation is to restore near normal anatomic contour and avoid the frequent aftercare of the mastoid cavity.

When the disease, either cholesteatomatous or necrotic, has extensive ramifications, and there is the slightest doubt in the surgeon's mind regarding complete removal, obliteration of the cavity is not advisable. Recurrent cholesteatoma behind the muscle may develop.

Myringoplasty (Type I Tympanoplasty)

Myringoplasty is an operation in which the reconstructive procedure is limited to the repair of tympanic perforation, utilizing a tissue graft. The ossicular chain is intact and mobile.

Since Zollner and Wullstein opened the way to tympanoplasty, there have been many grafting materials used. These include pedicled canal skin (Sooy), canal skin (House and Sheehy), vein (Tabb, Shea), fascia (Storrs), fat (Ringenberg), perichondrium (Goodhill), heart valves, corneas, Gelfoam and more recently, homograft tympanic membranes have been used (Chalat, Marquet, Perkins). *Temporalis fascia* has become the most widely used of all the grafting materials.

If there has been any discharge from the ear or any moisture in the ear during the previous 6 months, myringoplasty alone is contraindicated. If there has been recent discharge, the repair should be combined with inspection of the attic, aditus and antrum.

An edema or polyposis of the mucous membrane of the middle ear will render the operation unlikely to succeed. Poor eustachian tube function is also a contraindication.

Tympanoplasty Without Mastoidectomy

This refers to an operation to eradicate disease in the middle ear and to reconstruct the hearing mechanism without mastoidectomy, with or without tympanic grafting.

The cardinal principles of tympanoplasty have been and still are: first, control of infection through eradication of disease, and second, reconstruction of the middle ear sound-conducting mechanism.

Type I Tympanoplasty

This was previously discussed. Tympanoplasty type I or myringoplasty is an operation in which the procedure is limited to repair of the tympanic membrane perforation.

It is a good habit to routinely evaluate the middle ear and ossicles to rule out ossicular fixation or discontinuity. This can be done by elevating the tympanomeatal flat. It is important to prepare the graft recipient site first since it is difficult to denude the drumhead after it becomes flaccid. A wide recipient site should be established. In the presence of a total tympanic perforation, this necessitates reflecting the graft up onto the adjacent denuded bony canal wall for at least several millimeters. It is especially important to position the graft "tightly" in the anterior sulcus where graft failure occurs most commonly as a result of technical error. It is here that the branches of the anterior tympanic and deep auricular arteries provide critical blood supply to the graft.

The graft may be placed on the denuded outer surface (overlay graft) or on the denuded inner surface (underlay graft) of the eardrum. In the latter case, the graft is supported by Gelfoam in the middle ear.

One of the persistent problems encountered with lateral placement of the graft is lateral migration of the graft away from the handle of the malleus or the anterior sulcus causing a thick blunting or rounding off in this area. Medial placement of the graft avoids this problem. However, when the perforation is total or involves the anterior half of the tympanic membrane, one should be aware that the anterior portion of the medially displaced graft may have a tendency to be pulled away from the drum remnant toward the medial wall of the middle ear and may result in postoperative perforation in this area.

Type II Tympanoplasty

Type II tympanoplasty consists of a graft placement directly upon the incus. This can result from a destroyed malleus but more commonly it is seen in instances in which wide atticotomy or removal of the posterior bony canal wall with mastoidectomy is done, in which case the graft necessarily rests upon the body of the incus.

Type III and Type IV Tympanoplasty

Type III and type IV tympanoplasty are more often done in association with complete mastoidectomy. It is important to expose the facial recess (suprapyramidal recess) and sinus tympani (infrapyramidal recess for the removal of the disease).

Type V Tympanoplasty

While performing tympanoplasty fixation of the stapes due to either otosclerosis or more commonly to tympanosclerosis may be found.

When tympanosclerosis fixation is found it is best to effect a mobilization by removal of the tympanosclerotic tissue, if possible. If this is not possible, all infected tissues should be removed, a graft applied, and the observed for at least 6 months to a year.

Assuming a dry ear with no tendency toward infection, good tubal function, and good auditory function in the opposite ear, type V tympanoplasty can be considered.

