OTORHINOLARYNGOLOGY

Return to Clinical Section Welcome Page

CONTENTS

INTRODUCTION EXTERNAL EAR EXTERNAL AUDITORY CANAL OTIC PREPARATIONS MIDDLE EAR CAUSES OF EAR PAIN (Table) INNER EAR DISEASES OR CLINICAL SYNDROMES OF OTOLOGICAL ORIGIN THE DIZZY PATIENT RHINOLOGY MOUTH AND PHARYNX AUDIOLOGY BASIC HEARING TESTS OTORHINOLARYNGOLOGIC DRUGS OF CHOICE (Table) INTRODUCTION

Otorhinolaryngology (Ear, Nose and Throat or ENT) faces the same problems in the military that are found in civilian medical practice, but the problems are compounded by (1) the exceptional environmental conditions, and (2) the fact that some of the symptoms experienced may degrade performance to the point that the safety of the Seabee is unable to function. The exceptional environmental conditions include heat, cold, moisture, dryness, high ambient noise levels, and poor hygiene conditions. These ENT conditions can elicit episodes of pain, vertigo, disequilibrium, and nausea. They may also introduce communication problems through temporary or permanent impairment of auditory function. In addition, these conditions may be of sudden onset in apparently normal individuals, such as external otitis or an acute tonsillitis.

These lectures describe clinical ENT issues and audiology. Seabee physicians may find themselves at long distances from large medical facilities when ENT problems arise, or they may have to care for patients until they can get an appointment at the nearest facility; therefore, this section is intended to assist the physician with common clinical problems.

EXTERNAL EAR

Anatomy of the pinna

- Helix: Crus, Ant. Incisure
- Antihelix: Crura, Triangular Fossa
- Concha: Cymba, Cavum
- Scaphoid Fossa
- Tragus
- Antitragus
- Intertragic Incisure
- Post-Auricular Sulcus

Trauma

- Lacerations
 - Atraumatic cleaning with copious irrigation
 - Suture with fine (6-O) monofilament nylon
 - Primarily interrupted skin sutures
 - Occasional through and through sutures
 - Antibiotics
 - Pressure dressing
- Hematoma
 - Utilize aseptic skin technique
 - Needle aspiration or Incision and Drainage
 - Pressure dressing
- Perichondritis
 - Etiology: External Otitis or Trauma
 - Antibiotics: coverage for Staph. and Pseudomonas
 - Debridement of dead tissue
- Keloids
 - Intralesional Cortisone
- Burns
 - Gentle cleansing
 - Sulfamylon cream/dressing
- COMEDONES or BLACKHEADS
 - Nothing
 - Skin prep with direct small ring extraction

Infections

- Erysipelas
 - Usually a Staphylococcal infection
 - Staph. resistant PCN: Oxacillin, Augmentin
- Herpes Zoster Oticus

EXTERNAL AUDITORY CANAL

Anatomy

- 25 mm to posterior/superior TM
- 31 mm to anterior/inferior TM
- Outer 1/3:
 - Thicker skin
 - Pilosebaceous glands
 - Cerumen glands
 - Incisure of Santorini
- Inner 2/3:
 - thinner skin
 - vertical and horizontal wrinkles
 - no hair and minimal glands

Cerumen impaction

• Water, alcohol, or acetic acid irrigations

Foreign bodies

• Inert, hydroscopic, round or animal

Epithelial growths

• Inclusions, benign or malignant growths

Infections

- EXTERNA
 - Acute
 - Recurrent
 - Chronic
 - Eczematoid
 - Seborrheic
- FURUNCLE
 - Outer 1/3
 - Usually Staphylococcal
 - Often spreads to periauricular cellulitis
 - scant pus
 - hot compresses, oral antibiotics, I&D necrotic area
- INFECTIVE OTITIS (Swimmers Ear)
 - Debridement
 - Intra-auricular wick
 - Antibiotic drops
- OTOMYCOSIS
 - Debridement
 - Antifungal medications, (Domeboro's, Lotrimin)
 - Keep ear dry

- Acid-Alcohol drops
- ECZEMATOID EXTERNA
 - Allergic reaction
 - Look for contact substances or ID reaction infection elsewhere
 - Gentle cleaning
 - Hypoallergenic medication
 - Topical Cortisone preparations
- SEBORRHEIC EXTERNA
 - Look for associated skin or scalp infections
 - Treatment with anti-seborrheics, cortisone preparations, and skin emollients

OTIC PREPARATIONS

The most common organisms found in external otitis are *Staphylococcus aureus*, *Pseudomonas aeruginosa*, *Proteus*, *Bacteroides fragilis*, *E. Coli*, Klebsiella, and Enterobacter. Thorough cleaning of the ear canal is the most important first step in the treatment of any type of external otitis.

The next step is to choose a topical otic agent. For prevention as well as treatment, most otic preparations contain some type of acid, such as a boric or acetic acid since most organisms flourish best when the canal is alkaline. Most of your "swimmer's ear" prophylactics are a mixture of white vinegar and rubbing alcohol. Most otic preparations also contain antibiotics. Neomycin is active against Staphylococcus and Proteus, but the most common Pseudomonas strains are resistant. Polymyxin B and polymyxin E (Colimycin) are bactericidal to most gram negative organisms, notably Pseudomonas, but not proteus, Bacteroides fragilis (found in 13% of infected cholesteatoma), gram positive organisms. Chloromycetin otic is a bacteriostatic preparation against all common pathogens; as it comes in an acid carrier, chloromycetin is often extremely painful in the ear canal and or middle ear, so certain patients are given the powdered Chloromycetin. "Chlor" is used when there is a neomycin-polymyxin failure in a chronic disease or when a reliable anaerobic culture suggests Bacteroides fragilis. Garamycin is available in an ophthalmic solution to instill in ears; its spectrum includes Pseudomonas and most other pathogens of chronic suppurative otitis except Bacteroides fragilis.

At this time there is no otic preparation containing specific fungicides, however topical Amphotericin B (Fungizone) and Nystatin (Mycostatin) both being effective when used in treatment of Candidiasis (Monilia), can be adapted. Non-specific antiseptics effective against Aspergillus and Candida are 2% acetic acid and aluminum acetate solution (Otic Domeboro), 3% boric acid in 70% alcohol (Swimmer's Otic), or 25% m-cresyl acetate (Cresatin or Cresylate). Gentian Violet 2% in 95% alcohol, topical mercurials, or a boric acid and iodine mixture have been used in chronic mastoid cavity infection after the debris and secretions are removed, but they are messy. Athlete's foot solutions and ointments can be used if there's no TM perforation.

Anti-inflammatory agents, most notably hydrocortisone, ar used to decrease mucosal and cutaneous edema in bacterial infections or for the treatment of psoriasis and seborrheic dermatitis.

For dermatitis, the steroid should be used alone or in a low sensitizing preparation like Orlex HC or VoSol HC.

Anesthetic agents such as Auralgan or Lidosporin, which contain benzocaine or lidocaine, may provide some relief of pain in otitis media and bullous myringitis and some relief from itching in dermatitis. A few people become allergic to these medications.

There are ceruminolytic agents in use, the most popular agent being carbamide peroxide in glycerol (Debrox). Using this agent or simple 3% peroxide solution may take several days to soften hard wax. Ceruminex, an agent that softens wax rapidly, has been cited as occasionally causing a severe eczematoid reaction and should be limited to 15 to 30 minutes of skin contact and then thoroughly removed.

Remember also, the disadvantages of otic preparations: All drops produce debris requiring further cleaning after use. Most preparations are painful, due to the acid and alcohol base, when used in the middle ear. The water based suspensions or ophthalmic preparations cause less pain and reaction with the mucosa and are the best preparations applied in treatment of a perforated tympanic membrane.

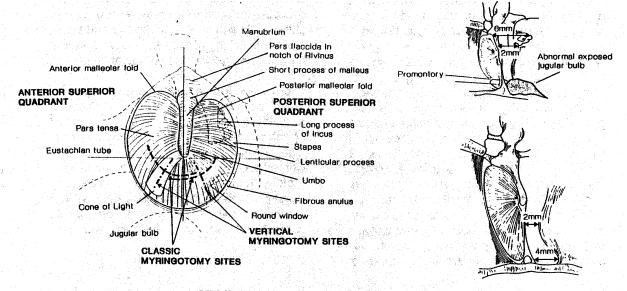
Use your otic preparations wisely. Clean the ear first, use only what is necessary, discontinue as soon as possible and remove remaining debris.

MIDDLE EAR

Anatomy

- Tympanic Membrane-Landmarks
 - Pars Flaccida Shrapnell's membrane over the notch of Rivinus
 - Pars Tensa
 - ➢ Manubrium and Umbo
 - > Short process
 - \succ Light reflex
 - Behind the membrane
 - Chorda Tympani nerve
 - ➢ Incus
 - Stapedial Tendon
 - Round window niche
 - Promontory
 - Special features
 - Migratory surface epithelium
- Tympanic cavity
 - Epitympanum, Mesotympanum, and Hypotympanum
 - Mastoid antrum and air cells are connected to the Tympanic Cavity through the Addus an Antrum

- Tympanic Plexus
 - Greater superficial Petrosal Nerve and Caroticotympanic Nerve form the Vidian Nerve.
 - Innervates the Sphenopalatine ganglion causing stimulation of nasal glands and the lacrimal glands.
 - The Lesser Superficial Petrosal and Deep Petrosal Nerve innervates the otic ganglion which stimulates the Parotid gland



Middle ear anatomy and myringotomy sites

Physiology

- The Eustachian tube is closed except when swallowing, yawning or forced open by air pressure greater than 20 to 40 mm Hg.
- Negative pressure differential of 80-90 mm of Hg locks the tube against opening by muscle or Valsalva.

The Valsalva Maneuver

The procedure for self or mechanical inflation of the middle ear space is termed the Valsalva maneuver. It has been frequently observed in young student pilots and aircrewmen receiving ear blocks in the low-pressure chamber or in flight during rapid descent, that they were unable to perform a proper Valsalva, frequently because they did not know the correct technique or were trying too hard. Several physiological conditions make the Valsalva maneuver more difficult. They are flexing the head or the chest, twisting the head to one side, pressure on the jugular vein, and being in the prone position.

The Valsalva maneuver requires the nose and mouth to be closed and the vocal cords open. Air is

then forced into the nose and nasopharynx forcing open the Eustachian tube and increasing the pressure in the middle ear space. This can be observed as a bulging of the tympanic membrane, especially in the posterior superior quadrant.

The most frequently observed problems with the students were fear that they would damage or rupture their eardrums, closing the vocal cords when they build up pressure like in the M-1 maneuver, and straining so hard that marked venous congestion in the head further prevents opening of the Eustachian tube.

Although it is possible to rupture the tympanic membrane when it is abnormally weak from previous disease, simple inflation done properly has little danger. Repeated overinflation does carry some risk and is discussed under politzerization and round window rupture.

One of the best methods to prevent vocal cord closure is to instruct the patient or aircrewman to close his nose with his fingers and then attempt to blow his fingers off his nose, causing the nose to bulge from the pressure. The buildup of pressure should be rapid and sustained no longer than one to one and a half seconds to prevent the venous congestion that reduces the efficiency of the Eustachian tube function.

Should the examiner fail to see any movement of the tympanic membrane when he is evaluating the patient for Valsalva, he should then look for the small, quick retraction movement of the Toynbee maneuver, accomplished by closing the nose and swallowing. If a Toynbee is present and the aircrewman feels pressure in his ears during Valsalva, has no sign of ear disease, and no history of problems with pressure changes, he usually can be qualified for aviation. The best evaluation for candidates is, of course, the low-pressure chamber or an actual unpressurized flight with rapid descent. Difficulty with pressure equalization during SCUBA diving is often a poor prognosis for aviation.

Acute otitis media

- Etiology: eustachian tube dysfunction
- Causes
 - Anatomic
 - UTI
 - Allergic rhinitis
 - Adenoiditis or nasopharyngitis
 - Sinusitis
- Symptoms
 - Fullness
 - Pain
 - Decreased hearing
 - Rarely dizziness or TM perforation
- Treatment
 - Antibiotics

- ➤ Amoxicillin x 7-10 days
- ➢ Emycin/Sulfa
- > Augmentin
- ➤ TMP-SMX
- ➢ Ceclor
- ➢ Ceftin
- > Suprax
- Decongestants
 - > Sudafed
 - > Entex
- Pain medication
- Hydration
- Treat any other associated conditions

Chronic or recurrent otitis media with effusion

• Consider pressure equalization (PE) tube

Bullous myringitis

- May be form of otitis media
- Often seen after viral URI or mycoplasma pneumonia
- Signs & symptoms
 - Sudden onset of deep ear pain
 - May have serosanguinous drainage
 - Single of multiple bullae form on TM and adjacent bony wall
- Treatment
 - Same as acute otitis media
 - Rupture bulae with suction tip but do not do myringotomy
 - Auralgan ear drops occasionally helpful warm

Otitis medial complications

- Mastoiditis
 - Usually 1-2 weeks after acute illness
 - Pain deep or behind ear
 - Pus formation beneath temporalis muscle
 - Treatment
 - IV Cefuroxime plus metronidazole
 - > Chloromycetin
- Chronic perforation
 - Frequent suction cleaning of drainage
 - Avoid water in ear
 - Cortisporin otic drops only if draining

Cholesteatoma

- Squamous epithelium growing in the middle ear space
- Usually from a pars flaccida perforation or retraction pocket or similar pathology in the posterior superior quadrant of the pars tensa or a marginal perforation
- 90% require surgical intervention and one year minimum follow-up
- Should be on limited duty
- Could lead to serious complications, a surgical emergency, or even death if left untreated or improperly followed-up.

Traumatic perforation of the TM

- Keep water out of ear
- Suction cleaning only
- No ear drops unless ear is infected and draining
- Get baseline audiogram
- Marginal stimulation with trichloroacetic acid (TCAA) 25%

Perforations usually heal within three weeks, but the patient must avoid any significant barometric pressure changes as the perforation nears closure, and at no time should water or other fluids be allowed in the ear. Keep The Ear Dry! Never use ear drops. unless a true infection with purulent drainage develops and then use only the suspension preparations.

Chronic Perforation of the TM

Small, dry, central perforations may be closed by cauterizing the edge of the perforation with trichloroacetic acid. It can be left open or one may elect to place a small patch made from cigarette paper or other thin paper over the perforation. Usually the patch is moistened in antibiotic drops before application.

Large perforations with a dry middle ear may be closed by a tissue graft if the Eustachian tube is functioning. Testing of this function is fairly accurate by tympanography. Poor or absent Eustachian tube function gives surgery a decreased chance for success. If the ossicles show fixation or if there is considerable scarring with adhesions, hearing might decrease somewhat further even though the perforation is closed, as a result of the poorer transmission of sound and the cancellation effect of sound striking both windows at the same time. A perforation, per se, which allows for equalization of pressure between the middle ear and the atmosphere does not affect flying. Sudden cold or hot air or water and loud noise may cause vertigo more easily in the perforated ear. Of course, water in a perforated ear usually leads to infection and drainage.

Barotrauma

Aerotitis media occurs rather frequently in the aviation community and is directly related to the function of the Eustachian tube in equalizing the pressure between the atmosphere and the middle ear space. The tympanic end of the Eustachian tube is bony and usually open, whereas the pharyngeal end is cartilaginous, slit-like, and closed, acting like a one-way flutter valve. Opening of the Eustachian tube occurs with the contraction of the levator and tensor veli palatini muscles

during acts of chewing, swallowing, or yawning. As one ascends to altitude, the outside pressure decreases, and the greater middle ear pressure forces open the "flutter valve," pharyngeal end of the Eustachian tube every 400 to 500 feet to about 35,000 feet, and then every 100 feet thereafter. During descent, the collapsed, closed, pharyngeal end of the Eustachian tube prevents air from entering the tube. The increasing relative negative pressure in the middle ear further holds the soft tissues together, and muscular (active) opening of the Eustachian tube must be accomplished before the differential pressure reaches 80 or 90 mm Hg. Once this magnitude of differential pressure is established, muscular action cannot overcome the suction effect on the closed Eustachian tube, and the tube is said to be "locked." This relative negative pressure not only retracts the tympanic membrane but pulls on the delicate mucosal lining, leading to effusion and hemorrhage. Pain may be severe, with nausea and occasionally vertigo. On rare occasions rupture of the tympanic membrane has been seen, and some aircrewmen have developed shock or syncope.

