Chapter 6

THE OTOLARYNGOLOGIC ASPECTS OF AEROSPACE MEDICINE
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INTRODUCTION

Abnormalities in the ear, nose and throat (ENT) area can conveniently be divided into two areas, those conditions which are directly related to aerospace operations and those relatively common entities which affect aircrew along with the general population. Human beings are terrestrial creatures with sense organs that are well suited for surface living; however, when they venture above the surface of the earth (or below that portion which is aqueous) certain difficulties may be encountered. Those conditions most directly related to flying are: a.) the effects of changing barometric pressure on body cavities, and b.) the functional inadequacy of the spatial orientation system under flight conditions.

Pathologic changes and definite symptoms can result from exposure to decreasing barometric pressure (ascent) and increasing ambient pressure (descent). With the exception of hypoxia, the term dysbarism has historically denoted all disturbances within the body resulting from a change in barometric pressure, including both increases and decreases. However, current authorities prefer to classify the effects of exposure to changing barometric pressure as either mechanical effects (expansion or contraction of gases trapped in body cavities) or decompression sickness (gases evolved from body fluids). Mechanical effects include the creation of relatively positive pressure in the middle ear space causing alternobaric vertigo and the development of relatively negative pressure in the middle ear space or a paranasal sinus causing barotitis media or barosinusitis. Mechanical effects of trapped gases can produce symptoms in other areas of the body as well (e.g., abdominal pain). Decompression sickness is discussed in Chapter 3. The conditions resulting from poor adaption of the human spatial orientation system to nonphysiological flight accelerations include spatial disorientation and airsickness. These topics are discussed in detail in Chapter 4.

ENT diseases may similarly affect fliers and non-fliers, either acutely or chronically. However, for the flier the aeromedical implication is usually clear: the crew member must be grounded until the pathologic process has cleared and normal function returns. Additionally, acute, limited conditions (e.g., a viral upper respiratory infection) may make the crew member more vulnerable to other physiological complications in the flight environment including ear/sinus block or vertigo. A primary consideration in determining the aeromedical disposition of an ENT abnormality, is whether or not the condition is capable of producing sudden incapacity jeopardizing flight safety. For this reason, vertiginous entities are particularly important.
This chapter will discuss those otolaryngological abnormalities etiologically related to aerospace operations (barotitis, barosinusitis and alternobaric vertigo), commonly encountered vertiginous entities, and general ENT problems frequently seen by the flight surgeon.

MECHANICAL EFFECTS OF BAROMETRIC PRESSURE CHANGES

General

The mechanism producing mechanical barotrauma is fairly simple. Boyle's law states that at any given temperature a given mass of gas varies in volume inversely with pressure. The pressure of the atmosphere decreases with altitude on a curvilinear gradient. A container that is closed but capable of expanding will vary in size as it ascends or descends depending on the ambient pressure. When a rigid-walled cavity that cannot change in size is moved through an environment of decreasing pressure (ascent), a pressure differential will be created, with the pressure inside the cavity being greater relative to its environment. When this same cavity is moved through an environment of increasing pressure (descent), the pressure inside the cavity will be less or relatively negative. If such a cavity contains an opening through which these pressure changes can be equalized, there will be no pressure differential. However, if this opening becomes occluded there will be an increase in pressure within the cavity on ascent and a decrease in pressure (relative negative pressure) on descent. The rate of ascent or descent will determine the rate and extent to which a pressure differential develops. Physiologically, symptoms may be produced by the relatively positive pressure which can develop in gas-containing cavities during ascent or by the relatively negative pressure which can develop during descent. Experience has shown that more serious symptoms are much more likely to develop when a relatively negative pressure exists in a gas-containing cavity or space (e.g., ear block or sinus block on descent).