The original type V tympanoplasty consisted of fenestration of the horizontal semicircular canal (type Va). If anatomic characteristics are suitable, stapedectomy can be performed (type Vb). Surprisingly good hearing may be obtained.

Stapes mobilization or stapedectomy at the time of tympanic membrane grafting, even in the dry ear, may result in a sensorineural hearing loss. At least 12 months should elapse between the initial tympanoplasty and secondary stapedectomy.

Tympanoplasty with Mastoidectomy

The elimination of infection by mastoidectomy is combined with reconstruction of the hearing mechanism.

Intact Posterior Wall Mastoidectomy with Tympanoplasty

The intact canal wall technique has been popularized in recent years by many otologists. This procedure prevents a mastoid cavity postoperatively. It should be stressed, however, that the primary objective should always be the removal of the disease and not the preservation of the posterior canal wall.

Following a complete mastoidectomy, the antrum and aditus are enlarged so that the incus is readily seen. The epitympanum is inspected through the aditus. The facial recess (suprapyramidal recess) and sinus tympani (infrapyramidal recess) are exposed and are cleared of disease and tympanoplasty is accomplished. If a large area of the posterior superior canal wall is removed, retraction of the tympanic membrane may develop. A silicone rubber sheet is placed to prevent adhesions. This procedure is contraindicated in the following situations.

1. The only hearing ear.
2. Labyrinthine fistula when the other ear has cholesteatoma.
3. Inadequate exposure due to a severely contracted mastoid.
4. Extensive canal wall destruction by disease.

In these situations, it is "safer" to remove the posterior bony canal, and perform modified radical mastoidectomy combined with tympanoplasty.

Ossicular Reconstruction in Tympanoplasty

1. The goal of functional reconstruction is to obtain a permanent restoration of hearing with neither conductive nor sensorineural hearing loss.

2. *When the stapes is normal*, a carefully fitted ossicular prosthesis (autograft or homograft) is the procedure of choice. The incus is the most readily available ossicle. The incus (shaped or sculptured) may be placed between the handle of the malleus and the head of the stapes, or it may be placed adjacent to the manubrium. When the stapes capitulum is more deeply seated, the malleus head may be used instead of the incus.

More recently, partial ossicular replacement prosthesis (PORP) (Shea) has been successfully used when the incus is missing while the stapes is intact.

3. The most common ossicular problem encountered is necrosis of the long crus of the incus.

4. The most common cause of failure is separation of the ossicle from the head of the stapes, followed by fixation of the ossicle to adjacent bony structures. Extrusion of the repositioned ossicle is uncommon.

5. *When the suprastructure of the stapes is missing*, a shaped incus may be placed between the handle of the malleus and the mobile footplate, or it may be placed adjacent to

the manubrium of the malleus. A strut of cartilage may be used instead. Cartilage has advantages. It does not become fixed by bone and it rarely extrudes.

6. When the malleus handle is absent, there is nothing to stabilize the graft. A two-stage procedure may be necessary: at the first stage, disease is removed, a Silastic sheet inserted to prevent adhesion, and a graft applied. The second stage involves removal of the plastic and insertion of a suitable prosthesis (cartilage). One may use a homograft tympanic membrane en bloc ossicles as a single procedure.

7. The plastipore total ossicular reconstruction prosthesis (TORP) as introduced by Shea is simple to insert, flexible enough for many situations, and suitable for bridging the gap when the incus and the suprastructure of the stapes are missing. To prevent the extrusion of a TORP, or PORP, placement of a piece of cartilage between the prosthesis and the undersurface of the malleus handle or the eardrum has been suggested.

8. The most common cause of ossicular fixation in case of chronic otitis media is tympanosclerosis. It may fix the malleus head and the stapes. When the malleus head is fixed, it should be amputated and the incus repositioned. When the stapes is fixed, disease is removed and the tympanic membrane repaired. At the second operation, a stapedectomy is performed.