Otoscopic presentations vary greatly, but they can range from a retraction of the tympanic membrane with the classic backward displacement of the malleus, a prominent short process, and anterior and posterior folds, to hyperemia or hemorrhages in the tympanic membrane. There may also be varying amounts of serous and bloody fluid visible behind the membrane.

Active treatment is directed toward equalization of pressure, relief of pain, and prevention or treatment of infections in the ear, Eustachian tube, or nasopharynx. In an aircraft or low-pressure chamber, descent should be stopped, and, if possible, there should be a return to a higher altitude where equalization can be attempted using the Valsalva maneuver or Politzer method. Descent should then be gradual, if possible. Middle ear inflation (politzerization) should be done especially if a negative pressure appears to remain on the ground and there is pain present. Caution should be exercised if there is an upper respiratory infection present. Oral decongestants may be helpful and are recommended, but the effect of antihistamines is questionable. In cases of thick effusion and poor Eustachian tube function or inability to Valsalva, daily or every other day politzerization, but thick mucoid or organized blood must be removed by myringotomy if it has not cleared after two or three weeks of intensive therapy. Antibiotics are used only when infection is present in the upper respiratory region or develops during treatment.

CAUSES OF EAR PAIN

I. Primary Pain (originating in ear)

A. External ear

- 1. Furunculosis
 - 2. Foreign body in external ear canal
 - 3. External otitis
 - 4. Abscess of auricle
 - 5. Perichondritis of auricle
 - 6. Eczema of meatus
 - 7. Impacted cerumen
 - 8. Frostbite of auricle
 - 9. Herpes simplex and herpes zoster oticus
 - 10. Malignant and benign growth of external ear
 - 11. Fungal infections (otomycoses)
 - 12. Acute myringitis and myringitis bullosa
 - 13. Trauma of TM and external canal
- B. Middle ear or mastoid
 - 1. Acute otitis media
 - 2. Acute mastoiditis
 - 3. Acute aerotitis media (barotrauma)
 - 4. Acute eustachian tube obstruction
 - 5. Complications of otitis media and mastoiditis
 - a. Petrositis
 - b. Facial paralysis
 - c. Labyrinthitis
 - d. Inner ear deafness
 - e. Subperiosteal abscess
 - f. Extradural abscess
 - g. Subdural abscess
 - h. Brain abscess
 - i. Venous sinus thrombophlebitis
 - j. Meningitis
 - 6. Malignant and benign growth of middle ear and mastoid process
- C. Idiopathic (tic-like pain confined to ear)
 - 1. Geniculate complex of VIIth cranial nerve
 - 2. Tympanic branch of IXth cranial nerve

II. Secondary (referred) pain

- A. Larynx
 - 1. Cancer
 - 2. Ulceration
 - 3. Perichondritis and chondritis
 - 4. Arthritis of cricoarytenoid joint
- B. Pharynx (nas-, oro-, and hypopharynx)
 - 1. Pharyngitis
 - 2. Acute tonsillitis (palatine, lingual, pharyngeal)
 - 3. Peritonsillar abscess

- II. Secondary (referred) pain, continued
 - 4. Retropharyngeal abscess
 - 5. Ulceration
 - 6. Postadenoidectomy or posttonsillectomy
 - 7. Nasopharyngeal fibroma
 - 8. Malignant growth
 - C. Oral cavity
 - 1. Dental neuralgias
 - a. Dentine exposed, pulp inflamed, or nerves dying
 - b. Unerupted or impacted wisdom or molar teeth
 - c. Traumatic occlusion of teeth, faulty closure of jaw, and improper fit of dentures
 - 2. Acute diffuse glossitis or stomatitis
 - 3. Carcinoma of tongue
 - D. Esophagus
 - 1. Foreign body
 - 2. Hiatal hernia
 - 3. Inflammation
 - 4. Malignant and benign growths
 - E. Miscellaneous
 - 1. Mumps
 - 2. Acute thyroiditis
 - 3. Trigeminal neuralgia
 - 4. Sinuses
 - a. Inflammation
 - b. Malignant or benign growth
 - 5. Temporomandibular arthritis
 - 6. Erysipelas
 - 7. Raynaud's disease
 - 8. Chilblains
 - 9. Post auricular lymphadenitis
 - 10. Glossopharyngeal neuralgia
 - 11. Sphenopalatine ganglion cephalgia
 - 12. Elongation of the sphenoid process
 - 13. Involvement of upper three cervical nerves
 - a. Whiplash injury or other cervical spine lesions
 - b. Inflammation such as tabes dorsalis or herpes
 - 14. Angina pectoris
 - 15. Thoracic aneurysm
 - 16. Innominate artery aneurysm
 - 17. Affections of nasal passageway
 - 18. Affections of lung and bronchus

Politzerization

Politzerization is the mechanical inflation of the middle ear usually required for treatment of acute ear and sinus blocks, chronic Eustachian tube dysfunction, or middle ear disease. To perform this procedure, one needs a source of pressure, either an air pump or rubber bag, with a one-way valve. For the air pump, it is most important to have variable control of the pressure and a pressure gauge, if possible. Most pressure/vacuum units in the Navy have a pressure gauge calibrated in pounds per square inch. If no gauge is present, the starting pressure should just be sufficient to blow off a lightly applied finger. When a pressure gauge is available, initial attempts should be done with ten pounds per square inch or less. To seal and deliver the pressure into the nose, an olive tip of metal, hard rubber, or glass is the most efficient. This tip may be attached to an atomizer if smoke or mist is desired for diagnostic or therapeutic reasons. If the patient has a very thin tympanic membrane, lower pressure must be tried first. An explanation to the patient is important to assure cooperation and prevent sudden movements that could injure the nose.

The first attempt at politzerization should be done by inserting the olive tip into a nostril, getting a good seal but not striking the vestibule or septal walls. The opposite naris is occluded, and the patient is instructed to repeat K-K-K-K, loudly and sharply, as a one second burst of air is delivered. A characteristic soft palate flutter sound is heard if the procedure is performed correctly.

If no results are obtained with this technique, the patient is instructed to swallow, and as the thyroid notch raises up, air pressure is again applied in the nose. For people who have trouble with a dry swallow, a sip of water may be given. In the low-pressure chamber, this method is most often used to get maximum opening of the Eustachian tube. It must be remembered that with the water technique, prolonged or high pressure might cause damage to the tympanic membrane with even a remote possibility of damage to the round window membrane and inner ear. As it is important to look at the patient's tympanic membranes before inflation, it is equally as important to observe them afterwards to determine the extent or success of the procedure.

INNER EAR

Anatomy

Situated medial to the middle ear entirely within the petrous portion of the temporal bone lies the inner ear. It is composed of dense, compact bone two to three millimeters thick, forming the osseous labyrinth. This is divided into semicircular canals, vestibule, and cochlea. Within the bony labyrinth is a membranous counterpart. The supporting fluid outside of the membranous labyrinth is perilymph. It is somewhat similar to cerebrospinal fluid and is high in sodium content. The fluid inside the membranous labyrinth, endolymph, has a high potassium content.

The cochlea is a two and a half-turn coil about a central core called the modiolus, with the apex pointing anteriorly and laterally. There are three compartments. The first two, the scala vestibuli associated with the oval window and the scala tympani associated with the round window, contain perilymph and are joined at the apex of the cochlea through the helicotrema. The third or central compartment is the scala media or cochlear duct, containing endolymph. It contains the neural end organ of hearing, the organ of Corti, which rests on the thick basilar membrane that separates this compartment from the scala tympani. The delicate Reissner's membrane separates the scala media from the scala vestibuli. The organ of Corti contains about 24,000 hair cells arranged throughout the cochlea as a single row of inner cells and from three to five rows of outer cells. Between them, they form a somewhat triangular tunnel of Corti that has its own slightly different fluid, cortilymph. It is known that high frequency sounds stimulate the hair cells near the vestibule, and low frequency sounds stimulate those near the apex. The area of the promontory or basilar turn of the cochlea is stimulated by frequencies in the range of 3000 to 5000 Hz; it appears to be the most vulnerable to acoustic trauma, probably from the shearing force in the fluid so near the stapes footplate and the beginning curve in the scala.

Trauma

Temporal bone fractures are of two types for the most part of two types. The longitudinal or middle fossa fracture that parallels the long axis of the petrous pyramid is usually due to forces applied to the temporoparietal region. The middle ear is always damaged. The tympanic membrane is torn and bleeds. The labyrinthine capsule is usually spared, as is the facial nerve. Longitudinal temporal bone fractures are four times more frequent than the transverse variety. The transverse or posterior fossa fractures usually result from forces applied to the occipital or occipitomastoid region. There is essentially a fracture of the labyrinth that spares the middle ear. There may be hemotympanum, but rarely rupture of the tympanic membrane. Usually, there is both cochlear and vestibular function loss, and the facial nerve is damaged in the internal auditory meatus or horizontal portion. Only sterile ear instruments should be used for examination, and *dry ear precautions must be taken*.

Initial treatment should include cranial checks, prophylactic antibiotics, and a complete neurological evaluation. The patient should be moved to the care of a neurosurgeon/otologist as soon as condition permits. A baseline audiogram is valuable if the patient's condition permits.

Barotrauma

In the past few years, an increasing number of cases of barotrauma to the inner ear have been reported from the diving community, and several cases of proven rupture of the round window membrane have been reported or evaluated at the Naval Aerospace Medical Institute (NAMI). These have been associated with overly aggressive use of the Valsalva maneuver to clear what the patient thought was an ear block. In reality, the problem was an over-inflated middle ear and distended tympanic membrane, which gives a similar blocked feeling, but usually has no pain. When the round window membrane ruptures, there may be variable degrees of tinnitus and persistent or positional vertigo, often with nausea and vomiting. Calorics are usually diminished on the involved side, and a sensorineural hearing loss, often across the board, is present with poor discrimination of words.

The key to successful treatment is early suspicion and diagnosis and immediate repair by the otologist. Most complete recoveries have had repairs within 48 hours. A quick, simple tuning fork test will separate nerve loss from a conductive loss.

Nystagmus

The search for the presence or absence of spontaneous or positional nystagmus is an integral part of the otoneurological examination and the fitness for duty examination.

Nystagmus is called right or left according to the direction of the rapid eye movement or quick component. When nystagmus is provoked only in the direction of the quick component, it is termed "first degree." When nystagmus is also noted in forward gaze, it is "second degree," and with nystagmus present in all directions of gaze, it is "third degree." Nystagmus is further categorized as vertical, oblique (rare), horizontal, or rotatory. Proper evaluation calls for observation of the eyes in the right, left, upward, downward, and primary positions. If the patient is asked to look too far on lateral gaze, a few flicks of nystagmus are frequently seen and are a normal phenomenon of accommodation. After the test for spontaneous nystagmus, tests for positional nystagmus are carried out with the patient's eyes in the straight ahead position. The method most often used is that of Cawthorne, Dix, and Hallpike. The patient is rapidly placed supine with the head hanging over the edge of the table, and the eyes are observed for 60 seconds. The patient is then raised up and then returned to the hyperextended position with the head in one direction, again for 60 seconds. The procedure is repeated in the opposite direction. Nystagmus, if present should be immediately recorded as to type, direction, amplitude, and intensity. The position should be held until the nystagmus subsides; however, if it persists longer than 60 seconds, it is considered permanent. In older persons where vertebral artery occlusion may be the cause of the nystagmus and vertigo, one must use caution and good judgement to assure that the patient is not left in this position too long.

Unidirectional nystagmus is usually of peripheral origin and occurs in the horizontal plane. The quick component is toward the uninvolved ear. Caloric response is usually hypoactive or absent. When caloric tests are normal, unidirectional nystagmus may be of central origin. The nystagmus is usually the strongest, and often only present, when gaze is directed toward the side of the quick component (first degree). The diagnostic characteristics of nystagmus are given in the following tables.

	Peripheral	Central
Latency	2 to 20 seconds	None
Persistence	Disappears within 50 seconds	Lasts longer than one minute
Fatigability	Disappears on repetition	Repeatable
Positions	Present in one position	Present in multiple positions
Vertigo	Always present	Occasionally absent, & only nystagmus present
Direction of nystagmus	One direction	Changing directions in different positions
Incidence	85 percent of all cases	10 to 15 percent of all cases

Differences between peripheral and central positional nystagmus

	Peripheral (Labyrinth, Vestibular Nerve	Central (CNS)
Form	Horizontal-rotatory	Horizontal; vertical; diagonal; rotatory; multiple; retractorius; convergence; pendular; alternating
Frequency	1/2-6 seconds	Any frequency, usually low or variable at long intervals (weeks to months)
Intensity	Decreasing intensity	Constant
Direction of fast component	Towards "stimulated" labyrinth or away from "destroyed" labyrinth	Towards side of CNS lesion
Duration	Minutes to weeks	Weeks to months
Dissociation between eyes	None	Possible
Vertigo	Present	Present or absent
Cochlear signs	Frequently present	Seldom present
Autonomic nervous system signs	Definite	Less definite or absent
Past pointing and falling	Direction of slow phase	Direction of fast phase

Spontaneous Vestibular Nystagmus

	Peripheral (Labyrinth, Vestibular Nerve)	Central (CNS)
Hallucination of movement	Definite	Less definite
Onset	Usually paroxysmal	Seldom paroxysmal
Intensity	Usually severe	Seldom severe
Duration	Minutes to weeks	Weeks to months
Influenced by head position	Frequently	Seldom
Nystagmus	Present	Present or absent
Autonomic nervous system symptoms	Definite	Less definite or absent
Tinnitus	Frequently present	Seldom present
Deafness	Frequently present	Seldom present
Disturbances of consciousness	Seldom present	More frequently present
Other neurological signs	Usually absent	Frequently present

Differentiation of central from peripheral vertigo

Multidirectional nystagmus is suggestive of central involvement (i.e., a lesion anywhere in the brain). Most often, however, it results from a posterior fossa lesion where the bulk of the vestibulocerebellar units are located. The quick component is usually permanent and toward the side of the lesion. True vertigo is less frequent, and ataxia may be evident in central lesions. The table on the previous page provides a listing of diagnostic criteria helpful in differentiating between central and peripheral vertigo.

Drugs often produce characteristic nystagmus. Opium and Demerol produce a vertical downward nystagmus. Positional nystagmus is found with barbiturates and alcohol. Any patient who demonstrates a spontaneous positional nystagmus with no other abnormality of labyrinthine function should be checked for barbiturate ingestion.

A most interesting and characteristic positional nystagmus is seen with alcohol intoxication. The

nystagmus is typically in two phases and is often recorded as PAN (positional alcohol nystagmus) I and II. As little as 0.02 percent blood concentration may produce both phases. Phase I begins about 30 minutes after ingestion, as the blood alcohol peaks, and lasts approximately three and a half hours. The nystagmus is always in the direction of the gaze or toward the position of the head (a right-beating nystagmus appears with right gaze, head turned to the right side or if the right of the patient's head is down in the lateral position). There is a gradual diminution after the peak and an intermediate period of about 1.7 hours in which there is no nystagmus. Approximately 5 hours after the initial ingestion, PAN II begins; the nystagmus is in the opposite direction of the gaze or lateral head position and persists for several hours after the blood alcohol level has disappeared. PAN II nystagmus is greatest when the "hangover" symptoms are greatest.