Altitude chamber reactions can be utilized to suggest the potential incidence of barotrauma in flight operations. Table 6-1 lists the distribution of altitude chamber reactions in the USAF for calendar years 1979-1982. Barotitis was the most common adverse reaction encountered, comprising almost two-thirds of all the reactions for each of the years. Symptoms from barotrauma result from relatively negative pressure developing during descent. The next most common condition, about one-fourth as frequent as an ear block, was abdominal gas pain (which results from trapped gas expanding during ascent). Barosinusitis was a close third; here symptoms result from relatively negative pressure. All other reactions are much more infrequent. Therefore, roughly 75% of chamber reactions occur during descent.
TABLE 6-1. DISTRIBUTION OF ALTITUDE CHAMBER REACTIONS IN AIRCREW TRAINEES DURING CALENDAR YEARS 1979-1982. (%).

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<tr>
<td>Abdominal gas pain</td>
<td>.776</td>
<td>.754</td>
<td>.697</td>
<td>.694</td>
</tr>
<tr>
<td>Aerosinusitis</td>
<td>.245</td>
<td>.262</td>
<td>.243</td>
<td>.272</td>
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<tr>
<td>Bends</td>
<td>.051</td>
<td>.055</td>
<td>.067</td>
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<tr>
<td>Barodontalgia</td>
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<td>Neurological</td>
<td>.008</td>
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Hg positive pressure will force air out through the eustachian tube and relieve the symptoms, or the symptoms can be readily relieved by swallowing. However, if this positive pressure is not relieved, more severe symptomatology will result, impressively manifested in most individuals as vertigo. The resulting vertigo will usually over-shadow the accompanying tinnitus or pain. This is *alternobaric* or *pressure vertigo* and it is discussed in more detail below.

On descent, however, a totally different situation exists. The eustachian tube acts as a flutter valve and remains closed unless actively opened by muscle action or high positive pressures. If the eustachian tube opens, any existing pressure differential is immediately equalized. If the tube does not open regularly during descent, a pressure differential will develop. If this pressure differential reaches 80-90 mm Hg, the eustachian tube muscles cannot overcome it and re-ascent, or a nonphysiologic maneuver, will be necessary to open the tube.

The most common cause for failure of the middle ear space to ventilate on changing from low to high atmospheric pressure is swelling of the nasopharyngeal end of the eustachian tube, usually due to an acute upper respiratory infection or allergic rhinitis. Even minimal edema in the narrower portions of the eustachian tube wall may produce sufficient luminal constriction to cause blockage during descent.

Certain contributory etiologic considerations to otic barotrauma should be mentioned. Ignorance of the necessity for swallowing at frequent intervals during descent in aircraft is a significant factor. The ability to recognize the early pressure changes during descent and then adjusting the interval between swallowings to meet the demands of the rate of descent comes only with flight experience. The rate of descent is also a significant factor. This has been recognized for many years by commercial airlines. Civilian airlines descend gradually, generally less than 400 ft per minute. Descent rates in military aircraft are usually greater, frequently several thousand feet per minute.