9. Plastic and wire prostheses tend to be extruded. Bone chips tend to resorb in time.

10. Tympanoplasty can be successfully accomplished *for atelectatic ears* in selected cases. Paparella and Jung proposed a method of tympanoplasty which consists of reestablishing the mesotympanic space, strengthening the tympanic membrane by an underplant fascial graft, cutting the tensor tympani, extirpation of any disease, reestablishment of ossicular mobility and continuity, and the insertion of a ventilation tube in the anterior drumhead remnant along with silicone rubber sheeting and a moist Gelfilm implant.

Otologic Homografts

1. Homograft ossicles, mostly obtained from patients when the ossicle must be removed for various reasons, are autoclaved and then stored in 70% alcohol. These ossicles may be used interchangeably with autografts in any situation in which ossicular repositioning is indicated. They are well tolerated and it is difficult to tell microscopically a repositioned incus from a homograft incus.

2. The most frequently used homograft ossicle is homograft incus, followed by malleus and stapes. Smith employs a stapes homograft when there is a loss of the stapedial arch and there is a mobile footplate.

3. Homograft septal and tragal cartilage maintains its shape and structure with the exception of the chondrocytes which are missing from the lacunae. It is well tolerated and becomes covered with mucosa.

4. Homograft tympanic membranes are now available through the ear bank of Project Hear, Palo Alto, Ca. It was first used by Chalat, then Marquet, later by Brandow, and Perkins.

5. Tympanic membrane ossicles en bloc homografts may be indicated in the following situations (Perkins):

a. Large tympanic membrane perforations with malleus manubrial disease (malleus fixed, retracted, defective, or absent).

b. Large tympanic membrane perforations with malleus head and incus disease (attic cholesteatoma).

c. Tympanomastoid reconstruction (radical mastoidectomy cavity).

6. Many techniques to reconstruct the radical mastoidectomy cavity have been proposed. They include:

a. Obliteration procedures with soft tissues (musculoplasty) - soft tissues atrophy but cavity filled with dense fibrous tissues.

b. Obliteration with bone (Shea, M.C.).

c. Cartilage reconstruction of the ear canal either with tragal or homograft knee cartilage.

d. Intact posterior canal bone homograft with a homograft tympanic membrane.

e. Reconstruction with a homograft dura form and autogenous bone plate (Perkins 1976) using a homograft tympanic membrane with ossicles en bloc for middle ear reconstruction.

7. Complications of otologic homografts are absorption, infection, fibrous hyperplasia, extrusion, and rejection. They should not be used in an infected cavity.

Complications of Suppurative Otitis Media

Factors That Influence the Development of Complications

1. Factors influencing the spread of infection beyond the middle ear space include: The type and virulence of the infecting organism, its susceptibility to available antibacterial medication, the adequacy of treatment, the resistance of the host, and the presence or absence of associated chronic systemic illness.

2. The type III pneumococcus has a particular predilection for intracranial extension. The resistance of the host to this organism especially is lowered in infancy, old age, and diabetes. Intracranial extension of an acute infection of the middle ear occurs more often in poorly pneumatized than in well pneumatized temporal bones and in ears with a previous history of recurrent otitis media.

3. The bone-invading types of chronic otitis media that lead to complications are the relatively uncommon chronic osteomyelitis of the temporal bone, and the much more common

cholesteatoma of either the attic retraction or secondary acquired variety.

4. Complications often result from an insufficient dosage or an insufficient period of administration of the drug or selection of a less effective drug without the benefit of culture and sensitivity tests.

5. Cholesteatoma is potentially dangerous because of its capacity to destroy bone. This allows spread of the infection beyond the middle ear and the pneumatized areas of the temporal bone, and may result in otologic or intracranial complications. As it expands and spreads beyond its origin cholesteatoma can impede sound conduction and prevent the natural self-cleaning mechanisms of the ear from functioning.

6. Bone destruction by cholesteatoma results from pyogenic osteitis and enzymatic bone resorption.

7. The matrix of a cholesteatoma consists of the keratinizing squamous epithelium and the subepithelial stroma.

8. The proteolytic enzymes of the cholesteatoma are located primarily at the interface of the epithelium and subepithelium. The squamous epithelium has enzymatic activity as does the superficial layer of the subepithelium. Both together, however, have three times the enzymatic activity of either. The keratinous debris has no enzymatic activity.