DISEASES OR CLINICAL SYNDROMES OF OTOLOGICAL ORIGIN

The majority of cases of dizziness which the Medical Officer will see associated with disease or injury of the inner ear or eighth cranial nerve are acute labyrinthitis, epidemic vertigo, vestibular neuronitis, Meniere's disease, acoustic neuroma, benign paroxysmal positional vertigo, and trauma. These must be differentiated from the many causes of dizziness or vertigo (see table on following page).

Labyrinthitis

Labyrinthitis has many classifications, but, in general, it is serous, diffuse, destructive, or toxic. Serous and diffuse destructive labyrinthitis are associated with otitis media, cholesteatoma, or ear surgery. When the disease is of the serous type, the vestibular and cochlear functions are depressed, with the vestibular symptoms usually preceding the cochlear depression by a few hours to several days. There is usually spontaneous nystagmus to the opposite ear, nausea and vomiting, true vertigo, ataxia, past-pointing, and loss of hearing.

In patients with chronic ear disease, especially cholesteatoma, a fistula test should be performed by exerting pressure and then suction using a pneumo-otoscope. Production of nystagmus and vertigo indicates the presence of a labyrinthine fistula. An acute, initially severe, and sudden onset of symptoms may be associated with the erosion into the labyrinth; however, in cholesteatoma, the lining or sac protects the labyrinth, and only quick head movements or pressure applied in the canals cause vertigo in many cases. Patients who have had ear surgery or manipulation of the stapes may have all the usual findings, except nystagmus.

Causes of dizzyness and/or vertigo

Otological

Impacted cerumen Trauma to the inner ear or eighth cranial nerve (commotio labyrinth) Dysfunction of the eustachian tube

Acute otitis media

Labyrinthitis Labyrinthine fistula Bilateral nonfunctioning equilibrial labyrinthitis Motion sickness Meniere's disease Lermoyez syndrome Vestibular neuronitis Vasculitis involving the internal Auditory artery or vestibular emmisary arteries Tumors of middle ear and inner ear Ototoxic drugs "Focal infection" from tonsils, adenoids periapical tooth abscesses, and chronically infected sinuses Sinusitis, acute or subacute

Systemic

Allergic reactions involving the inner ear Dizziness of the aged due to indeterminate vascular cause or endorgan degeneration due to aging process Cardiac diseases Hypertension (paroxysmal) Hypotension (syncope) Hyperactive carotid reflex Blood dyscrasias (anemia, leukemia, lymphomas, reticulosis, polycythemia, purpura) Cogan's syndrome (nonsyphilitic interstitial keratitis with vestibular symptoms panarteritis nodosa) Episodes of hypoglycemia Hypocortoadrenalism Cervicl myalgia

Neurological

Degenerating and demyelinating diseases especially multiple sclerosis Posterior fossa lesions Fractures, cystic arachnoiditis, syringobulbia, platybasia and Arnold-Chiari malformations Neoplasms and subdural hematomas Supratentorial lesions with displacement of the brainstem Migraine-like symptoms Convulsive disorders (vestibular epilepsy, vertiginous epilepsy Temporal lobe lesions with irritation cortical vestibular areas Toxic-infectious conditions Aseptic meningencephalitis Brain abscess Common viral diseases such as mumps, measles, whooping cough Vascular, including atherosclerosis, thrombosis, embolic occlusion, and hemorrhage, especially in vessels to the brainstem

Ophthalmological

Nonconcomitant strabismus Refractive errors Optokinetic vertigo

Psychogenic

Tension-anxiety state Conversion reactions Neuroses (agaraphobia, claustrophobia) Hyperventilation syndrome In isolated serous labyrinthitis, there is usually return of labyrinthine function over weeks or months. If any fistula is suspected or injury occurred in surgery, systemic antibiotics are indicated. With fistulas, there is often a permanent nerve-type hearing loss, and some patients have chronic positional vertigo.

Suppurative labyrinthitis results in violent and sudden onset of vertigo, disturbed equilibrium, nystagmus, and vomiting. Cochlear and vestibular responses are lost. Complications such as meningitis or brain abscess lead to toxic symptoms of headache, malaise, and fever. Vigorous therapy with antibiotics and surgery must be instituted, and some small mortality can be expected even with treatment. For those who recover, there is usually no recovery of the cochlear or vestibular responses, and three to five weeks are required for compensation. Return to a flying status is not recommended, except in the mildest cases. It is often impossible to be sure of complete eradication of disease, and there is questionable compensation for loss of hearing and labyrinthine function and occasional residual ataxia.

Toxic labyrinthitis is one of the most common types seen, and a great deal of disagreement remains about its classification. The etiology ranges from acute febrile diseases to toxic or chemical substances to idiopathic. The most common characteristic is whirling vertigo with gradual onset reaching a maximum in 24 to 48 hours, and at its height, there may be nausea and vomiting. There may be no cochlear or vestibular abnormalities in those cases associated with or following acute febrile illness, but when associated with drugs, either system may be affected. Usually there is recovery from vertigo in three to six weeks.

Most commonly, toxic labyrinthitis is associated with pneumonia, cholecystitis, influenza, allergy, extreme fatigue, overindulgence in food or alcohol, and certain ototoxic drugs. Palliative treatment with antivertiginous drugs and bed rest is helpful. The physician should always be aware of a missed or changing diagnosis with these patients. They should not be dismissed with the "they always get well" attitude.

Epidemic Vertigo

Although to a great extent this disease may be of central origin, it is important to differentiate it from other vertiginous conditions, and this can often only be done by exclusion. Characteristically, symptoms are acute onset of severe dizziness, nausea, vomiting, a slight fever, headache, and asthenia, with a duration of several weeks to months. Recovery, however, is usual. There is usually an epidemic character to the disease, and it is associated with either an upper respiratory infection or gastroenteritis. Caloric and audiological tests usually are normal, but spinal fluid may show some lymphocytic cells. Cases with gastrointestinal symptoms are more frequent in mid-January, and those with upper respiratory symptoms occur in the autumn. Laboratory tests are of little value.

Treatment is supportive, with variable help from antivertiginous and antinausea drugs such as Dramamine, Vontrol, Torecan, and Tigan. These patients should be able to return to flying within one month after all symptoms have ceased.

Vestibular Neuronitis

Vestibular neuronitis is characterized by an attack of sudden, debilitating vertigo, nausea, vomiting, and spontaneous nystagmus. In most cases, there appears to be an antecedent or concomitant infection in the upper respiratory tract, maxillary sinuses, or teeth. The cochlea is spared, but one or both of the labyrinths have abnormal calorics. Vestibular symptoms decrease somewhat after a few hours, but they remain fairly severe for the first week, slowly decreasing over the next four to eight weeks. About 70 percent of these patients have permanent, decreased caloric function.

Management is directed toward supportive treatment of the symptoms and an aggressive workup to rule out other possible diagnoses. Vestibular neuronitis is a self-limiting disease, although return to work may require from three to twelve weeks. Generally, an aviator is permanently grounded for military flying because of the sudden debilitating nature of the attacks which can be recurrent even as long as four years after the initial attack.

Meniere's Disease

Although much disagreement persists as to whether this is a disease or a symptom complex, and its etiology is still unknown, there is usually the classical triad of episodic vertigo, tinnitus, and deafness. The average age of onset is 44 (Cawthorne & Hewlett, 1954), and it is predominantly unilateral, with only about ten percent of the patients having bilateral involvement.

The onset of symptoms is insidious, usually with a sensation of dullness or fullness in the ear, and an initial fluctuation in hearing of 10 to 30 dB, usually in the low tones. The hearing improves somewhat between attacks, but it continues to deteriorate as time goes on. There may be increased sensitivity to sound, or music may sound distorted. Tinnitus, varying from a whistle to a roar, develops, followed by a turning or whirling vertigo that may lead to nausea, vomiting, and even prostration. Any head movement aggravates the condition, with the vertigo lasting several hours. Some patients can have fleeting attacks lasting several minutes, and still others have attacks lasting a week or longer and may take months to regain normal equilibrium.

Besides the fluctuating hearing, spontaneous nystagmus, usually rotary and often direction-changing, and a direction-fixed, positional nystagmus are the most common findings. The caloric reaction is usually abnormal. Aside from the hearing loss, Meniere's patients frequently have recruitment and diplacusis, low threshold discomfort, and low discrimination scores. Tone decay and a Type II Bekesy are present. A fairly reliable diagnostic test is the glycerin test, where a patient ingests 1.5 gm/kg body weight of glycerol mixed with equal parts of normal saline and a few drops of lemon juice. Audiograms are taken immediately and at one, two, and three hours after ingestion. A positive test is said to be an improvement in hearing of 15 dB in any one frequency from 250 to 4000 Hz or 12 percent improvement in the discriminating score.

There is no effective, long-term treatment for Meniere's disease. For many years, some physicians have controlled their patients with a neutral-ash, salt-free diet, supplemented with diuretics. Shea

(1975) recommends a regimen of bed rest, Valium, low salt, diuretics, and no smoking, plus inhalation of five percent carbon dioxide and 95 percent oxygen for 30 minutes q.i.d. and 2.75 mg of histamine diphosphate in 250 cc of lactated Ringer's solution I.V. b.i.d. Other drugs, given individually, that are reported to be effective for an acute attack are 1/150 grain Atropine I.V., Valium 10 mg I.V., and Innovar, which must be administered in the hospital or by an anesthesiologist. Vasodilators, such as nicotinic acid, beta-pyridylcirbinol, Roniacol, or Arlidin, are usually ineffective in Meniere's, as are the antivertiginous drugs. There have been several surgical treatments for Meniere's with some success in a certain percentage of patients. These range from the endolymphatic shunt to destructive labyrinthotomy in the most severe, uncontrolled cases.

Generic Name	Trade Name	Hours of effect	Adult Dose (mg)	Availability
Cyclizine	Marezine	4	50	Tablets: 50 mg Parenteral inj.: 50 mg/ml Suppos., Peds: 50 & 100 mg
Dextroamphetamine	Dexedrine	8	5-10	Tablets: 5 mg Spanules: 5, 10, 15 mg Elixir: 5 mg/tsp
Dimenhydrinate	Benadryl	6	50	Capsules: 25 & 50 mg Elixir: 12.5 mg/5 ml Parenteral: 10 & 50 mg/ml
Diphenidol	Vontrol	4	25 50	Tablets: 25 mg Ampules: 2 ml (20 mg/ml) Suppositories: 25 & 50 mg
Meclizine	Antivert	12	50	Tablets: 12.5 & 25 mg Chewable, 25 mg
	Bonine	12	50	Tablets, chewable: 25 mg
Promethazine	Phenergan	12	50	Tablets: 12.5, 25 & 50 mg Syrup: 6.25 & 25 mg/5 ml Suppositories: 25 & 50 mg
Scopolomine	Donphen	4	0.2-1	1 or 2 tabs, 3-4 times/24 hrs 6µg/tablet with phenobarbital, 15 mg and atropine 0,02 mg

Antivertiginous drugs				

Acoustic Neuroma

An acoustic neuroma is a fairly rare, extremely slow growing neoplasm that originates on the vestibular portion of the eighth cranial nerve in the internal auditory canal. It constitutes about eight to ten percent of all brain tumors and is most common in the fourth and fifth decade of life. Early diagnosis, which offers the best chance for a surgical cure and the least morbidity and mortality, is often based on a strong suspicion. Symptoms, often difficult to pinpoint but most often present, are steady, unilateral tinnitus, hearing loss, and a feeling of unsteadiness. Some patients have vague complaints of headache, local retroaural discomfort, and facial paresthesia or pain. A significant finding is speech discrimination much more severe than indicated by a pure-tone hearing test.

Diagnostic evaluation should include a complete audiological examination of pure tone and speech, stapedial reflex, acoustic reflex decay, and a Brainstem Evoked Response, Auditory (BERA). Stenver's and Town's X-rays are valuable for an initial screen, but computerized axial tomography or a posterior fossa myelogram are more often necessary. Typically, there is a sensorineural-type hearing with poor speech discrimination that is inconsistent with the pure-tone test, absence of recruitment or low small increment sensitivity index (SISI) scores, pronounced tone decay, a type III or IV Bekesy tracing, reduced caloric response, widening of the internal auditory canal, decreased corneal sensitivity on the involved side, and decreased or absent stapedial reflex.

Suspected cases, which are not diagnostic, should be kept under the watchful eye of an otolaryngologist or neurologist and not dismissed or forgotten after the initial workup.

Benign Paroxysmal Positional Vertigo

Benign paroxysmal positional vertigo must be differentiated from Meniere's and eighth nerve tumors. In general, onset of nystagmus and vertigo occur when the head moves to a certain position. There usually is a latent period of several seconds, and the nystagmus fatigues with repeated testing. Most cases have normal calorics and audiological examinations. Symptoms abate in about eight weeks, but they may recur or even last for years. There is no treatment except avoidance of the position that creates the nystagmus and vertigo, as well as reassurance to the patient. Pilots should be grounded until all symptoms have disappeared, and each case must be considered on an individual basis.

THE DIZZY PATIENT

Vertigo: An hallucination of movement

- True vertigo
 - A sensation of motion or turning
 - Patient turning in relation to surroundings or surroundings turning around patient
 - Usual origin is vestibular system

- Dizziness
 - Less severe and less distinct
 - Sensations of giddiness, faintness, confusion, blankness, unsteadiness, or lightheadedness
 - A floating sensation
- Causes of dizziness and/or vertigo
 - Otologic
 - Impacted cerumen
 - > Trauma to inner ear of VIIIth cranial nerve (*commotio labyrinthi*)
 - Acute otitis media
 - Labyrinthitis
 - ➤ Labyrinthine fistula
 - Bilateral non-functioning equilibrial labyrinths
 - Motion sickness
 - Meniere's disease
 - Lermoyez syndrome
 - Vestibular neuronitis
 - Vasculitis involving internal auditory artery or vestibular emissary veins
 - Tumors of middle and inner ear
 - Ototoxic drugs
 - Focal infection from tonsils, adenoids, periapical tooth abscess, and chronically infected sinuses
 - Sinusitis, acute or subacute
 - Neurologic
 - > Degenerating and demyelinating diseases, especially multiple sclerosis
 - Posterior fossa lesions
 - □ Fractures, cystic arachnoiditis, syringobulbia, platybasia, and Arnold-Chiari malformations
 - □ Neoplasms and subdural hematomata
 - > Supratentorial lesions with brainstem displacement
 - Migraine-like syndromes
 - Convulsive disorders (vestibular epilepsy, vertiginous epilepsy)
 - > Temporal lobe lesions with irritation of cortical vestibular areas
 - Toxic-infectious conditions
 - □ Aseptic meningoencephalitis
 - \Box Brain abscess
 - □ Common viral diseases such as mumps, measles, whooping cough
 - Vascular; including thrombosis, atherosclerosis, embolic occlusion, and hemorrhage, especially in brainstem vessels
 - Systemic
 - Allergic reactions involving inner ear
 - > Dizziness of the aged due to indeterminate vascular cause or end-organ degeneration
 - Cardiac diseases

- Hypertension (paroxysmal)
- Hypotension (syncope)
- Hyperactive carotid reflex
- Blood dyscrasias (anemia, leukemia, reticulosis, polycythemia, purpura)
- Cogan's syndrome (nonsyphilitic interstitial keratitis with vestibulo-bulbochochlear symptoms; panarteritis nodosa)
- Episodes of hypoglycemia
- Hypocorticoadrenalism
- Cervical myalgia
- Ophthalmologic
 - Non-concomitant strabismus
 - Refractive errors
 - Opticokinetic vertigo
- Psychogenic
 - Tension/anxiety states
 - Conversion reaction
 - Neuroses (agoraphobia, claustrophobia)
 - Hyperventilation syndrome

RHINOLOGY

Nasal and Sinus Physiology

The primary functions of the nose are filtration, warming, and humidification of air; it also subserves the sense of smell, and it is the origin and recipient of numerous reflex areas. The sinuses have no primary function.