The use of oxygen during flight also increases the likelihood of barotrauma. Oxygen from an aircraft's oxygen system is quite dry and may produce irritation of the upper respiratory mucosa. Also, the absorption of oxygen by the mucous membranes contributes to the relative negative pressure in the various upper respiratory cavities, including the middle ear space. When oxygen absorption is the primary factor in the development of a pressure differential, the term "delayed barotitis media" is sometimes used. Personnel who fly in certain jet aircraft equipped with a system that delivers 100% oxygen from the beginning to the end of the flight are most prone to develop this type of ear block. However, this absorption of oxygen must be combined with the infrequency of swallowing during sleep in order to produce an ear block. Individuals who fly aircraft equipped with this type of oxygen system are aware of this possibility and will keep their ears ventilated following termination of a flight. However, if a flight is completed during late evening hours or at night, and the individual retired a short time later, a significant pressure differential may develop during sleep due to the combined factors of oxygen absorption and infrequent swallowing. Once the pressure differential develops during sleep, the situation is exactly the same as failing to ventilate the eustachian tube during descent from altitude. It should be added that sleeping during descent in an aircraft can lead to an ear block, again due to the infrequency of swallowing while asleep.
The **pathological changes** vary with the magnitude of the pressure differential between the tympanic cavity and the ambient air pressure, and also with the length of time the pressure alteration acts upon the tissues before equalization takes place. For practical purposes, the differential results in the development of a partial vacuum in the middle ear space. This produces retraction of the tympanic membrane and engorgement of the blood vessels in the ear drum and middle ear mucosa. In mild degrees of barotrauma, the pathological changes may be limited to these. If the pressure differential is great enough and persists long enough, a transudate will usually form. This is usually serous but may be serosanguinous or even hemorrhagic. Rarely, with the development of a severe pressure differential, the ear drum may rupture. The rupture of the tympanic membrane usually occurs in the weakest area or in an area previously damaged by an earlier pathologic process. The formation of a transudate is a more frequent occurrence. Once the fluid accumulates in the middle ear space the pressure differential is relieved. This is eventually followed by auto-inflation with bubbling and resolution of the process when the eustachian tube opens.

**Symptoms** can occur on ascent but are much more likely on descent. Subjective appreciation of the relatively negative pressure in the middle ear space varies with the degree of pressure differential between the tympanic cavity and ambient air pressure as well as the length of time the pressure differential has existed. In minimal barotitis, symptoms are usually limited to a sense of fullness in the ear, mild ear pain, low-pitched tinnitus and decreased hearing (conductive type). With this degree of involvement, symptoms usually disappear soon after ventilation is established. In moderate degrees of pressure differential, all symptoms are of increased intensity. If a transudate occurs, the patient may notice sensations due to fluid movement as head position is changed or during the act of swallowing. In severe barotitis, the pain may develop quickly and become incapacitating. The hearing loss will usually be more severe and the tinnitus greater. Vertigo may be experienced on descent with severe barotrauma; however, this is more likely to occur on ascent with the development of positive pressure in the middle ear space. If the tympanic membrane ruptures, pain and any other symptoms usually subside quickly.

**Clinical findings** during ascent are related to the development of positive pressure in the middle ear space which will cause the ear drum to bulge outward. This is usually of brief duration and unlikely to be seen by a physician unless one is monitoring a flight. The clinical findings on descent vary with the degree of pressure change and the length of time the tubal blockage persists. Development of a relatively negative pressure in the middle ear space produces retraction of the ear drum with prominence of the short process of the malleus and fore-shortening of the long process. The vascular engorgement produces injection and hyperemia of the ear drum which is most marked peripherally and along the long process of the malleus. Hemorrhagic areas may be seen in the drum, again most likely along the long process of the malleus and in the drum periphery. These tiny hemorrhages also occur throughout the middle ear mucosa but usually cannot be visualized. The formation of a transudate may be manifested by a middle ear simply full of serous fluid or there may be an air-fluid interface in the form of a relatively straight line (which will shift with changes in head position) or bubbles may be seen. A hemorrhagic transudate may produce a classical hemotympanum. Perforation of the tympanic membrane may occur at any
point; this will usually be the weakest area in the drum and may be at the site of an earlier perforation which resulted an atrophic scar.

A conductive-type hearing loss is the usual finding; it is generally mild unless there is hemorrhage into the tympanic cavity in which case it may be greater. Weber and Rhinne tests are consistent with a conductive loss (Rhinne: BC>AC and Weber lateralized to affected ear).

The diagnosis in most cases should not be difficult and is based primarily on a history of pain and hearing loss developing during or immediately following descent. Retraction of the tympanic membrane and hemorrhage into the substance of the drum as well as the presence of serosanguinous or sanguinous fluid in the middle ear space aid in diagnosis. The differential diagnosis should include serous otitis media, acute or chronic otitis media, external otitis, and myringitis bullosa. An adequate history of barotrauma as well as the characteristic drum findings should aid in differentiating barotitis media from the other entities.