9. A cholesteatoma is more destructive of bone when the non-inflamed subepithelium is thin. The bone appears to be protected, somewhat, by thicker subepithelium as long as that epithelium is noninflamed. If the subepithelium is infected and composed of granulation tissue, as in an "active" cholesteatoma, the bone destructive enzymatic activity is enhanced. Similar proteolytic enzymatic activity has been found, to a lesser degree, in normal skin and in chronically infected ears without cholesteatoma.

Pathways of Spread of Infection

Extension of Osteothrombophlebitis

Infection may pass along the vascular channels through intact bone by the process of osteothrombophlebitis. Complications from this pathway usually occur within 10 days of the original infection.

Extension by Bone Erosion

This is the most frequent manner of spread leading to a complication in case of acute otitis in a well pneumatized temporal bone, and it is nearly always the manner of spread in cases of chronic suppurative otitis media.

Complications resulting from this spread of infection usually do not occur for several weeks.

In acute otitis media the bone erosion is the result of a coalescent mastoiditis. In

chronic otitis media the bone erosion usually is due to a cholesteatoma, less often to chronic osteomyelitis. Through bone erosion, infection can spread to the mastoid cortex, the petrous portion of the temporal bone, the facial nerve, the labyrinth, the lateral sinus, or the dura. The treatment of a complication by bone erosion is directed toward the complication and *always* includes surgical removal of the focus of suppuration in the temporal bone.

Extension by Preformed Pathways

The preformed pathway may be a normal opening in the bony wall such the oval or round window leading from the middle ear to the labyrinth or the internal auditory meatus, perilymphatic duct, or endolymphatic duct and sac leading from the labyrinth to the meninges. The pathway may be developmental dehiscence of the floor of the hypotympanum or it may be the result of a skull fracture or previous ear surgery such as a fenestration, a stapedectomy, a labyrinthotomy for Ménière's disease, or a mastoidectomy in which dura was exposed. A perilymph fistula following partial or total stapedectomy establishes an open pathway for infection to extend into the labyrinth.

Acute infection often spread through a preformed pathway, causing early complications. Extension in chronic ear infection usually is secondary to bone erosion, causing late complications.

Types of Complications

There are two major categories of complications: extracranial and intracranial. Complications can usefully be classified as follows:

Extracranial

1. Subperiosteal abscess.
2. Facial paralysis.
3. Labyrinthitis.
4. Petrositis.

Intracranial

1. Extradural abscess.
2. Subdural abscess.
3. Brain abscess.
4. Lateral sinus thrombosis.
5. Meningitis.

6. Otitic hydrocephalus.

Subperiosteal Abscess

1. *Postauricular abscess* (the most common type) is formed by pus spreading through the minute vascular channels in the suprameatal (Macewen's) triangle and presents as a swelling between the tip of the mastoid and Macewen's triangle. The auricle is displaced forward, outward, and downward.

2. *Zygomatic abscess* is formed by pus escaping from the zygomatic cells near the squama. This presents as a swelling above and in front of the ear and may be confused with a parotid swelling. Rarely, the pus may spread downward and forward into the mandibular fossa with a displacement of the mandible toward the normal side so that the teeth no longer meet in occlusion.

3. *Bezold's abscess* results from the perforation of the tip of the inner aspect of the mastoid by the pus which will track down the sternocleidomastoid muscle and present as a swelling in the posterior triangle of the neck.

4. *Sagging of the posterior meatal wall* by a subperiosteal abscess rarely may occur as a result of a coalescent mastoiditis. Today, this is much less likely to occur in an acute otitis media than in a chronic otitis media with cholesteatoma formation.

5. *Parapharyngeal or retropharyngeal abscess* may result from an acute suppurative otitis media or mastoiditis. The pus may track from peritubal cells or by formation of an abscess below the petrous pyramid.

6. *Treatment of postauricular, zygomatic, and Bezold's abscess* is simple mastoidectomy, although a Bezold's abscess may require separate additional incision and drainage.

Facial Paralysis

Direct extension of infection into the facial canal through a dehiscence in the bony covering of the tympanic portion of the nerve or by destruction of the bone overlying the nerve causes facial nerve paralysis.