Air filtration is accomplished by the vibrissae in the anterior nares and by mucus. Most of the mucous glands are in the nasal mucosa. The mucous blanket is moved by cilia toward the nasopharynx at the rate of five mm per minute. Although amazingly resistent to heat, cold, fumes, dust, and chemicals, the cilia are most vulnerable to drying from inspired dry air, such as central heating or 100 percent oxygen.

Air flow during inspiration is directed over the turbinates to the roof of the nasal cavity and then into the nasopharynx. The air is warmed by heat transfer from the mucous membranes. During expiration, the air makes a loop before exiting the nose anteriorly, allowing for retention of the moisture in the air. The air flow volume is regulated by the changing size of the turbinates.

Bacterial Diseases of the Nose

<u>Vestibulitis</u>. An inflammation of the hair follicles in the nasal vestibule may cause chronic crusting and tenderness of the nasal tip or ala; it is often recurrent. Treatment consists of gentle cleaning of the nasal vestibule and the application of topical antibiotic ointment, usually containing Neomycin, two or three times daily. Ophthalmic ointments work well, but treatment must be

continued for two to three weeks after symptoms disappear to prevent recurrences.

<u>Furunculosis</u>. Furunculosis of the vestibule is also common and usually associated with digital trauma and nose blowing. A crack in the skin allows the entrance of strep or staph organisms. Most infections localize, but occasionally they may become a spreading cellulitis. Squeezing or incising the area is dangerous, as it may cause spread to the cavernous sinus. Pain and systemic symptoms may be marked. Treatment consists of a "hands off" policy, adequate doses of appropriate antibiotics, hot, moist packs, and good analgesics.

<u>Rhinitis</u>. Rhinitis can develop as a complication of an upper respiratory infection if symptoms last longer than seven to ten days. Thick yellow or greenish nasal drainage, fever, throat and ear pain, and productive cough suggest complications. The most common causes are excessive nose blowing (forces bacteria into the sinuses and Eustachian tube traumatizes the sinus orifices) and severe coughing (strips the cilia from the bronchial lining)

Treatment should place emphasis on maintaining good nasal and sinus drainage, good tissue hydration, and rest; antibiotics are used for bacterial infections or complications. The penicillins, erythromycin, or the tetracyclines, in order of preference, handle most complications, but cultures should be taken to help in resistant cases.

In general, pilots or flight personnel should not fly with a cold. Even a slight amount of nasal congestion and tissue edema may be enough to interfere with pressure equalization of the sinuses and ears, leading to aerotitis, aerosinusitis, or barometric vertigo. The examiner should strongly advise against self-medication and frequently reiterate the many predictable, immeasurable factors, such as level of awareness and performance, that may be affected by disease or medication. The examiner must make individual judgements, depending on the aircraft, aircrewman's job, type of flight, and medication, when deciding to ground flight personnel. Before personnel are allowed to return to flight status, a careful examination of the ears, nose, and throat should be made. Symptoms are often gone several days before the tissues return to normal and before essential functions return sufficiently to handle the many different and rapid environmental changes associated with flying.

Diseases of the Nose and Sinuses

<u>Allergic Rhinitis</u>. Allergic rhinitis, a very unpredictable and difficult problem, may be acute or chronic, seasonal or perennial. Common symptoms are nasal obstruction, clear rhinorrhea, sneezing, itching of the eyes, soft palate, and nose, and occasional associated headache, mostly frontal. Some cases of allergic rhinitis are similar to a cold, but they usually last only one or two days or over 10 days and are more frequent than viral upper respiratory infections.

Seasonal allergies are often caused by pollens from grasses, trees, or flowers and last two or three weeks. If a specific allergen is found, desensitization is often effective. After an allergy shot, a pilot is grounded for at least six hours. Perennial rhinitis can be quite variable with no pattern, or it may be nearly constant. Allergies may be caused by house dust, molds, dog dander, wool,

feathers, tobacco pollutants, or food. Avoidance, if possible, is the best method of control; however, desensitization may be effective for dusts and molds.

Examination of the nasal mucosa often reveals edema and pallor of the turbinates, especially the inferior turbinates and the anterior tips of the middle turbinates. The turbinates may be so engorged they appear purple. The posterior turbinate tips may protrude into the nasopharynx or become irregular and look like mulberries. Red or inflamed mucosa has also been noted, especially if the allergen is a pollutant.

A basic allergy workup should include the following:

- History present, childhood, family.
- Nasal smear for eosinophiles.
- Sinus X-rays.
- Complete blood count.
- Thyroid function test.
- Total protein, IgE, and gamma globulin blood level.

Basic treatment measures are as follows:

- Take antihistamines, with or without decongestants. Alternating the antihistamine every two weeks is often effective. Consider topical nasal steroids or *Nasalcrom* spray.
- Cover pillows and mattress with plastic.
- Cover overstuffed furniture.
- Eliminate wool from bedding.
- Remove domestic animals from the house.
- Air-condition the house.
- Avoid milk and egg products; other foods can be eliminated, one at a time, a week apart.
- Use nonallergenic cosmetics.

Severe allergy attacks may require a short course of systemic steroids for control. Milder cases that create obstruction of the nasal airway and sinus orifices can often be helped by topical steroids in an aerosol form, such as Decadron or Beclomethasone.

<u>Non-allergic Rhinitis</u>. Non-allergic rhinitis, often included under the term of vasomotor rhinitis, has as the most common symptoms chronic, intermittent, often alternating nasal stuffiness or obstruction, and postnasal drip. In the course of treatment, it is important to rule out allergies, to explain the physiology of the nose to the patient, and to prevent the overuse of nose drops or inhalers that may cause a rhinitis medicamentosa. Once rhinitis medicamentosa develops, it can only be cured by complete abstinence from nose drops. In about three weeks, the normal reflex activity should return. Septal deviations should be corrected if they are a factor in obstructions. Humidification of the house or bedroom, or the use of Proetz solution or ointments to prevent drying of the mucosa is often helpful. Thyroid function may be a factor in some cases; for borderline hypothyroid states, thyroid extract or Cytomel has been effective. Certain emotional states cause nasal symptoms, and they often respond when this problem has been explored or

treated. Rhinitis of pregnancy usually responds to no treatment except delivery. Certain antihypertensive and birth control pills may cause nasal congestion; decrease or change in the drugs often improves or cures the problem.

Polyps and Polypoid Degeneration. When the nasal mucosa, and in some cases the sinus mucosa, reacts to allergies or inflammation, edema develops due to increased capillary permeability and transudation of fluid into the cell and extracellular spaces.

<u>Polyps and Polypoid Degeneration</u>. When the nasal mucosa, and in some cases the sinus mucosa, reacts to allergies or inflammation, edema develops due to increased capillary permeability and transudation of fluid into the cell and extracellular spaces.

Air conditioners may contain much dust and mold, causing more trouble for a person with allergies to these substances. Electrostatic filters may do a better job, but may produce ozone which is toxic. If the first outlet is eight to ten feet from the unit, it is usually safe. Humidification is good for the dry nasal mucosa but it also increases the growth of molds in the house.

The mucosa appears "waterlogged" or "intumescent." Over a period of time, with the help of gravity, this tissue may elongate to form nasal polyps, especially in the region of the middle meatus and maxillary sinus ostia. In some cases, the anterior tip of the middle turbinate may just remain edematous, and this condition is called polypoid degeneration, rather than a polyp. The tissue may lose some of its cilia and is replaced with goblet cells.

Polyps and polypoid degeneration may obstruct the sinus ostia leading to acute and chronic sinus disease or sinus blocks and, therefore, should be removed when obstructive. Small, or single, nonobstructive polyps need not be removed unless they enlarge. Occasionally, polyps are found within the maxillary sinus; these polyps eventually move out of the sinus ostium and into the nasopharynx, where they expand in size. These polyps are called anterochoanal or choanal polyps, and their removal requires a Caldwell-Luc antrostomy to remove the base and prevent recurrence. Polyps in the maxillary sinuses are disqualifying for aviation candidates, as is nasal polyposis. A possible exception can be made for a single, small polyp on one side in an asymptomatic, non-allergic candidate. Recurrence of polyps after removal is common; this is especially true when the disease remains in the ethmoid sinuses. In some cases, the use of short courses of broad spectrum and topical steroids, such as aerosol Decadron or Beclomethasone, may reduce the size of the polyps. A common dose schedule is two sprays in each nostril, twice daily for one week, then one spray in each nostril twice daily for four days, finishing with one spray daily in each nostril for the remainder of the week or longer, if desired. The use of topical steroids may be irritating to the mucosa, and use beyond one month is not recommended.

Epistaxis

The majority of nosebleeds are caused by trauma and occur from the vascular plexus on the

anterior septum, known as Kiesselbach's plexus or Little's area. Common causes are air drying, violent sneezing or blowing the nose, and picking the nose. Severe bleeding, especially high anterior and posterior bleeding, occurs from the ethmoid artery, a branch of the internal carotid, and the sphenopalatine artery, a branch of the external carotid artery.

In general, treatment of simple anterior bleeding should first be direct pressure, for at least five or ten minutes, against the anterior septum. Pledgets of cotton moistened in a vasoconstrictor, such as one percent Neo-Synephrine, one percent epinephrine, or one to four percent cocaine, along with pressure, are even more effective; large clots should be gently suctioned away. If bleeding is controlled, the bleeding site may be cauterized with 25 to 50 percent trichloroacetic acid, five percent chromic acid, or silver nitrate in a 50 percent solution or on a stick applicator. These solutions should be applied with a small, moist applicator under direct vision. Anterior bleeding sites not controlled by direct pressure or chemical cautery should be infiltrated with Xylocaine and epinephrine, using both the tissue wheal and the epinephrine effect for control. The site may then be cauterized by chemical or electrocautery; deep burns or cauterization of adjacent structures, such as the ala or vestibule, must be avoided. If the coagulated fluid and blood stick to the tip of the cautery and are pulled off with the coagulum, the bleeding may restart. In those cases where bleeding cannot be controlled, one might attempt cautery with a suction tip electrode; if this fails, the nose can be packed with Vaseline and antibiotic ointment impregnated in half-inch selvage gauze. It is best to pack both sides to prevent loss of the pack by shifting of the septal cartilage from a one-sided pressure. The pack should be left in place for at least 24 hours, but usually never more than 72 hours. All raw or cauterized surfaces should be lightly covered with an antibiotic ointment, and a small piece of compressed Gelfoam over the anterior septum further protects against air trauma. The ointment application should be repeated three or four times a day.

Posterior bleeding, usually in the older age group, is a serious condition, and, if coupled with hypertension, it requires aggressive medical and rhinological management. The patient should be admitted to sick bay, sedated, and kept in a head-elevated position. After vasoconstrictor and topical anesthetic application to both nasal passages, an attempt can be made to control the posterior bleeding by the use of a specifically designed postnasal balloon, or a common, 15 cc-size Foley catheter. The balloon is checked before insertion, then it is passed along the floor of the nose, and when it is in the lower nasopharynx, the balloon is filled with about 5 cc of water. It is then drawn back up against the posterior choana and further filled to the point of tolerable discomfort to the patient. Anterior packing is inserted bilaterally with fixation of the catheter to the lip or against the packing, but never against the ala or septum to prevent pressure necrosis. The posterior pharynx is checked hourly for bleeding, and the hemoglobin and hematocrit are monitored according to the amount of oozing or bleeding; blood typing and cross matching are advisable. Blood coagulation studies are usually done, but it is unusual to see only nasal bleeding with abnormality of the clotting mechanism. A patient with a posterior nasal pack or balloon is never sent home. He should be closely monitored because of the possibility of a nasovagal reflex action when the nasopharynx is packed, that might lead to apnea or hypoxia. Uncontrolled bleeding of the ethmoidal arteries requires ligation in the orbit or, as a last resort, an internal

carotid ligation. Uncontrolled sphenopalatine artery bleeding requires ligation through a transmaxillary sinus approach, or ligation of the external carotid in the neck.

Barotrauma to Sinuses

Aerosinusitis results when equalization of pressure between the sinus cavities and the atmosphere is prevented by obstruction of their orifices. There are numerous causes, but heading the list are the common cold and allergies. Other causes are anatomical defects, infection, and polyps.

As an aviator goes to altitude, the outside pressure decreases, and discomfort may be felt in the obstructed sinus. It is usually not severe, however, and most often air forces its way out past the obstruction. When the aviator descends, the pressure in the obstructed sinus remains less than the surrounding pressure, creating a vacuum effect on the delicate, thin, mucosal lining and resulting in pain that is often severe. Some fluid may be drawn into the cavity, but the more serious complication is pulling away of the mucoperiosteum, with formation of a hematoma. Sinus blocks occur most often in the frontal sinus (70 percent), and the aviator must be grounded until the hematoma resolves, and the ostium is patent. This may require three weeks to three months. For this reason, anyone suspected of a sinus block should have sinus X-rays to determine the extent of injury and then should be followed at approximately three-week intervals, until clear.

Treatment of the acute block

- Stop descent in aircraft or low-pressure chamber, if possible, and return to altitude for pain relief.
- If available, spray the nasal passage with a vasoconstrictor nasal spray (nose drops).
- Do the Valsalva maneuver or use the Politzer method.
- Make a slow descent equalizing pressure with the above maneuvers.
- Place patient on antihistamine-decongestant or decongestant therapy.
- Take screening Water and Caldwell sinus X-rays.
- If an upper respiratory infection is present, treat with antibiotics.
- Control severe pain with Codeine or Percodan.
- If a frontal sinus hematoma is present, ground the aviator for at least three weeks. With no apparent X-ray pathology, the aviator should be grounded for at least 72 hours, or until any nasal symptoms have been cleared. Recurrent trauma may result in mucocele formation, requiring a major surgical procedure and permanent gounding.

Sinusitis

The majority of acute sinusitis cases follows an acute upper respiratory infection, like the common cold, and they are often the result of improper nose blowing. Another cause, which may have a more rapid onset, is swimming or diving; occasionally, an upper molar tooth abscess breaks into the maxillary antrum. The extent and persistence of the infection depends on two major physiological principles, ventilation and drainage; the treatment is directed toward these principles. The most common bacterial causes are the Gram-positive cocci.

Acute suppurative sinusitis usually has symptoms of nasal congestion and pressure or pain over

the involved sinus. Toxicity is usually mild, except in cases of pansinusitis when the frontals or sphenoids are involved. Pus draining from the middle meatus or above the middle turbinate, pain and pressure over a maxillary or frontal sinus, and decreased transillumination may be sufficient to make a diagnosis. The X-ray is indispensable, however, in determining the extent of the disease, fluid levels, and response to medication, all of which may indicate the proper approach to treatment.

Maxillary sinusitis, usually has the least toxicity, but a persistent fluid level or pain after 48 hours of adequate antibiotic therapy suggests the need for irrigation of the antrum, either through the canine fossa or through the thin, bony wall of the inferior meatus. The maxillary sinus mucosa has great reparative power; after removal of the pus by irrigation, it may clear within a few days. If the antral infection is dental in origin, it is useless to attempt a cure without treatment of the offending tooth.

Ethmoid sinusitis is probably the most common infection. Due to the proximity of the ethmoid sinuses to the frontal and maxillary sinuses, ethmoid sinusitis either causes or is associated with the infections in those sinuses also. Ethmoid infections usually cause more inflammation and mucosal swelling. Pain may be near the root of the nose or frontal region. Edema of the lower lid is often present in children. Orbit involvement may result in painful eye movement due to a periostitis about the pulley of the superior oblique muscle or, in the case of rupture into the orbit, proptosis.