Treatment depends on the point at which intervention is attempted. In-flight measures should consist of the performance of the Valsalva maneuver as soon as a feeling of fullness is noted in either ear. If the individual has a topical nasal decongestant, the nose should be sprayed. This is best done by spraying each side initially and then applying a second spray a few minutes later after the initial one has had time to shrink the anterior mucosa. The second application has a much better chance of reaching the nasopharyngeal area and shrinking the tubal orifice. If the ear cannot be ventilated by the Valsalva maneuver, return to a higher altitude if operational conditions permit. The ear should then be ventilated and gradual descent carried out, performing the Valsalva maneuver frequently.

If the individual presents to the flight surgeon's office with a fully developed ear block, the initial treatment should be determined by the clinical findings. If there is no evidence of a transudate, an attempt should be made to ventilate the ear and relieve the pressure. Politzerization will usually be required since Valsalva will not be effective (or the individual would probably not have developed an ear block). Either the Politzer bag or a source of compressed air may be used. The nose should be well sprayed with a decongestant solution and maximum shrinkage of the mucosa obtained. For the bag method, the olive tip is placed in one nostril, the nose is compressed between the physician's fingers and the patient is then instructed to say "kick, kick, kick" while the bag is squeezed, thereby increasing the pressure in the nasopharyngeal cavity while the hypopharynx is closed, hopefully to the point where the eustachian tubes will be opened and the middle ear spaces ventilated. If the ear cannot be ventilated by the patient vocalizing, it may be repeated while having the patient swallow a small amount of water during the application of air pressure. If compressed air is used, the pressure is turned down to 4 to 5 PSI and a suitable empty spray bottle that will take a nasal tip (such as a DeVilbiss nebulizer) is utilized for delivering the air to the patient's nose. It may be necessary to increase the pressure gradually several times until air enters the middle ear; however, the pressure should not be greater than 5 to 6 PSI. If politzerization cannot be accomplished at this pressure level, further increase in pressure will very likely not result in success. It must be emphasized that successful inflation of the ear does not necessarily mean immediate resolution of the process. The degree of trauma to the eustachian tube determines this and, if there has been significant trauma to the eustachian tube,
simply ventilating the ear will not be sufficient. The patient can be relied upon to verify that the middle ear has been inflated. If desired, another physician can apply the air to the nasal cavity and one can directly observe the ear drum for evidence of ventilation. It may be necessary to repeat the politzerization for a period of several days until eustachian tube adequacy is established.

If transudation into the middle ear space has occurred by the time the patient is first seen, no attempt should be made to ventilate the ear. The formation of fluid will relieve most of any pain and the patient's only complaint will be the feeling of fullness in the ear and the mild hearing loss. The patient should be started on conservative therapy consisting of topical and systemic decongestants. When the tube begins to open (as manifested by bubbles behind the ear drum) resolution of the process (3-7 days) may be hastened by the institution of the Valsalva maneuver by the patient.

Hemotympanum should be managed conservatively. Prolonged treatment may be necessary since considerable time may be required for blood to clear from the middle ear space. This may be as long as several weeks but usually 1-3 weeks.

Myringotomy is rarely indicated and should be avoided if at all possible. Probably the only absolute requirement for myringotomy would be an flier who absolutely had to return to flying duties for compelling operational reasons during actual warfare. Myringotomy would not restore the eustachian tube to functional status but it would open the middle ear space and prevent the development of further ear symptoms.

Perforation of the tympanic membrane should be treated conservatively. The ear should simply be kept dry and the patient followed. If healing is not well under way by the end of 2 weeks, the patient should be referred to an otolaryngologist.

It cannot be over-emphasized that the management of barotitis is essentially conservative. It should be borne in mind that the mucous membrane of the upper respiratory tract is delicate and nothing should be done that could possibly add to the existing trauma. Catheterization of the eustachian tube orifice is not indicated. It should be obvious that this would only further traumatize the eustachian tube and probably prolong the healing process.