Immediate surgical decompression is recommended for facial nerve paralysis caused by chronic ear disease. Surgical decompression of the nerve is usually unnecessary for patients with acute otitis media and is offered to those patients who fail to respond to the usual treatment with myringotomy and antibiotics and/or whose nerves undergo degeneration as determined by electrodiagnostic tests.

Labyrinthitis

Labyrinthitis is the most frequent complication of otitis media due to extension of infection within the temporal bone. Three types of labyrinthine inflammation may occur as a complication of acute otitis media and mastoiditis. They are, in the order of ascending

severity, perilyabyrinthitis, perilyabyrinthitis, serous labyrinthitis, and suppurative labyrinthitis.

Perilyabyrinthitis (Labyrinthitis Fistula). Perilyabyrinthitis may be *surgically produced* (simple or radical mastoidectomy, fenestration, labyrinthotomy, or stapedectomy) or it may occur *spontaneously* due to bone erosion by cholesteatoma. A spontaneous fistula usually results from erosion of one of the semicircular canals, especially the lateral semicircular canal.

The most common severe complication from cholesteatoma is fistulization of the horizontal semicircular canal.

The diagnosis of labyrinthine fistula is established by eliciting the *fistula sign*. This consists of nystagmus and vertigo when positive and negative pressure is applied to the soft tissue covering the fistula. The nystagmus is produced by a movement of endolymph toward the ampulla with inward pressure displacement, with the quick component toward the affected ear. With negative pressure there is outward displacement and a movement of endolymph away from the ampulla, with the quick component of nystagmus toward the normal ear. The absence of the fistula test does not rule out the presence of a fistula. A positive fistula test is present in two-thirds of the cases with a labyrinthine fistula. The presence of a fistula in the horizontal semicircular canal may be demonstrated on the AP polytome x-ray.

The patient with an active fistula of the labyrinth often complains of dizziness if he presses against the tragus, manipulates the auricle, or quickly turns the head. Rarely, he may experience momentary vertigo when exposed to a loud noise (Tullio's phenomenon).

A strong positive fistula sign is always an indication for surgical examination of the labyrinth. When erosion is associated with chronic otitis media, a radical or modified radical mastoidectomy must be performed to eradicate the pathologic condition. In this way, spread of infection into the labyrinth can be prevented.

The cholesteatoma matrix can be removed with reasonable safety from most small (less than 2 mm) semicircular canal fistulae. When the matrix is firmly adherent to a large area of the membranous semicircular canal, its removal is not recommended because of a high incidence of postoperative sensorineural disease. When the fistula involves the cochlear wall, the cholesteatoma matrix should not be removed. Its removal carries a high risk of postoperative sensorineural deafness. If the contralateral ear has no auditory function and the ear with the fistula is the only hearing ear, the cholesteatoma matrix over the fistula is best left undisturbed. In such cases, the cavity should be kept open and the patient should be followed closely to determine if further active suppuration develops.

Serous Labyrinthitis. Serous labyrinthitis is a diffuse intralabyrinthine inflammation without pus formation, and is not followed by permanent loss of auditory and vestibular function. It is, however, a prepurulent condition and a potential precursor of suppurative labyrinthitis.

The treatment of serous labyrinthitis secondary to acute otitis media is primarily medical with a large dosage of antibiotics. Surgery is mandatory in chronic infections when serous labyrinthitis develops.

Suppurative Labyrinthitis (Purulent Labyrinthitis). Suppurative labyrinthitis is a diffuse intralabyrinthine infection with pus formation and is associated with permanent loss of auditory and vestibular function.

This may occur as a result of direct extension of the purulent process in the middle ear or mastoid into the labyrinth, or it may result from the spread of meningeal inflammation into the labyrinth through the internal auditory canal or less frequently through the cochlear aqueduct.

Clinical symptoms include nausea and vomiting, intense vertigo, tinnitus, hearing loss, and nystagmus.

Treatment should consist of intense antibiotic treatment and surgical drainage of the labyrinth.