Frontal sinusitis usually is associated with toxicity, frontal headache, often in mid-morning to late afternoon, tenderness to percussion over the sinus, or pressure on the floor in the supraorbital region; swelling of the upper eyelid may be highly suggestive. Treatment should be vigorous to prevent osteomyelitis of the skull or fistulas that lead to complications, such as soft tissue or sinus cavity abscesses, meningitis, brain abscess, and even death.

Sphenoid sinusitis is uncommon, but it may result as a direct extension of infection in neighboring sinuses, nasal mucosa, or the nasopharynx. The symptoms are variable, but they may consist of a deep, boring, occipital or parietal headache with inability to concentrate, fever, malaise, and anorexia. Rupture or osteomyelitis from sphenoid infection leads to rapidly fatal meningitis or cavernous sinus thrombosis. Diagnosis can usually only be made by suspicion and X-rays, using proper contrast in the lateral and submental vertex positions; fluid levels will only be seen if the X-rays are taken in the upright position. These patients require high doses of intravenous antibiotics and emergency surgical intervention.

Since the cardinal principle of treatment in sinus infections is ventilation and drainage, the following treatment is suggested:

- The nasal mucosa must be protected from drying. The patient must be kept hydrated, and, in some cases, use of a humidifier or vaporizer may help.
- An oral decongestant may be used alone or with an antihistamine. Antihistamines may make secretions too thick or the mucosa too dry, so it is often helpful to use a mucous-thinning

medication, such as glyceryl-guaiacolate.

- Antibiotics are given orally in adequate doses for at least seven days in most uncomplicated cases, but in pansinusitis or cases of moderate to severe toxicity, and especially in frontal or sphenoid involvement, intravenous antibiotics are necessary. Most organisms are sensitive to penicillin or erythromycin, but it is strongly recommended that a culture be taken from the turbinates and the meatuses. Be sure not to touch the nasal vestibule and hairs, as these areas may have different predominant organisms. The nasopharynx is another area from which to obtain a culture of prevalent sinus drainage.
- Bed rest, hydration, and adequate pain medication are important in patients with toxicity.
- Antral irrigation, either through the natural ostium or the canine fossa or inferior meatus puncture approach, is indicated for persistent pain or fluid levels after 48 hours of antibiotic therapy. Persistent swelling, and pain in the frontal sinus region, in spite of intense therapy, may signal the need for a frontal sinus drainage, usually done by trephine through the sinus floor. A rubber or plastic tube drain is sutured in place to allow irrigation and drainage until the natural ostium drainage is reestablished.
- Daily mucosal shrinkage and gentle nasal suction cleaning may help promote drainage.Local heat is often helpful, not only as comfort to the patient, but to increase the vitality of the mucosa.
- Persistent or subacute ethmoid disease may respond to Grossan nasal irrigation.
- Acute sphenoid infection with toxic signs is an emergency life-threatening situation requiring immediate hospitalization and surgery, so one should always be alert to this disease, by itself or as a complication from the other sinuses.

Neglected sinus infections or subacute disease lead to chronic irreversible changes in the sinus mucosa. With chronic purulent drainage or sinus blockage, one usually as to resort to surgery after conservative treatment fails. For the maxillary sinus, an endoscopic opening of the natural ostia (ostiotomy) is most often used. Removal of the ethmoid cells is more difficult and is done with an intranasal approach when polyps and persistent disease are present. Chronic sphenoid disease is not only rare, but most difficult to diagnose, because X-rays may be inconclusive and symptoms extremely variable. Chronic disease in the frontal sinus, be it osteomyelitis or mucocele formation, dictates a major surgical procedure through either a bicoronal incision flap approach or the osteoplastic eyebrow incision approach, with complete removal of the sinus mucosa and obliteration of the sinus, usually with fat. These surgical procedures and treatment may not result in relief of nasal symptoms or remove the tendency toward recurrent infection. A frontal sinus obliteration is usually disqualifying for most types of aviation.

Antral cysts (frequently seen on the Waters X-ray as a smooth, rounded density in the lower aspect of the maxillary sinus) are benign, filled only with xanthochromic or clear fluid. They usually require no treatment, unless they fill the sinus, obstruct drainage, and become symptomatic

Maxillary Sinus Irrigation-Inferior Meatus Puncture

- Anesthesia.
 - Spray mucosa initially with a vasoconstrictor.

- For local anesthesia use four or five percent topical cocaine and two percent Xylocaine with Epinephrine 1:1000 (dental carpule or equivalent).
- Apply pledgets of cotton moistened with cocaine (never sloppy wet) in the inferior meatus and on the inferior turbinate. After initial application, cocaine on a wire applicator is placed against the lateral wall of the inferior meatus about one inch or 1.5 to 2.5 cm behind the anterior edge of the meatus for five minutes.
- Insert a long (3 1/2 inch) needle into the inferior meatus until it strikes bone in the area of the intended puncture and infiltrate with local anesthetic.
- Equipment.
 - Straight three and a half inch, 18 gauge spinal needle or equivalent trocar with stylet.
 - Sterile saline to which a small amount of Neo-Synephrine may be added.
 - One 30 to 50 cc syringe and one 5 cc syringe.
 - Plastic or rubber extension tubing.
 - Culture tube.
- Technique.
 - With the patient in an upright position and the head against a firm headrest, the puncture needle or trocar is inserted into the inferior meatus about two centimeters posterior to the edge of the inferior meatus and engaged in the thin bone of the lateral wall of this area.
 - The thumb is placed against the stylet and the needle is directed laterally in line with the outer canthus of the eye, using the fingers of the opposite hand to steady the needle. Pressure is slowly, but steadily, increased until the needle is felt to penetrate into the sinus.
 - The needle is pushed into the sinus until it strikes the lateral sinus wall and then withdrawn about one centimeter. If a low-lying cyst is present, the needle is directed as far inferior as possible just after penetration to puncture the cyst.
 - Direct observation of the drainage or aspiration with a small syringe may be diagnostic or produce a pure specimen for culture.
 - The large syringe and extension tubing filled with normal saline are inserted into the needle and aspiration is attempted. Air bubble or exudate indicates the needle is in the proper position. No aspiration may mean the needle is in the mucosa, plugged, or not in the sinus proper.
 - Irrigation is carried out with the patient leaning forward over a large basin with his mouth open, and gentle, but steady, pressure is applied to the syringe.
 - Instant, severe pain suggests the needle is in the mucosa; readjust the needle's position and repeat. Intolerance to irrigation pressure dictates termination of the procedure and possible attempt at natural ostia irrigation. A slow buildup of pressure and occasionally pain is expected with an obstructed ostia, but it is usually tolerable or relieved as the sinus is irrigated.
 - Irrigation should be carried out until the washing is clear or, in the case of a clear irrigation, until at least three full syringes have been used.
 - The final irrigation should be made with the sinus ostia dependent. Insufflation of air into the sinus has been associated with air isolation and should not be performed.

• The needle is withdrawn with a smooth rapid movement and the nasal passage immediately inspected for retained pus or thick mucus. This material is aspired, being sure to include aspiration of the posterior floor and middle meatus.

Nasal Fractures

Nasal fractures are common injuries which can usually be handled in the clinic or sick bay by the examiner. There are basically three types: (1) a simple fracture of the nasal bones, most often just the tip, (2) lateral displacement of the nasal bones to one side, often as a green stick fracture on one side and impaction on the other, and (3) marked flattening of the nasal bridge with comminution of the bones or an accordion fracture displacement of the septum. Diagnosis of a displaced fracture can best be made by inspection, palpation, lateral X-ray of the nasal bones and comparison of the patient to his or her ID card or recent photo.

Shortly after injury, when the airway is compromised, or before profuse swelling has occurred, reduction should be accomplished under local or general anesthesia. When the injury is very recent and the patient is still in a shock or "numb" like state, stable lateral displaced fractures can often be reduced without anesthesia by simple, quick, firm, thumb pressure on the convex side of the nose. When soft tissue swelling is marked, distorting the true alignment of the nasal bones, one may elect to wait four or five days for the swelling to recede before reduction. A compound fracture should be reduced within a few hours and then have a plastic-type laceration closure since reduction maneuvers usually tear out delicate sutures. Antibiotic coverage is recommended to prevent complications.

- Anesthesia Technique for Reduction of Nasal Fractures.
 - Shrink the nasal mucosa with one percent Neo-Synephrine.
 - Use fresh cocaine from one to five percent, most common is four percent or plain two or four percent Xylocaine.
 - Moisten long, thin, cotton pledgets with cocaine, squeeze out the excess, and insert them into the superior and middle aspects of the nasal passages. They should touch the septum and turbinate mucosa beneath the nasal bones where reduction instruments are inserted. The sphenopalatine and long nasopalatine nerves may be blocked by applying anesthetic on a long applicator to the area of the sphenoid rostrum. The area can be reached by inserting the applicator back past the posterior tip of the middle turbinate. The ethmoid nerves may be blocked by applying anesthetic on an applicator to the middle turbinate tip.
 - Local anesthesia, using two percent Xylocaine with epinephrine (dental syringe carpule), is
 obtained by inserting a long dental needle into the nasal vestibule just above the upper
 lateral cartilage at the limen nasi. Slide the needle is slid beneath the skin in the
 subcutaneous tissue but external to the nasal bones to the desired locations.
 - Infiltration sites are the glabella for the superior trochlear nerve, the inner canthus region for the ethmoid and the infratrochlear nerves. A more lateral reinsertion of the needle to the infraorbital notch will block the infraorbital nerve. Following an initial wheal, the needle is slowly withdrawn while injecting a tract of anesthesia. Repeat on each side.

- Local anesthesia of the superior septum is obtained by injection of the septum just beneath the tip of the nasal bones and obtaining a "run" of the anesthesia beneath the mucoperichondrium. For the entire septum several anterior injections are required with the bevel toward the cartilage.
- Equipment for Nasal Fracture Reduction.
 - Elevator Most often used is the Sayer elevator. Others are flat scalpel handles or the Salinger reduction instrument.
 - Bayonet forceps.
 - Gelfoam pledgets.
 - Half-inch vaseline-impregnated gauze, minimum of two tubes.
 - Antibiotic ointment.
 - Rubber finger cot.
 - Quarter-inch regular or plastic tape.
 - Malleable metal nasal splint.
- Nasal Fracture Reduction Techniques.
 - Septal Fractures Only
 - Grasp the septum between two fingers, pull forward up, and side to side, using a thumb or finger of the opposite hand to unbuckle a concavity.
 - The nose is then packed (beware of toxic shock syndrome) on both sides to maintain a good alignment alone or against a stint.
 - Stints of dental wax or Teflon sheets can be used and held in position with through and through septal sutures.
 - Depressed Nasal Tip
 - Place a finger cot over the elevator and insert it in either nostril to just beneath the fracture. Using the fingers of the opposite hand to move and guide the fragment, lift the fragment with the elevator and slowly withdraw.
 - Place compressed Gelfoam beneath the fracture site on both sides. Selvage gauze anterior packing, external taping, and a metal splint offer the best results.
- Lateral Displaced or Comminuted Fracture.
 - Measure the distance externally from the nostril to the glabella on the elevator. Insert the elevator the measured length into the most open side of the nose.
 - With a steady lift of the elevator, move the fracture further to the deviated side. Then
 move the nasal bones across the midline an equal distance to the opposite side; return the
 fragments to the midline. Some bleeding is expected, but in the majority of cases,
 Gelfoam is all that is required for control, and it helps prevent adhesions that may occur
 superiorly. External taping and metal protection aid in maintaining alignment.
- Taping and Splinting Techniques.
 - Apply benzoin solution to the forehead, nose, and cheek areas.
 - If the nose is packed, the initial tape should run from one side to the other parallel with the dorsum across the packed nares. Do not pull tight, and allow for tissue swelling by

cutting or pinching the tape at the tip.

- Fixation of the nose is provided by an initial tape across the dorsum from cheek to cheek, then a crisscross taping from the forehead to the cheek on both sides. This may be weaved in with the dorsal tapes. If the nose is packed, be sure all of the tip is covered with tape to prevent swelling.
- A malleable aluminum splint is placed over the nasal taping and held in place by similar crisscross taping.
- For drainage, a folded two by two-inch pad taped across the lower lip allows the patient to breathe and eat without interference.
- Errors in Nasal Fracture Treatment: According to DeWeese and Saunders, the following common errors are associated with treatment of nasal fractures:
 - The doctor attempts to set a nose that was also fractured years previously, but the patient becomes aware of the old deformity only when the new trauma calls attention to it. (Check the patients ID card).
 - X-rays reveal no fracture when one is present, altering the doctor's clinical judgement. In reality, X-rays are of little practical value in management of nasal fractures. However, they are of great value in management of fractures of the zygoma and infraorbital sinus.
 - The doctor regards easy to reduce fractures too seriously, and severe fractures too lightly, leading to unnecessary anesthesia or poor reduction because of limited anesthesia.
 - The doctor waits longer than five or six days to reduce; thereafter, reduction may be difficult.
 - In addition, attempting to reduce a fracture in a grossly swollen nose may lead to insufficient reduction or poor alignment.

Maxillary Fractures

Maxillary fractures should always be suspected in direct trauma to the face when there is malocclusion or restriction of mandibular movement, flattening of the side of the face, a "black eye" which included ecchymosis and subconjunctival hemorrhage, anesthesia over the face supplied by the infraorbital nerve, or the more serious sign of diplopia. X-rays are extremely important in diagnosis, as well as postreduction evaluation. A full series should include the Waters, Caldwell, lateral, and submental vertex.

Zygomatic arch fractures can be elevated under local anesthesia through a temporalis fascia approach or a buccal mucosa approach. All other fractures require more extensive open reduction, often with wire fixation or prosthetic support and protection requiring the assistance and training of an oral surgeon, otolaryngologist, or in the case of a true "blow out" fracture, an ophthalmologist.

THE MOUTH AND PHARYNX

This part of the ENT examination should be thorough and easy on the patient, but it is often most difficult and stressing, both for the physician and the patient. The following points and techniques are recommended. The patient should always be as comfortable as possible and in an upright

position. Explanation and instructions to the patient before the procedure is started are absolutely necessary. The physician should reassure the patient and refrain from using uncomfortable words, such as gag, and from putting the mirror down the throat, or pulling the tongue. The patient should be encouraged to relax his tongue during the oral and pharyngeal examination and to breath through his mouth. If there is concern about disease transmission, the physician can wear a mask.

The correct technique for using a tongue depressor is to insert the blade into the mouth without touching the tongue and then to press straight down on the anterior two-thirds of the tongue. Except for hypopharyngoscopy, the patient should not stick out his tongue because this raises and firms up the tongue, preventing good exposure of the tonsils and pharyngeal area. When warming a mirror over a flame, the physician should always test the back side of the mirror for proper warmth against his wrist or face so that the patient will not fear being burned. On introduction of the nasopharyngeal mirror, sizes zero, one, or two, it is helpful to slide the handle along the corner of the mouth and touch the patient's face with the finger to steady the mirror. This also helps distract the patient's thoughts about gagging. The nasopharyngeal mirror may be slipped into the nasopharynx alongside of the uvula and may even touch the tip, but touching the base of the tongue should be avoided. When holding the tongue for the laryngeal examination, the under surface should be wrapped with cotton gauze to protect it from the sharp edges of the teeth. The fingers can be steadied against the lower teeth and upper lip. If the patient sits up straight and brings his head and chin forward, the larynx is more fully visible. Fingers against the patient's face steady the mirror (three, four, five,) as it is introduced into the mouth, without touching the tongue, toward the uvula and soft palate. Often, the vocal cords can be seen without touching the soft palate, but if necessary, contact should be positive and firm, with little or no movement after contact is made.