Regardless of the condition in which the patient presents for treatment, therapy must be continued until the process has completely subsided and the eustachian tube and middle ear are functionally normal. In the aircrew member, it is imperative that the individual be grounded for this period and not be returned to flying duties prematurely.

Recurrent barotitis media is essentially the problem of chronic eustachian tube obstruction which is usually secondary to pathology in the nose or the nasopharynx. The most common causes are hypertrophic lymphoid tissue in the nasopharynx, allergic rhinitis, and chronic sinusitis. Gerlach's tonsil (the lymphoid tissue deposited submucosally along the anterior two-thirds of the eustachian tube may be hyperplastic. Occasionally, deflection of the nasal septum may be a significant factor. Treatment should be directed at resolving the primary problem. This may be either surgical or medical.
External Barotitis

External Barotitis is a relatively uncommon barotraumatic injury. It occurs when an object occludes the external ear, creating an air tight space. Unperforated ear plugs (such as some of those used for protection against aircraft noise), tightly impacted cerumen, or any other foreign matter may entrap air in the external canal. During ascent the plug may simply be extruded. However, during descent, this entrapped air may become relatively negative with respect to the outside air pressure. Due to this mechanical effect, the outer epithelial layer of the tympanic membrane or the external canal wall may be sucked away from the underlying tissue and form hemorrhagic areas beneath the epithelium. If these areas become large enough, hemorrhagic bullae may be formed. Small subepithelial hemorrhages usually require no specific treatment. If large hemorrhagic bullae have formed, recovery will be more rapid if the blood is evacuated (with a syringe and needle or by means of a small incision).

No one should be subjected to barometric pressure changes if the external auditory canal is completely obstructed. Noise protectors, small ear phones and similar devices must be vented in some manner so that pressure equalization can occur.

Barosinusitis

Barosinusitis is an acute or chronic inflammation of one or more of the nasal accessory sinuses produced by a pressure difference (usually negative) between the air in the sinus cavity and the surrounding atmosphere. The condition is characterized by pain in the affected region; this pain can develop suddenly and be so severe that the individual will be incapacitated.

The etiology results from a paranasal sinus being a rigid-walled cavity which communicates with the nasal cavity or nasopharynx by way of an ostium. As shown in figure 6-1, during ascent the air in such a cavity will move out by way of the ostium until equilibrium is reached at altitude. During descent air will move back into the sinus cavity until equilibrium is again reached at the earth's surface. This normal movement of air out of and back into the sinus cavity is not perceived and no symptoms occur. Abnormal conditions may alter or even prevent this free flow of air from the sinus and produce symptoms and pathologic changes. The larger sinuses are more often involved than the smaller ones. Those having small caliber tubal structures as exits (such as the frontal sinus) are more likely to be involved than those with large openings. The frontal sinuses are most often involved with the maxillary sinuses second.
Figure 6-1. Barometric Adjustment Within the Sinus on Change of Altitude. On ascent, adjustment of sinus pressure is made by escape of air from the sinus. On descent, adjustment of sinus pressure is made by entry of air into the sinus.

As shown in figure 6-2, obstruction of a sinus opening by redundant tissue or anatomical deformities can occur during ascent or descent. Even though obstruction of a sinus ostium can occur during ascent, it is much more likely to develop during descent. Abnormalities capable of blocking a sinus opening are more frequently intra-nasal than intra-sinal. Since the flow of air from a sinus cavity is outward during ascent, an edematous flap or small polyp will be pushed away and pressure equalized at altitude. During ascent, air can even bubble through thick secretions and permit equalization at altitude. However, on descent the relatively negative pressure inside the sinus pulls the flap or polyp or exudate into the ostium forming an airtight seal and producing a sinus block (figure 6-3). If aspiration of mucopurulent material failed to produce a sinus block, it would contaminate the sinus cavity and could result in a purulent sinusitis.
Figure 6-2. Occlusal Factors in Maxillary Antrum in Flight.