Petrositis

Petrositis is an inflammation of the petrous portion of the temporal bone, characterized by that clinical triad of *otitis media*, *paralysis of the sixth cranial nerve*, and *pain of the fifth cranial nerve (Gradenigo's syndrome)*. The symptoms of petrositis depend upon the area of the petrous pyramid affected.

Past the age of 3 years approximately 30% of the temporal bones have pneumatization in the petrous apex. Even the pneumatized petrous apex has unpneumatized areas containing marrow. This makes this site more susceptible to osteomyelitis. Air cells extend into the petrous pyramid in two main groups: a *posterior group* from the epitympanum and antrum, around the semicircular canals into the base of the pyramid, not infrequently extending to the apex; and an *anterior group* from the tympanum, hypotympanum and eustachian tube, around the cochlea into the apex of the pyramid. The posterior group of cells is present in about 30% of the temporal bone, whereas the anterior group of cells is present in about 15% of the temporal bones.

Additional symptoms of petrositis, although not frequent, include transient facial weakness, mild recurrent vertigo, and fever. If the suppuration extends beyond the petrosa, there may be added symptoms of localized and/or generalized meningitis; a cerebellar or a temporal lobe abscess; thrombophlebitis of the inferior petrosal sinus and jugular bulb; or a lateral pharyngeal, retropharyngeal, or deep neck abscess (Bezold's abscess).

Petrositis should be suspected in any patient whose ear continues to drain after surgery for chronic infection, or in any patient with ear disease who complains of persistent pain that is otherwise unexplained.

Treatment is by surgical drainage. A systematic search of the common pathways for extension of infection into the petrous area must be undertaken. The easiest and safest surgical approach to infected petrous cells is along the route by which the cells invaded the petrosa. Surgical drainage should always be preceded by a complete simple mastoidectomy for the posterior group of cells, and a radical mastoidectomy for the anterior group of cells. Care must be taken to prevent injury to the carotid artery in exploring the anterior cell group.

Extradural Abscess

An extradural abscess is a collection of pus between the dura and the bone. Apart from coalescent mastoiditis, this is the most common complication of otitis media. It is usually secondary to bone erosion rather than a result of osteothrombophlebitis or via preformed pathways. If the pus lies against the dura of the posterior fossa medial to the sigmoid sinus, it is called an *extradural* or *epidural* abscess. If it lies against the split of the posterior fossa dura enclosing the lateral sinus, it is called *perisinus* abscess.

The most common symptom is persistent headache, however, many extradural abscesses are unnoticed before surgery. Other symptoms include an unusually severe earache and a malaise with low-grade fever. Marked pulsation of the purulent discharge accentuated by compression of the jugular vein is noted. One clinical feature that is somewhat pathognomonic of an extradural abscess is relief of headache by profuse drainage from the ear. The treatment is surgical drainage.

Subdural Abscess

A subdural abscess develops when pus accumulates between the dura and the arachnoid. This is uncommon. This may develop as a result of extension of an infection of the middle ear and mastoid through the intact bone and the dura by means of a thrombophlebitis of the veins or by direct extension with erosion of bone and dura.

The symptoms include headache, malaise, delirious state, focal seizures, and other neurologic signs such as hemiplegia, aphasia, or hemianopsia. Signs of meningeal irritation almost always are present.

Attacks of jacksonian epilepsy with hemiplegia developing in association with middle ear infection should be regarded as indicative of subdural abscess until proved otherwise. Treatment is surgical drainage of the subdural space.

Brain Abscess

Otogenic brain abscess occurs usually in the temporal lobe of the cerebrum (more frequent) or in the cerebellum. It is to be the *most frequent cause of death from otitis media*.

The abscess may develop as a result of direct extension of the otologic infection, by means of thrombophlebitis, or along the preformed pathway. It may result from a previous skull fracture. An extradural abscess usually forms before the development of a brain abscess. Cerebellar abscesses from otitis media usually form through preformed pathways, whereas temporal lobe abscess results from seeding through bone erosion. Three clinical stages of brain abscess have been observed:

The first stage of initial encephalitis. Elevation of temperature, headache, nuchal rigidity, or other meningeal signs often are noted.