If a patient is unable to breathe through his mouth when requested, it may be necessary to have him hold his nose closed. These examinations should last only 10 to 15 seconds because of salivation, anxiety, and discomfort. For patients with hyperactive gag reflexes, mild mucosal anesthetics such as Chloraseptic or Benadryl Elixir can be tried first. Stronger anesthetic agents such as Cetacaine, *Hurricaine*, four percent Xylocaine, or five percent cocaine may be necessary, but some are toxic and rapidly absorbed from the oral mucosa, so care must be exercised in the amount and rapidity with which they are applied. For extremely difficult cases, I.V. diazepam (Valium) of 2.5 to 5 mg over a 90 second period of administration, gives an excellent effect for 15 to 20 minutes. Since apnea, caine reactions, or cardiac arrest are always a definite danger with these drugs, resuscitative equipment should be at hand.

Common Oral Diseases

<u>Thrush</u>. Since the advent of antibiotics, thrush, formerly seen chiefly in children, is now being seen in adults when the normal flora is altered. The usually white mucosal lesions are scraped for microscopic diagnosis of the characteristic yeast cells.

Treatment is Mycostatin suspension, 1 cc (10,000 units) swished in the mouth for a full 5 minutes

daily, for 7 or more days. Stop all other antibiotics. A one percent Gentian Violet solution may also be applied b.i.d. to the lesions, but the messy staining properties of this solution have decreased its use.

<u>Herpetic Lesions</u>. Fever blisters and cold sores caused by the herpes simplex virus begin with a vesicle that, unlike the aphthous ulcer, usually involves the gingiva; the vesicle breaks and forms an irregular ulcer. These lesions are most common after a febrile illness, trauma, actinic exposure, or stress.

Treatment is symptomatic with nonirritating mouthwashes and oral irrigations; mild anesthetic ointments and solutions may be helpful. Benzoin and Orabase may protect or dry the vesicles and ulcers. Early application of Stoxil ointment or ether has been used, but the success rate is variable, and there is some suspicion of RNA alteration to a carcinogenic state. Corticosteroids are contraindicated.

<u>Aphthous Stomatitis</u>. Recurrent canker sores are found most often as multiple, well-delineated shallow ulcers on the buccal and labial mucosa, tongue, soft palate (including tonsillar pillars), and pharynx; occasionally, there is only a single lesion. These yellow-gray, membrane-covered ulcers heal spontaneously in one to two weeks. There may be severe pain requiring topical and oral anesthetics for eating. Longer relief can often be obtained by cleaning the lesion off and applying Kenalog in Orabase, while it is still dry. This may be repeated three or four times daily. Some physicians advocate the use of Tetracycline suspension, 250 mg/per tsp., held in the mouth for at least two minutes and then swallowed, four times daily. Aphthous stomatitis should be differentiated from the herpetic gingival stomatitis by lack of bleb or vesicle formation or associated systemic disease, before cortisone treatment is started.

<u>Pharyngitis Sicca or Chronic Dry Throat</u>. The pharynx is usually dry, smooth, and shiny, with some yellow-green crusts. Treatment usually provides only temporary relief, but 50 percent potassium iodide, 10 drops in milk t.i.d., SSKI, 6 drops in half a glass of water t.i.d., or painting the throat with Mandel's solution may be helpful. Occasionally, three to five grams of ammonium chloride t.i.d. also helps, but fluid and electrolyte balance must be watched.

<u>Pharyngeal Infection</u>. It is sometimes difficult to determine if a pathogen is responsible for an infection in the nose or throat, or which pathogen is responsible. Many organisms such as *Streptococcus viridans*, Neisseria, anaerobic streptococci, *Staphylococcus albus*, or yeast are always present and termed normal flora. Although a culture, which takes 24 to 48 hours to grow, may be helpful in treatment and should be obtained, it should be remembered that staphylococci can be obtained from 60 to 80 percent of the population, and beta-streptococci are often isolated from patients with a viral infection. Furthermore, pathogens may become established in the host and remain for months without causing disease. This is referred to as a carrier state. In treatment, the physician must make an intelligent "guess" about the etiology of the infection, using the most important clinical picture, a smear from the infected area for pus cells and predominant organisms, and then correlate this information with the bacteriological findings. Always keep in

mind the possibility of gonococcal or chlamydial infections.

<u>Acute Tonsillitis</u>. Acute bacterial tonsillitis or pharyngitis is most often caused by beta-hemolytic streptococci, Group A. It usually has a rather abrupt onset, with fever of $101^{\circ}F$ + and chills. The mucosa is grossly inflamed, with white or yellow exudate on the lymphoid follicles. If the exudative tonsillar tissue becomes necrotic, it is termed necrotizing tonsillitis.

The antibiotic treatment of choice is penicillin, most often given orally, 250 mg, q.i.d. An initial I.M. dose of 1.2 to 1.5 million units of procaine penicillin may be given to adults, to obtain a more rapid blood level. Therapy should be continued ten days. Alternatives are clindamycin for failures or cephalexin, cefadroxil, and erythromycin.

With toxic symptoms, the patient should be on bed rest and forced fluids. Hot throat irrigations hourly or at least four times daily, coupled with analgesics, such as Empirin Compound #3, Ascodeen - 30, or Tylenol #3, are necessary for both comfort and a more rapid recovery.

Infection of the lingual tonsils at the base of the tongue, often not properly diagnosed without the aid of the laryngeal mirror, may cause considerable dysphagia. Besides the normal treatment for tonsillitis, the physician may need to add, by direct application, gargle or spray, soothing substances such as Chloraseptic solution, Mandel's paint, or a topical anesthetic, such as Dyclone, 0.5 percent or 1 percent or *Hurricaine* 20%.

<u>Nasopharyngitis</u>. Occasionally, a physician may see a patient who appears toxic and febrile, with pressure or pain in the ears, a severe headache, or retrobulbar pain. Usually, the oropharynx is somewhat inflamed, and there is occasionally neck stiffness or edema of the uvula. Examination of the nasopharynx with a mirror will make the diagnosis of nasopharyngitis with the discovery of exudate in upper reaches of the nasopharynx. Treatment with I.M./I.V. antibiotics initially, plus supportive treatment, is advocated.

<u>Viral Pharyngitis</u>. Sore throat, lymphoid injection without exudate, general or posterior cervical adenopathy, and malaise are the usual symptoms of a viral pharyngitis; a normal white blood count with increase in the lymphocytes is often the blood picture. Tonsillitis that has a membranous exudate, marked lymphoid hypertrophy, often a negative throat culture, and does not respond to penicillin, should be evaluated for infectious mononucleosis. Diagnostic tests include white blood count, differential, and a mononucleosis spot test.

In areas of frequent cases of gonorrhea, resistant or unusual cases of pharyngitis should be cultured, specifically for Neisseria gonococci.

<u>Thornwaldt's Disease</u>. Physicians should be aware of a nasopharyngeal bursa or pouch that sometimes forms in the midline of the adenoid tissue and, when it becomes infected, produces occipital headaches and an irritating, purulent postnasal discharge; it can also be present after adenoidectomy. Diagnosis is made by ruling out sinus disease and visualization of the draining

bursa with the nasopharyngeal mirror, or, more clearly, the nasopharyngoscope, or Yankhauer scope. Treatment requires either electrocoagulation or surgical removal of the cyst or pouch.

<u>Peritonsillar Abscess</u>. Known also as quinsy (sore throat), this abscess results when tonsillar infection spreads or breaks through posteriorly into the potential areolar space between the tonsil and the superior constrictor of the pharynx. Formation of the abscess results in displacement of the tonsil toward the midline, anteriorly and downward, with displacement of the uvula to the opposite side; it also causes fullness or cellulitis of the soft palate. There is a variable degree of trismus, pain referred to the neck or ear, variable adenopathy, and often the classic "potato" speech that results from the spasm or cellulitis involving the pharyngeal muscularity.

Treatment consists of high doses of systemic antibiotics for 10 to 14 days and incision and drainage (I & D) of the abscess immediately, if fluctuant, or as soon as fluctuance develops. If spontaneous drainage is noted from a necrotic site, gentle suction and blunt, careful widening of the opening assists in providing adequate drainage. Hot saline throat irrigations every few hours and adequate analgesics are necessary for the first few days. The I & D site should be reopened in 24 hours. Occasionally, a second or third reopening may be necessary if considerable pus continues to accumulate. Emergency tonsillectomies are performed by some physicians as a treatment for the abscess, but this should only be done by experienced hands because of the danger of bleeding or sepsis. It is advised that an elective tonsillectomy be considered in about six weeks, after the acute infection has subsided.

Incision and Drainage of Peritonsillar Abscesses.

- Equipment
 - Long handle, curved Kelly forceps with smooth blunt tips.
 - Suction machine with tonsil and/or nasal suction tips.
 - Long knife handle with #15 blade.
 - Large metal basin.
 - Culture tube.
- Anesthesia
 - Premedication with I.M. Demerol & Vistaril or I.V. Valium is recommended.
 - Topical Cetacaine, one percent cocaine, or four percent Xylocaine is helpful.
 - Local infiltration at the incision site with dental two percent Xylocaine and epinephrine, 1:100,000, is often used, but some physicians are against infiltration into cellulitic tissue.
- Procedure
 - The best site for incision is at the point of intersection of a vertical line from the last molar tooth on the involved side and a horizontal line from the lower edge of the soft palate on the opposite, uninvolved side. The incision should be made from lateral toward the midline, about 1.5 to 2 cm long, just through the mucosa.
 - The curved Kelly is introduced into the incision and spread, opening over the top, but never through, the tonsil. The tip is at first directed straight in, then slightly downward

and somewhat lateral.

- When the abscess cavity is opened, there is a sudden, often forceful, release of thick pus, for which both the physician and the patient should be prepared.
- With the patient leaning slightly forward, immediate, rapid, but gentle, suction is applied to the draining pus and incision site.
- The incision must be opened sufficiently. Bleeding is usually slight and clots form in five or ten minutes. A sterile nasal suction tip may be inserted into the incision site for better evacuation of the pus, but strong suction should not be applied, as this may create severe bleeding.
- Hot saline irrigations, three or four times per day, are recommended. One or two liters of saline are used for each irrigation. The solution can be used directly from a commercial container or mixed by the pharmacy. Murphy drip bottles, irrigation cans, or the solution bottles connected to I.V. tubing are placed eight to ten feet high. A small oral irrigation tip or glass or plastic eyedropper can be used to deliver a forceful, narrow stream. The solution should be as hot as tolerable without burning the oral tissue.
- An acceptable alternative to incision and drainage (I & D) is aspiration of the abscess by an 18 gauge needle attached to a 10 cc syringe.

Laryngology

There are four major functions of the larynx: airway, sphincter, protection, and phonation. As an airway, the vocal cords are constantly regulating the required air flow needed by the lungs and maintaining a proper resistance or back pressure.

When we strain or lift with our arms and chest muscles, the vocal cords close, trapping air in the chest cavity, fixing the chest wall, and allowing for maximum efficiency in the lift. This function comes into play for the cough and for the effort in defecation.

The larynx is said to be the "Watch Dog of the Lungs." Through the sensory branches of the superior laryngeal nerve, foreign bodies, abnormal mucus, pus, or fluids are prevented from entering the trachea by the rapid closure of the cords, followed by coughing or by clearing of the throat.

The vocal cords produce sound which is modified by the lips, teeth, tongue, and palate to form speech or singing tones.

Diseases of the Larynx

<u>Hoarseness</u>. Hoarseness is defined as roughness or discordance in the quality of the voice. It is apparent that it is a symptom and not a disease process in itself. Often, the first and only danger signal of serious disease, local or systemic, involves this area. Unfortunately, the degree of hoarseness presents no clue to the type of illness or its prognosis. A thorough examination is necessary in all cases to ascertain the exact cause and to prescribe the proper treatment. Generally, there are intrinsic lesions such as inflammation, benign or malignant neoplasms, allergies, and trauma. There may be disturbances in innervation, either central or peripheral.

Hoarseness may be a manifestation of system disease, such as TB, syphilis, muscular dystrophy, arthritis, or endocrine disorders, and finally, psychosomatic involvements must be considered when all else has been eliminated. No more than two weeks should pass before a examination is made of the vocal cords.

<u>Acute Laryngitis</u>. The chief symptoms of acute laryngitis are pain and hoarseness, and they may be secondary to an upper respiratory infection, most often viral, or hemophilus in children or streptococcus in adults. On laryngeal examination, the vocal cords and adjacent subglottic and arytenoid area are inflamed, and there may be various degrees of swelling.

In most cases, treatment of the primary illness with appropriate antibiotics, cough suppressants, steam inhalation, elimination of irritants, especially tobacco and alcohol, and voice rest, is sufficient. Lozenges such as Cepacol, anesthetics, troche with benzocaine, or throat sprays such as Laralgan, may be soothing. Laryngitis from vocal trauma and noxious gases is best treated with voice rest and humidification. Thermal burns or caustic injury may require, in addition to the other treatments, system steroids and tracheotomy.

<u>Chronic Laryngitis</u>. Chronic laryngitis includes many different conditions and implies longstanding inflammatory changes in the mucosa, as might be expected from recurrent acute episodes, chronic improper use of voice (singers, speakers, and hucksters), and exposures to adverse conditions, such as dust and fumes. Smoking and alcohol have been shown to contribute, as well as TB, syphilis, and chronic sinusitis or bronchitis. Chronic laryngitis may take the form of small, bilateral vocal nodules or large polyps at the junction of the anterior and middle third of the vocal cords. Other forms are hypertrophic or hemorrhagic changes on the cord or dry thickening of the interarytenoid area. Vocal activities must be limited and rest encouraged. Surgical measures will occasionally become necessary. Encourage the patient to keep well hydrated. Expectorant drugs, such as potassium iodide or ammonium chloride are advocated, as is the use of humidification. The Medical Officer should follow the patient closely by regular mirror laryngoscopy to assure early treatment should surgical pathology develop.

Salivary Glands

Calculi occur more frequently in the submaxillary duct and gland. A common sign may be painful swelling of the glands when the patient eats. Localization of the calculus can often be made by bimanual palpation of the gland or duct, along the floor of the mouth, and a dental X-ray of the floor of the mouth. If the calculus is in the duct, it can often be milked toward the papilla. Removal is facilitated, after local infiltration with Xylocaine, by cutting off the papilla to enlarge the orifice and then slitting along Wharton's duct. Calculi in the proximal duct or gland may require excision of the gland if the obstruction cannot be relieved. Infection behind the obstruction usually responds to drainage but may require antibiotics as in sialadenitis.

<u>Acute Sialadenitis</u>. The parotid is more often affected than the submaxillary gland, usually resulting from retrograde extension of the mouth infection and dehydration, especially in the elderly. The duct should be milked and a culture taken; however, the most common organism is

often a penicillin-resistant, coagulase-positive staphylococcus. The empirical choice of antibiotics would be adequate doses of anti-staphylococcal penicillin, cephalothin, or clindamycin. Correction of the dehydration is essential. In severe resistant cases, I & D of the gland may be lifesaving.

<u>Chronic Sialectasis</u>. Recurrent infections or, occasionally, congenital anomalies lead to stasis of secretions and chronic dilation of the ducts and alveoli, which can be diagnosed by sialography. Long-term therapy with tetracycline is often helpful, but unresolved symptoms may necessitate excision of the affected gland.

<u>Auriculotemporal Syndrome</u>. After parotidectomy or injury to the gland, the patient may experience gustatory sweating, called Frey's Syndrome. Temporary relief might be obtained by Scopolamine, but more lasting results may require a tympanic neurectomy of Jacobson's nerve and the chorda tympani nerve. Treatment with an antiperspirant may suffice.

AUDIOLOGY

Conductive Hearing Loss

- External Canal
 - Obstruction wax etc. producing total occlusion
 - Collapse false audiogram
- Tympanic Membrane
 - Stiffness thickened, scarred, or adhesions
 - Flaccid atrophic or atelectatic
 - Perforations usually large
- Ossicles
 - Stiffness adhesions, otosclerosis, or anatomic
 - Disarticulation without perforation usually 50-55 dB
 - Otosclerosis: estimated at 10% of population with only 12% clinical.
 - ➢ Bilateral in 50%
 - Onset in 4-5 decade with females predominating 2-4:1
 - Genetic: Dominant
 - Hearing loss: Conductive, early low frequency tilt, later flat. May have neurosensory loss with cochlear involvement.
 - Surgery: 97% successful A/B closure. Not recommended for pilots. Grounded up to two years. Occasional dead ear.