Pathology occurs when the relatively negative pressure inside a sinus results in a space-filling phenomena. The most common of these space filling phenomena are mucous membrane swelling and serous transudation into the sinus cavity or beneath the sinus mucosa. When sufficient space is filled to equalize the pressure differential, the valve mechanism is released and recovery begins. In mild and moderate degrees of sinus barotrauma vascular engorgement and generalized submucosal edema occur. With more severe trauma, mucosal detachment and submucosal hematoma may develop.
The symptoms of barosinusitis are much more likely to occur on descent. Symptoms are generally proportional to severity, ranging from a mild feeling of fullness in or around the involved sinus to severe excruciating pain. This pain can develop very suddenly and become incapacitating. It is thought to be associated with stripping of the sinus mucosa in the formation of submucosal hematoma. There may be tenderness over the involved sinus. There may also be some bloody nasal discharge. There is usually no fever at the onset of the condition; however, within a period of several hours, some temperature elevation may develop depending upon the extent of damage and the degree of associated infection.

The diagnosis of sinus barotrauma must be differentiated from acute purulent sinusitis. A history of pain over one or more of the paranasal sinuses during or shortly after exposure to barometric pressure change usually simplifies the differentiation. In the more severe cases the patient usually describes the onset of pain as quite sudden and very severe. In the less severe cases, the pain may be described by the patient as having slowly developed after return to ground level. This exacerbation of symptomatology after return to ground level may be due to an increase in the relative negative pressure with the sinus following oxygen absorption from the air.

Figure 6-3. Frontal Sinus During Flight.
trapped in the sinus. Epistaxis occurring during or after exposure to barotrauma is strongly suggestive of sinus involvement.

X-rays are probably the most valuable diagnostic aid. The standard sinus series in most institutions consists of Caldwell, Water's, lateral, and submentovertex projections. The Water's projection accomplished in the lateral decubitus position may be very valuable in determining whether or not fluid is present in the sinus cavity. The usual findings are mucosal thickening which may be localized (as in a hematoma) or so generalized and severe that it simply produces an opaque sinus. An air-fluid level may also be demonstrated. These are nonspecific findings and must be correlated with the history. If hematoma is present it is usually found in the frontal sinus and presents as an ovoid density varying from a few millimeters in diameter to practically complete occupancy of the sinus cavity. The hematoma may be single or multiple, unilateral or bilateral.

Treatment should begin at the first sign of barosinusitis and consists of returning to the altitude at which the block occurred, spraying the nose well with a decongestant solution, and then slowly descending to ground level. This may or may not be possible depending upon operational conditions. It is easy to see that this might not be feasible in a combat situation. If the patient is first seen at ground level, at some installations an altitude chamber might be available in which the individual could be returned to altitude. From a practical standpoint, this is rarely necessary and of questionable value in a fully developed case. Generally speaking, the transudate, whether it be serous or serosanguinous, will be sufficiently space filling to relieve the pressure differential thereby alleviating the pain and possibly releasing the flap or ball valve. Active treatment is usually limited to procedures which will relieve persisting pain, promote drainage from the sinus cavity and offer protection against infection. Oral analgesics are usually sufficient. The patient's nasal mucosa should be thoroughly decongested with topical agents and the patient should be given a supply of the same along with a systemic decongestant. The application of heat, preferably a hot pack, is generally appreciated.

In most cases, barosinusitis can be managed conservatively and uneventful recovery is the rule. In milder cases, involvement is usually self-limited and resolution takes place in a few hours to a few days. In the more severe cases, the clinical course may run from a few days to a few weeks. Complete resorption of a submucosal hematoma may take several weeks. If the barotraumatized tissues do not become infected, recovery is more rapid. Should infection occur, a severe purulent sinusitis may result due to the lower resistance of the traumatized tissues and the excellent culture media afforded by the presence of the transudate. Any hemorrhage that occurs is usually self-limited.