The second latent or quiescent stage. The symptoms are minimal or absent as an abscess is becoming organized and beginning to expand. This stage may last several weeks.

The patient may complain of headache, irritability, or lethargy.

The third stage of an expanding abscess. The symptoms and signs of this stage are due to *generalized increased intracranial pressure* and *localized pressure on brain centers*. They include severe and continuous headache (the most constant symptom), projectile vomiting, slowing of the pulse, Cheyenne-Stokes respiration, apathy and drowsiness, change in mental activity, disorientation, Jacksonian convulsions, ocular paralysis with pupillary changes, hemianopsia, aphasia, and elevated blood pressure. The most constant signs of increased intracranial pressure occur in the eyeground with blurring of the disc margins, hyperemia, or papilledema.

A left temporal lobe abscess in the right handed patient most commonly results in nominal aphasia. The next most common finding is paresis of the contralateral face and mouth, spreading to the extremities. A right temporal lobe abscess in a right-handed patient results in paralysis and numbness of the left side.

A *cerebellar abscess* usually gives more localizing signs. Among them are:

1. Ataxia (tendency to fall to the diseased side).
2. Ipsilateral hypotonia and weakness.
3. Spontaneous vertical or variable nystagmus.
4. Rapid emaciation.
5. Dysdiadochokinesia.
6. Intention tremor with past-pointing.

Diagnosis. Otogenic brain abscess is suggested by the characteristic clinical features described above in a patient with a coexisting or preexisting suppurative otitis media. Definitive diagnosis of brain abscess is made by special neurosurgical procedures which include lumbar puncture, ventriculography, angiography, electroencephalography, brain scanning, computerized tomography, and brain puncture. Ultimate diagnosis is made by finding the pus by brain puncture via a burr hole.

A lumbar puncture shows an increase in cerebrospinal fluid pressure and protein. A lumbar puncture is valuable in differentiating a brain abscess from other otogenic intracranial complications. However, it should be noted that a lumbar puncture in an advanced brain abscess with increased intracranial pressure can be followed by death as a result of herniation of the brain stem into the foramen magnum. A cerebellar abscess seldom reaches a large size since it is near the respiratory center. Compression of this center results in respiratory arrest and death. A temporal lobe abscess can keep expanding until it ruptures into the fourth ventricle causing fulminating meningitis. The treatment is primarily surgical drainage and an intense course of antibiotics.

Lateral Sinus Thrombophlebitis

Lateral sinus thrombophlebitis usually develops as a consequence of erosion of the lateral sinus plate by coalescent mastoiditis or chronic mastoiditis. First a perisinus abscess is formed. A mural thrombus then develops in the walls of the sinus, becomes infected, and spreads proximally and distally. The lumen of the sinus is eventually occluded by the increased thickness of the infected clot and by clotting of the stagnant blood. The ends of the infected clots then soften and infected material continues to escape into the systemic circulation (septicemia). Lateral sinus thrombophlebitis also can be caused by infections of the scalp or adjacent bones along the mastoid emissary vein. The usual organisms are hemolytic *streptococci* and *pneumococci*.

The clinical features vary according to the stage of the infection. When a perisinus abscess develops, headaches and malaise are usually the only symptoms. When the mural thrombus becomes infected within the vessel, septicemia develops with septic fever and chills. The primary symptoms of lateral sinus thrombophlebitis may be a persistent and *spiking type of fever* ("picket-fence"). Between bouts of fever the patient is often alert and feels well. *Anemia* may develop. With occlusion of the lumen of the sinus, interference with cerebral circulation results in headaches, papilledema, and increased cerebrospinal fluid pressure. When the thrombophlebitis spreads to the mastoid emissary vein, edema and tenderness may be produced over the mastoid process (Greisinger's sign). When the thrombophlebitis spreads to the jugular bulb and internal jugular vein, it may produce pain in the neck particularly on rotation of the neck. This may simulate neck rigidity of diffuse meningitis. The clot may be felt as a tender cord in the neck. The ninth, tenth and eleventh cranial nerves are occasionally paralyzed by the pressure of a clot in the jugular bulb.