Perceptive or Neurosensory Loss (Cochlear)

- Acoustic Trauma
 - Impulse 105 dB or greater
 - usually immediate and often persistent tinnitus

- ➢ occasional vertigo
- Accumulative (Gunners or Aviators ears)
 - develop a notch in 2-4 KHz range
 - ➢ tinnitus is variable
 - usually associated with sound pressure of 85 dB for 8 hrs. For every 5 dB above, decreases exposure time 50%
 - > usually requires several years to develop and is irreversible
- Inner Ear Trauma
 - Temporal Bone Fractures
 - Longitudinal C.N. 8/cochlear usually spared. Occasional C.N. 7 injury; most often Hemotympanum, TM and canal lacerations and ossicular disarticulation
 - Transverse C.N. 7 and/or C.N. 8 injury. Middle ear may be clear
- Inner Ear Fistula (PLF)
 - Mechanism an explosive over pressure through ruptured TM of Eustachian Tube, explosive pressure through cochlear aqueduct or internal auditory canal
 - Symptoms positional to persistent vertigo; fluctuating to profound Neurosensory hearing loss
 - Diagnosis positive fistula test with tympanogram or pneumo-otoscopy, exploratory tympanotomy.
 - Treatment keep head elevated and affected ear up, sedation and transport for diagnosis/surgery ASAP; avoid straining or nose blowing
- Meniere's disease
 - Symptoms Triad of fluctuating sensorineural hearing loss, fluctuating tinnitus, and attacks of true vertigo
 - duration of vertigo may range from minutes to several hours, but rarely several days
 - periods of vertigo usually interspersed with periods of total remission that may be as short as hours or as long as several years
 - ➤ the variable hearing loss does not exhibit periods of remission
 - ▶ 85% of the cases are unilateral with hypofunction of the labyrinths
 - approximately 60% of cases have a final remission with variable residual tinnitus, hearing loss and vestibular dysfunction
 - Diagnosis hearing loss is initially low tone sensorineural type with good discrimination
 - \succ with time the levels will decline across all frequencies.
 - discrimination will also deteriorate
 - Glycerol test
 - Treatment the unpredictable length of attacks and remissions, and the inability to measure the degree of hearing loss and vestibular dysfunction make Meniere's an enigma for treatment
 - Medical therapy includes diuretics, salt restriction, vestibular suppressants, vasodilators and occasionally vitamins.

- Surgical procedures are either destructive, conservative or preservative and have a wide variation of success
- Disposition Personnel with Meniere's disease are not fit for military duty and require a medical board.

Sudden Deafness

- Localized lesions in Temporal Bone
 - Acoustic Neuroma
 - CPA tumor
 - PLF
 - Aneurysm of A.I.C. artery
- Systemic diseases of the Temporal Bone
 - Viral infection
 - Accelerated coagulation
 - Hyperviscosity of Polycythemia and Macroglobulinemia
 - Arteriosclerosis secondary to
 - > Aging
 - ➢ Hypertension
 - Diabetes
 - > Hyperlipidemia
 - Collagen disease
 - Multiple Sclerosis
 - Syphilis
- Symptoms rare
- usually unilateral and often associated with transient vertigo and persistent tinnitus
- Treatment must be instituted within 48 hours to be most effective. The most effective treatment regimens are as follows:
 - Method A:
 - Mandatory bed rest for 3 to 10 days (component of all regimens)
 - Atropine 0.75 mg I.M. (only within 4 hrs of onset)
 - Procaine 0.2% in 250 ml of D5/W I.V. bid x 3 days
 - Benadryl 50 mg P.O. qid x 3 weeks
 - Arlidin 6 mg P.O. qid x 3 weeks
 - Vitamin C 1 gm I.V. with Procaine
 - Method B:
 - Histamine vasodilation using 2.75 mg of histamine in 200 ml of D5/W I.V. at a rate to cause flushing, but not cause headache or significant drop in blood pressure.
 - Dextran 10% 500 cc q 12 hours x 3 days
 - ➢ Nicotinic Acid 50 300 mg AC and HS x 3 weeks
 - Method C:
 - Systemic steroids, such as prednisone 60 mg daily x 7 days, tapered to zero over seven days.

- Method D:
 - Oragrafin 1 ml I.V. on first day, then 2 ml I.V. daily until recovery (37%). Not useful if vertigo is present.
- Evaluation Hearing thresholds and speech testing are done at regular intervals, initially q.o.d., then at longer intervals in the ensuing weeks and months.
 - Most patients have some residual hearing loss.
 - Aviators must be considered on an individual basis for return to duty. This is mostly determined by the amount of hearing deficit, the completion of an extensive workup for tumor and neurological or other disease, and discontinuance of maintenance medication, such as histamine and nicotinic acid.

BASIC HEARING TESTS

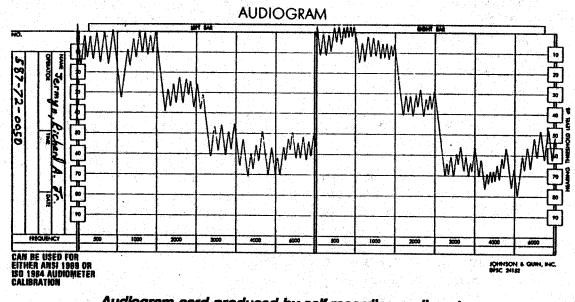
Pure-Tone Audiometry

The most common and also the most elementary test is done with pure tones. The patient is asked to respond whenever he hears a tone, regardless of the loudness of the signal. The lowest amplitude at which the patient responds at a particular frequency is called the hearing level (HL). HL's are determined at octave frequencies from 250 to 8000 Hz and at the half-octave frequencies of 3000 and 6000 Hz. Each continuous tone is presented for a period not exceeding one second. Intermittent (pulse) tones are also frequently used, especially in patients where tinnitus is present. There will be several tone presentations at a particular frequency before the HL is recorded on the audiogram. Pure-tone HLs are determined for both air conduction (earphones) and bone conduction (vibrator) stimuli.

Masking noise is used when one ear needs to be isolated from the other in order to get a correct threshold measurement for the test ear. Masking noise is generated within the audiometer and can consist of a broad or narrow-frequency band. Narrow band noise is most efficient for masking pure-tones. In a situation where one ear of the patient is "dead," incorrect information would be obtained for the nonfunctional ear if masking were not used for the good ear. By air conduction measurement, the nonfunctional ear would yield HL's around 50 to 60 dB. This is due to a phenomenon called "crossover." Even though the signal is presented at the nonfunctional ear, it is heard by the good ear primarily by direct energy transmission through the head from the vibrating earphone cushion. The head creates about a 50 to 60 dB "barrier" between ears. If proper masking noise is applied to the good ear in the case mentioned, then a correct determination of a profound hearing loss would be made.

An electromechanical vibrator is placed on the mastoid process for bone conduction (BC) testing. The threshold determination procedure is identical to that of air conduction (AC) testing. Since it requires more energy to drive a mechanical vibrator than an earphone, the maximum hearing loss that can be measured for BC is less than for AC, (e.g., 70 dB for BC and 110 for AC). Care should be taken to place the vibrator on the mastoid without contacting the pinna. This is to ensure that responses at low frequencies are auditory and not tactile in nature. Masking of the contralateral ear is done more frequently in BC than in AC. This is because interaural attenuation,

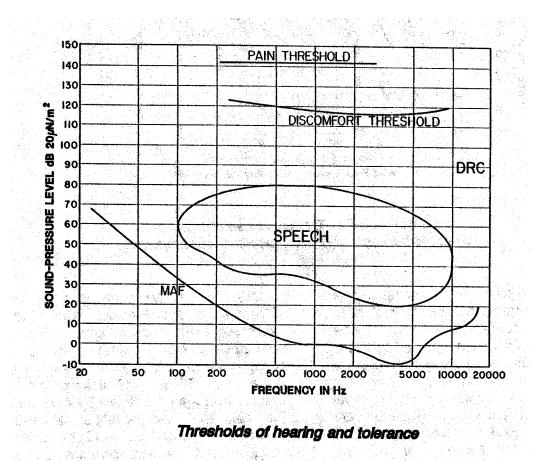
while about 50 to 60 dB for AC, is practically nonexistent (0 to 5 dB) for BC. In the previous example of the "dead" ear, a BC measurement without proper contralateral masking would have shown normal BC hearing in the nonfunctional ear due to the low (0 to 5 dB) crossover levels.



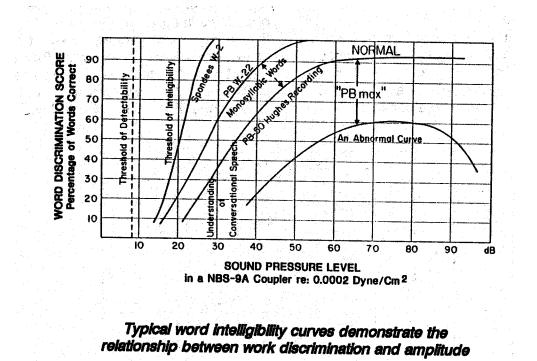
Audiogram card produced by self-recording audiometers

Speech Audiometry

Another aspect of the basic hearing test battery is speech audiometry. The purpose here is to discover two things. First, it is necessary to determine the amplitude at which the patient can repeat back approximately 50 percent of the two-syllable words presented to him. This measure is referred to as the speech reception threshold (SRT). There are six word lists, each list being a different scrambling of the same 36 words. The most widely used form is CID Auditory Test W-1. Secondly, the percentage of 50 single-syllable words the patient can correctly repeat back is determined. This test is called the "PB score" or "PB Max" and is a measure of speech intelligibility. The term "PB" stands for "phonetically-balanced." When these word lists (24 lists with 50 words each, and 200 words in the corpus) were developed in the late 1940's, it was believed that the phonemes in each 50-word list had to have the same proportionate frequency of occurrence as that in everyday English, in order for the test to be valid. This was later shown to be unnecessary, but the terminology "PB" still remains today.



A graph demonstrating the relationship between word discrimination and amplitude (SPL) is shown in following figure. The various curves shown are called articulation curves or performance intensity (PI) functions. The PB words, the most widely used form being CID Auditory Test W-22, are presented at a level of 40dB above the SRT in routine use. Since this represents a supra-threshold presentation, masking noise is almost always used in the contralateral ear. It is at this amplitude or sensation level (SL) that most patients would achieve maximum performance. However, there are instances where this is not the case. So, ideally, a performance intensity function would be generated by presenting the monosyllabic word lists at a variety of sensation levels. A phenomenon called roll-over is demonstrated by the abnormal curve. Roll-over is characterized by a worsening of discrimination as loudness is increased. This finding is characteristic of retrocochlear disorders (e.g., acoustic neuroma) and to a lesser extent Meniere's syndrome.



Often speech discrimination testing is done in a noise background. A variety of word lists and test formats are used for this purpose. The basic concept behind this is to provide a more realistic environment in the measurement of speech discrimination. It is a rare occasion, particularly in the naval environment, when the listening environment is absolutely quiet. There are several considerations for discrimination in noise testing. Probably the most important, single consideration is the signal to noise ratio (S/N) employed in the test. S/N ratio is expressed in dB, and the figure represents the number of dB the signal (speech in this case) is above or below the level of the noise. If the S/N is -4 dB, this would mean that the average speech level is 4 dB below the noise level. Typical S/N levels used in discrimination testing that would be reflective of typical naval aviation noise environments would range from 0 to +4 dB S/N.

Threshold Tone Decay Tests

Another component of the basic test battery is the threshold tone-decay test (TDT). This is a pure-tone, supra-threshold test. It is usually done at 4,000 Hz first, and, if positive, the test frequency is dropped by octaves until 500 Hz is tested. The tone is presented at 5 dB SL for one minute. If the patient can hear the tone for the entire period at the same level, the test is negative. If the level of the tone has to be raised by 20 or more dB above the starting level, the test is positive. The TDT is a measure of auditory adaptation and is considered a screening test for retrocochlear pathology. If the test is positive, other, more detailed, tests would be done in order to help establish the reason for the abnormal adaptation and the site of the lesion. The Suprathreshold Adaptation Test (STAT) is also frequently used. The test is positive if a high level (e.g., 100 dB) tone cannot be heard over a 60 second period.

Advanced Tests in Differential Diagnosis

<u>Short Increment Sensitivity Index (SISI)</u>. This is a pure-tone test presented at 25 dB SL that measures amplitude discrimination ability. The result is expressed in terms of percent correct identification out of twenty, one-dB increments, added to a reference pure-tone level. A high percent correct response is indicative of a cochlear pathology.

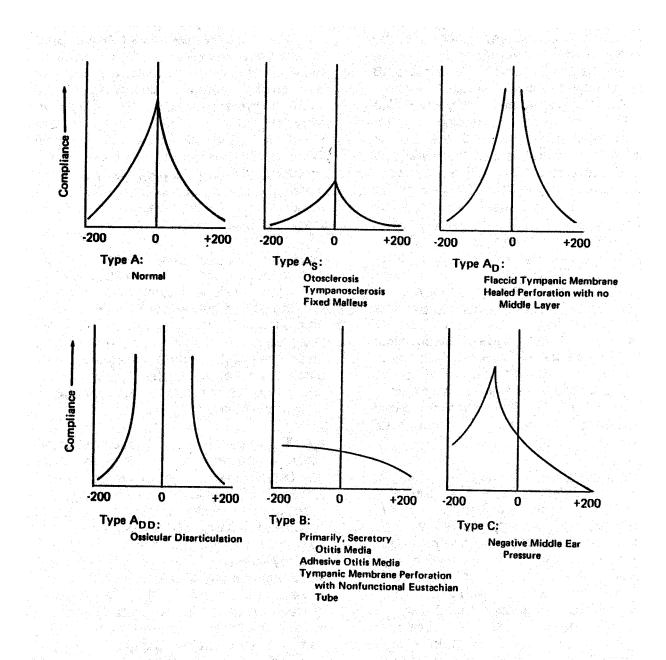
<u>Bekesy Audiometry</u>. Bekesy audiometry is an advanced site-of-lesion test and is a special form of the more routine, self-recording audiometry procedure. The patient is asked to track his pure-tone threshold by means of a response button, first for a pulsing tone and then for a continuous tone. Either a discrete frequency or continuous frequency tracing can be generated. The audiograms are traced on the same graph. The audiogram is then categorized according to the relationship between the pulsed and continuous tracings. There are five recognized types of Bekesy audiograms. Each type is supportive of a particular pathology and will be discussed in the section on interpretation of findings.

<u>Auditory Brainstem Response (ABR) Audiometry</u>. ABR audiometry and electro-cochleography (EChocG) are two relatively new objective hearing tests. Both are electrophysiological measures of auditory function. These are noninvasive techniques that involve computer averaging of the auditory system's electrical response to clicks or tone pips. Either of these tests could be used in cases of functional (nonorganic) hearing loss or psychogenic problems. The ABR is particularly useful in cases of suspected brainstem lesions. The examiner should have little contact with these test types.

Lengthened Off Time (LOT) Test. The LOT test is also used where malingering is suspected. This is basically a Bekesy test with the period between pulses lengthened and unequal to the duration of the pulse itself, (e.g., 800 msec off and 200 msec on). This temporal pattern magnifies the difference between the pulsed and continuous tracings, making the identification of possible malingering easier.

<u>Sensitized Speech Tests</u>. These are tests in which the auditory stimulus is speech that has been altered, either in the amplitude, temporal, or frequency domain. They are used when a central auditory disorder is suspected. "Central" is defined as a site of lesion somewhere in the brainstem or cortical auditory areas. Pure-tone tests are not sufficiently complex in nature to identify these lesions. In general, as the site of lesion proceeds centrally in the auditory system, the tests to identify it need to become more and more complex in structure. The examiner's contact with this type of test information would be quite rare in the active duty population. It would more likely occur in the retired or dependent groups.