It is imperative that the individual remain grounded until fully recovered and the nose and paranasal sinuses functional normally. The patient must not only be asymptomatic but any abnormality demonstrated radiographically must have cleared. A chamber flight should always be required if there is any doubt as to whether or not the individual is fully recovered and ready for return to flying duties.
Prevention is the key in barotrauma. The most important preventive measure which can be emphasized by flight surgeons on a continuing basis is that individuals must not fly when they have an upper respiratory infection. This point cannot be over emphasized; experienced fliers are not immune to barotrauma.

Any significant intranasal condition which could affect the ventilation of the paranasal sinuses should be corrected. This could be uncontrolled nasal allergy, the presence of nasal polyps, a significant septal deviation or even chronic sinusitis.

Alternobaric Vertigo

Ordinarily, expanding air within the middle ear readily escapes since the eustachian tube functions as a one-way valve favoring outflow of air from the middle ear to the nasopharynx. However, if this release of pressure does not occur and a significant positive pressure is produced in the middle ear cavity, symptoms may result, the most prominent and aeromedically significant one being vertigo.

The exact mechanism of production of the vertigo is not known but is has been postulated that unequal pressures between the ears results in differential stimulation of the vestibular system. The existence of positive pressure in the middle ear space is accepted and the resultant vertigo implies stimulation of the vestibular system, probably through an intact oval window. Jones (10) feels that sudden movement of the stapes causes stimulation of the vestibular end-organs. The increase in pressure due to failure of ventilation of the middle ear on ascent is gradual and, in most instances, this is not enough to produce vertigo. The addition of a sudden pressure increment by performance of a forceful Valsalva maneuver can be sufficient for vestibular stimulation. Minimal residual eustachian tube edema secondary to a resolving upper respiratory infection can make ventilation of the ears on ascent difficult and require a more forceful Valsalva maneuver than is usually necessary.

Alternobaric vertigo is probably a fairly common occurrence in pilots of high performance jet aircraft that are capable of a rapid rate of climb. The role that pressure change plays is corroborated by the higher incidence in divers since pressure changes are much greater in an aqueous medium. In 1957, Jones (10) reported an incidence of 10% in 190 pilots that he interviewed. In 1966, Lundgren and Malm (12) reported an incidence of 17% in 108 pilots that they surveyed. The understandable reluctance of pilots to report symptomatology of this type makes it reasonable to assume that this entity is more common than is generally realized.

The flier, usually in a high performance jet aircraft, relates a history of sudden onset of vertigo following performance of a forceful Valsalva maneuver, while attempting to relieve a feeling of fullness in one or both ears. This usually occurs on ascent, but has been described on descent following a particularly forceful Valsalva maneuver. The vertigo characteristically is of brief duration, ordinarily lasting from 10 to 60 seconds. The individual usually feels perfectly normal as soon as the vertigo resolves.
The treatment of alternobaric vertigo is essentially prevention. This implies a continual process of education of aircrew in which the common nature and potential hazards of this condition are emphasized. The admonition "do not fly with a cold" cannot be overly emphasized. It is certainly safe to assume that the most common reason for having difficulty ventilating the middle ear is residual eustachian tube involvement from an acute upper respiratory infection, most likely a common cold. It is also wise to advise aircrew likely to encounter this condition to clear their ears frequently during climb-out and avoid the necessity for a forceful Valsalva.

The aeromedical significance of alternobaric vertigo should be fairly obvious; it is capable of producing sudden incapacity in flight. The fact that it may be of brief duration is of little significance nor is it necessary to assume that it may occur at a critical time in flight. In the operation of high performance fighter-type jet aircraft, all aspects of flight are critical and a time period of several seconds is sufficient for an unsafe operational condition to develop. That alternobaric vertigo can occur and is not uncommon must be realized by aircrew, and receive regular emphasis by flight surgeons.