The diagnosis is based upon *positive blood cultures* taken during the febrile phase, and the demonstration of lateral sinus obstruction by a *Tobey-Ayer or Queckenstedt's test*. With the spinal needle in place, digital pressure is applied over the internal jugular vein in the neck. This maneuver on the normal side produces a prompt rise in the spinal fluid pressure, but no increase on the side of the lateral sinus obstruction.

The treatment is always surgical. The sinus should be completely uncovered and the perisinus abscess eradicated. A needle aspiration of the sinus will determine if the sinus is occluded. If the sinus is occluded and there is an intrasinus abscess, it is necessary to open the sinus and remove the infected thrombus. When embolism or cavernous sinus thrombosis appears to be developing, *anticoagulation* together with intensive antibiotic therapy is recommended. Ligation of the internal jugular vein may be necessary.

Lateral sinus thrombophlebitis has been said to be the second common cause of death from otitis media.

Meningitis

Meningitis is the most common intracranial complication from suppurative otitis media and mastoiditis. There are two types of meningitis: localized and generalized.

1. *Localized or circumscribed meningitis* (no bacterial organism present in the spinal fluid).

2. *Generalized meningitis* (bacterial organisms are present in the spinal fluid). The patient suddenly becomes very ill and restless with severe headache, vomiting, and pyrexia, but soon loses consciousness. The classic signs of meningitis soon appear: stiffness of the neck, positive Kernig's sign, nausea and vomiting, and delirious and confused mental state or coma. In children, convulsions, a low, weak cry, and bulging fontanelle suggest meningitis until proved otherwise. The cerebrospinal fluid looks turbid and shows an increase in pressure and cell count. The protein concentration is raised, but the glucose and chloride are reduced. In generalized meningitis, numerous microorganisms can be found.

Treatment of meningitis is chemotherapeutic. Surgery is indicated in those patients developing meningitis secondary to chronic otitis media when the patient's general condition permits.

Otitis Hydrocephalus

Otitic hydrocephalus is a syndrome of increased intracranial pressure without a brain abscess following several weeks or more of acute otitis media. The condition occurs most often in children and adolescents. The most constant symptom is headache, often with a sixth nerve paralysis on the same side, sometimes with vomiting. Otherwise the patient looks and feels quite well. The most constant findings are *papilledema* and *spinal fluid pressure* exceeding 300 mmH₂O. Unlike localized meningitis the spinal fluid is clear without increased cell count or protein content. There are no localizing neurologic changes. Ventriculography fails to show a space-occupying lesion.

The exact mechanism of increased cerebrospinal fluid pressure is not known, but it is assumed to be due to increased production or decreased resorption of cerebrospinal fluid secondary to a previous meningeal inflammation. Treatment is repeated lumbar puncture. Subtemporal decompression may become necessary.

Congenital Cholesteatoma (Epidermoid) of the Temporal Bone

Congenital cholesteatomas or epidermoids arise from aberrant epithelial remnants and are, therefore, considered blastomatous malformations. Their predilective sites are the intracranial cavity, the diploe of the skull, and the spinal canal. In the base of the skull the temporal bone is the most frequent site.

Epidermoids account for about 0.2-1.5% of all intracranial tumors. The majority originate in the cerebellopontine angle where they account for 6-7% of all tumors. Their age incidence reveals a great scatter from birth to 80 years. The majority are recognized during the third and fourth decades with the onset of clinical symptoms occurring much earlier. They

affect males more frequently than females. Their delicate capsule with a whitish, mother-of-pearl sheen lends them a typical appearance.

Epidermoids are generally slow growing lesions which may remain asymptomatic for years. The irritative effect of their content, however, can produce symptoms of dysfunction and intense inflammation. Malignant changes occur infrequently. Diploic epidermoids are easily recognized, whereas intradural epidermoids are more difficult to identify.

Epidermoids may arise in the vicinity of, on the outer aspect of, or within the temporal bone. Epidermoids originating in any of these locations have certain characteristic features which may arouse suspicion of their presence. Examples of an epidermoid with origin in the typical locations within the temporal bone and cerebellopontine angle are discussed to portray their individual characteristics (Laryngoscope 85 suppl, 2, December, 1975).