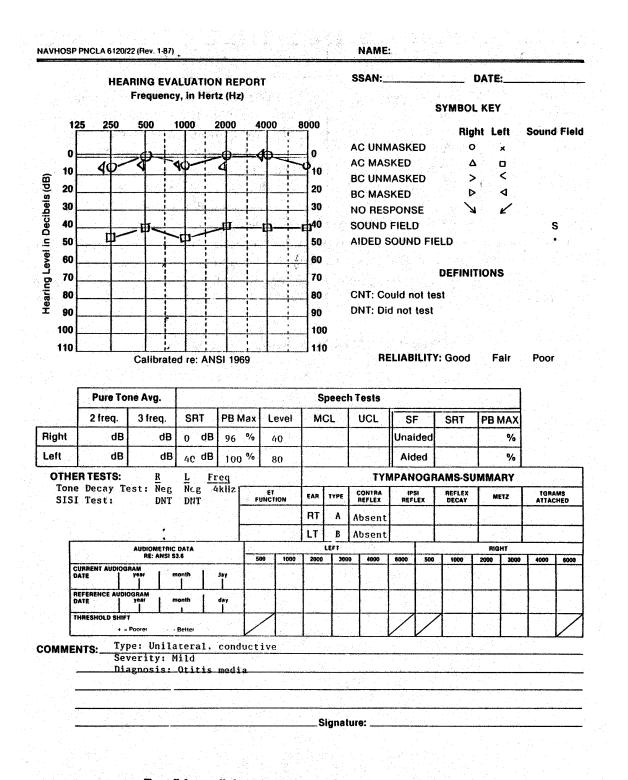
<u>Impedance Audiometry</u>. This is actually a subbattery of tests. The following figure depicts typical impedance audiometry configurations.



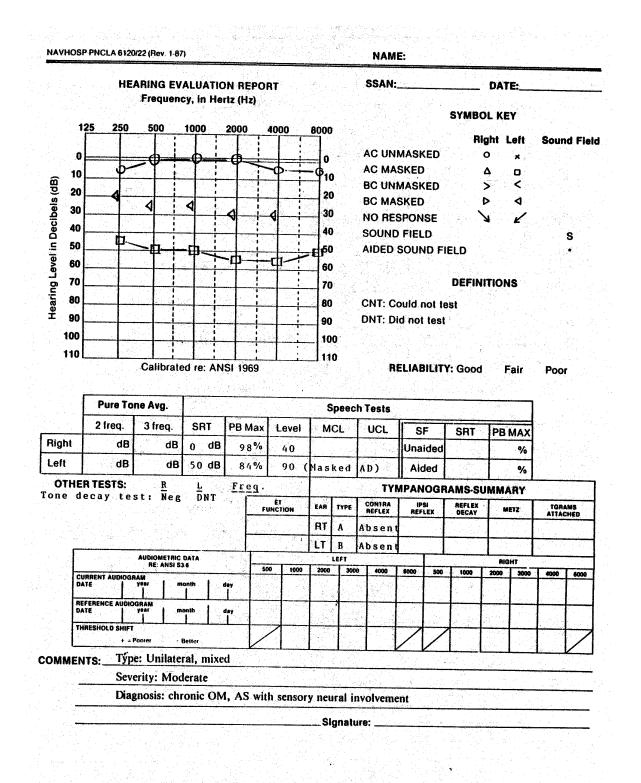
Tympanometric configurations

Average HL *	Classification	Ability to Hear Speech
0-25 dB	WNL	No ifficulty, even with faint speech.
26-40	Mild	Difficulty with faint speech only.
41-55	Moderate	Frequent difficulty with normal speech.
56-70	Moderately Severe	Frequent difficulty with loud speech.
71-90	Severe	Only hear shouted or amplified speech.
91+	Profound	Usually unable to understand even amplified speech

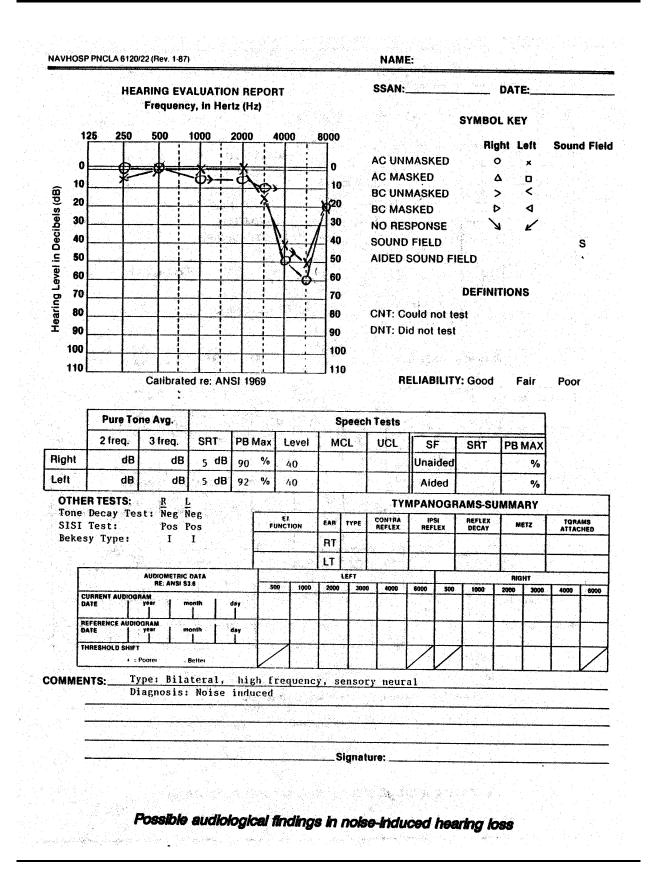
Classification of hearing impairment by severity

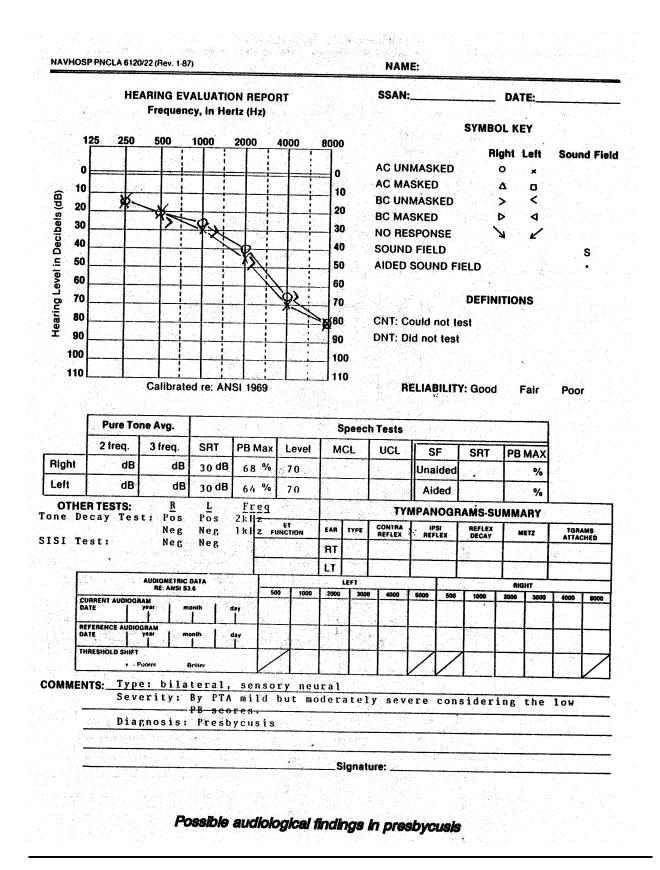


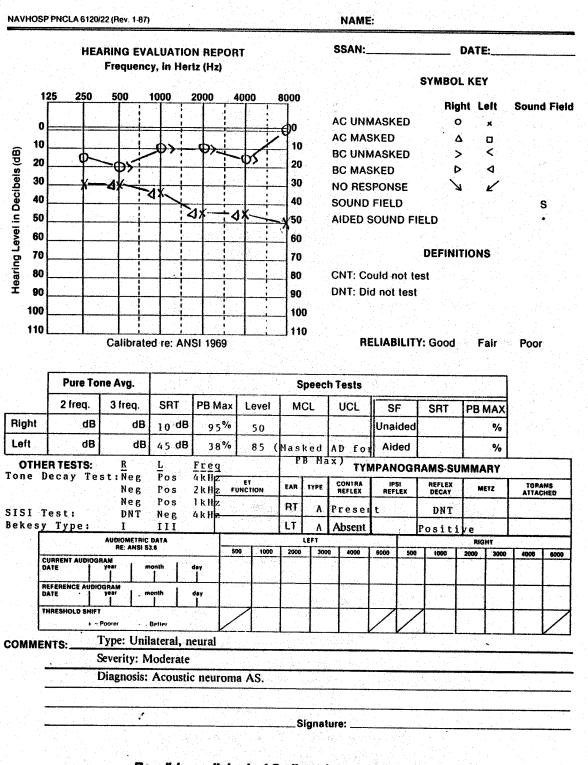
Possible audiological findings in unilateral otitis media



Possible audiological findings in chronic otitis media







Possible audiological findings in acoustic neuroma

OTORHINOLARYNGOLOGIC DRUGS OF CHOICE				
CLINICAL DIAGNOSIS	INITIAL CHOICE	ALTERNATIVES		
OTOLOGY				
Acute otitis media	Amoxicillin or erythromycin plus sulfonamide	Amox/clav TMP-SMX Cefaclor, cefuroxime, cefixime		
Bullous myringitis	Erythromycin plus sulfonamide Anesthetic otic drops	Amoxicillin Amox/clav Cefaclor, cefuroxime, cefixime TMP-SMX		
Otitis media with effusion	Same as otitis media			
Otitis media prophylaxis	Sulfamethoxazole	Ampicillin		
Acute mastoiditis (from acute otitis media)	Cefuroxime plus metronidazole	Chloramphenicol plus nafcillin, cefazolin, or vancomycin		
Acute mastoiditis (from chronic otitis media)	Ticar/clav plus chloramphenicol or cefuroxime	Ceftazidime plus chloramphenicol		
Chronic suppurative otitis media (perforation or cholesteatoma)	Neomycin plus polymyxin otic drops with or without clindamycin orally	Chloramphenicol otic drops		
Acute diffuse otitis externa (swimmer's ear)	Neomycin plus polymyxin otic drops	Boric or acetic acid and alcohol drops		
<i>Malignant</i> otitis externa	Tobramycin plus ticarcillin	Gentamycin or amikacin plus ticarcillin Ticar/clav Ceftazidime Ciprofloxacin		
Furuncle of ear canal	Antistaphylococcal penicillin	Cephalosporin Clindamycin		
Otomycosis	Boric acid or acetic acid and alcohol drops	M-cresyl acetate otic drops		
Neurosyphilis	Penicillin 2-4M IU x 10d IV			

OTORHINOLARYNGOLOGIC DRUGS OF CHOICE				
CLINICAL DIAGNOSIS	INITIAL CHOICE	ALTERNATIVES		
	or IM with probenecid			
RHINOLOGY				
Sinusitis, acute	Amoxicillin/ampicillin Erythromycin plus sulfonamide	Amox/clav Cefaclor, cefuroxime, cefixime TMP-SMX		
Sinusitis, chronic	Antistaphylococcal or augmented penicillin	Cephalosporin Clindamycin Doxycycline		
Orbital cellulitis	Ceftriaxone Cefotaxime Ceftazidime	Antistaphylococcal penicillin or vancomycin plus aztreonam Amoxicillin plus chloramphenicol		
Rhinitis/nasopharyngitis (carriers) Staphylococcus aureus Haemophilus influenza	Rifampin plus cephalosporin or antistaphylococcal penicillin Rifampin			
HEAD AND NECK INFECTIONS				
Tonsillo-adenitis	Penicillin Clindamycin for failures	Cephalexin, cefadroxil, erythromycin		
Pharyngitis (Streptococcal)	Penicillin	Erythromycin, cephalexin, cefadroxil		
Pharyngitis (gonococcal or chlamydial)	Ceftriaxone plus doxycycline	Penicillin plus doxycycline		
Stomatitis, aphthous	Tetracycline syrup (as mouthwash)	Antihistamine or antacid powder/suspension		
Laryngitis Viral Bacterial	Symptomatic care Erythromycin plus sulfonamide	Cefaclor, cefuroxime, cefixime Amox/clav		

OTORHINOLARYNGOLOGY

Epiglottitis	Cefuroxime, ceftazidime, cefotaxime, aztreonam	Chloramphenicol plus ampicillin
Subglottic bacterial croup	Amox/clav Antistaphylococcal penicillin plus sulfonamide	Cefazolin plus sulfonamide
Tracheobronchitis Viral Bacterial	Amantadine (for type A influenza) Erythromycin	Tetracycline
Parotitis, suppurative	Antistaphylococcal penicillin	Cephalosporin, clindamycin, vancomycin
Peritonsillar abscess	Clindamycin	Antistaphylococcal penicillin or amox/clav Cephalosporin
Deep neck abscess with or without necrotizing fasciitis	Cefoxitin plus gentamycin	Clindamycin plus gentamycin or ceftazidime
Cellulitis, cervical-facial	Ampicillin plus nafcillin plus chloramphenicol or amox/clav	2 ND or 3 RD generation cephalosporin plus chloramphenicol Imipenem
Meningitis or intracranial abscess		
From acute otitis or sinusitis	Ampicillin plus chloramphenicol	Ceftriaxone, ceftazidime, or cefotaxime with or without chloramphenicol
From chronic sinusitis From chronic mastoiditis	Nafcillin plus chloramphenicol Nafcillin plus ceftazidime plus piperacillin or tobramycin (intrathecal)	Same as above Vancomycin plus ceftazidime plus piperacillin or tobramycin (intrathecal)
Osteomyelitis	Clindamycin	Antistaphylococcal penicillin Cephalosporin
Prophylaxis in surgery (begin 2 hours before incision) Routine/healthy	First generation	Antistaphylococcal penicillin

Extensive surgery/prolonged hospitalization Endocarditis	cephalosporin Clindamycin plus gentamycin or tobramycin Penicillin	Ceftazidime Vancomycin or erythromycin		
Amox/clav = augmented amoxicillin/K+ clavulanateTicar/clav = ticarcillin/K+ clavulanateTMP-SMX = trimethoprim-sulfamethoxazole				

EAR DRESSING PROCEDURE

The purpose of the dressing is to splint, protect, and absorb drainage from the ear with maximum comfort to the patient. It must also resist movement or displacement.

The bandage material most commonly used is a supportive pad behind the ear, a fluff dressing of loose gauze or mechanic's waste, and a support covering of the dressing with material like *Kling* or *Kerlix* elastic or stretch gauze.

First, two or three 4 x 4-inch pads are folded together in half, and then a "C" shape is cut out of the center that will fit behind and around the ear. Next, the entire ear is covered with two or three inches of fluff dressing or mechanic's waste. If splinting of the pinna contours is important, as in lacerations, this can be accomplished by careful insertion of ointment-impregnated cotton in the grooves of the scaphoid fossa, canal meatus, and concha.

The external bandage of an elastic or stretch gauze usually begins on the forehead and is always wrapped from the front to the back of the ear. To keep the dressing out of the patient's eyes, two pieces of umbilical tape or thin gauze are laid vertically on both sides of the forehead. The stretch gauze is wrapped first across the center of the fluff, across the lower occiput, above the opposite ear, and then repeated below and above the first wrap, resulting in a football helmet like appearance. The forehead tapes are now tied and tape strips applied to hold the gauze in position, using intermittent applications about six inches in length.