OTHER VERTIGINOUS ENTITIES

General

Vertigo may accompany various pathological conditions and is particularly important in fliers due to the potential of vertigo producing sudden incapacitation. In considering these disease processes, a clear differentiation must be made between "vertigo" as the term is used by physicians and "pilot vertigo" (spatial disorientation) and alternobaric or pressure vertigo. A detailed history should differentiate these conditions without any particular difficulty.

Vertigo may be defined as a false perception of movement of position. A sensation of revolving or whirling is the most desirable history; however, patients will sometimes refer to a feeling of falling in one direction or another or of being tilted. These latter sensations may be regarded as vertigo since a false perception of position is involved. In any consideration of vertigo it is useful to bear in mind that the symptom is produced by some condition that creates a sudden imbalance in the vestibular system. The site of involvement is usually peripheral but it may be central. Dizziness is a more general term used by patients to describe a variety of sensations. The primary interest here will be with the patient whose symptomatology is in general consistent with vertigo. In this discussion the term "compensation" will refer to the fairly rapid restoration of balance in the vestibular system following acute loss of one labyrinth even though the condition of the end-organ remains unchanged.

The pathological conditions considered here include abnormalities of the inner ear, eighth nerve and central nervous system. There are many middle ear causes of vertigo but these should not be difficult to elucidate, e.g., otitis media. The various inner ear causes of vertigo are not as well defined and the pathological basis for some of them can only be inferred. Only the better
defined and generally accepted conditions will be included here. The reader is referred to textbooks of otolaryngology for more extensive discussions.

The history is the most important part of the work-up. There is no substitute for a detailed history which must be taken by the examining physician with care and patience. This necessarily requires time, a commodity frequently in short supply to flight surgeons. It is often easy to hasten the history and rely upon "tests" to clarify the diagnosis. If the physician does not have a fairly good idea of the diagnosis when the history is completed, it would be wise at that point to talk further with the patient. The physician should remember that if the examination and various tests are all within normal limits, the history will be all that there is to go on. The appreciation by the patient of some type of motion is important in the delineation of vertigo. The patient should be asked if the dizziness is associated with loss of balance; generally, if the patient is experiencing vertigo, they must hold on to prevent falling or sit down or even lie down. If possible, it should be determined whether or not the patient had nystagmus with the vertigo since nystagmus is the only objective manifestation of vertigo. This can best be determined from others who may have been in a position to observe the patient's eyes during the acute attack. The physician should ascertain whether the vertigo is episodic or continuous; also whether it occurs when the patient changes to a sitting or standing position or as a result of any other specific change of head position. Movement of the head will usually aggravate vertigo. Certain diseases or syndromes characterized by vertigo present a fairly typical history. Any "pattern" of the vertigo should be elicited, if possible. Certain characteristics of vertigo should be asked about. These include the mode of onset, nature of attacks, any precipitating factors, and any possible predisposing conditions. Associated complaints may be very useful in establishing whether the vertigo is peripheral or central in origin. Auditory phenomena include hearing loss, tinnitus, diplacusis (perceiving two sounds when only one is present), and recruitment; all are cochlear in origin and localize to the end organ. A sensation of fullness in the ear may be noted prior to and during attacks of Meniere's disease. Ocular phenomena include a jerking or motion of objects in the field of vision (oscillopsia), diplopia, and blurring of vision or blindness. Oscillopsia is probably the subjective appreciation of nystagmus which is more likely on a central basis (in peripheral involvement the vertigo is usually severe and over-powers the appreciation of nystagmus). Diplopia is usually central in origin as is blurring of vision or blindness. Nausea and vomiting are usually of peripheral origin unless the vomiting is projectile. Headache is uncommon in peripheral labyrinthine conditions. Loss of consciousness is a definite central sign; if associated with vertigo, the vertigo will be due to a central lesion.

In addition to a complete ear, nose and throat examination, the patient should be checked for spontaneous nystagmus. This should be carried out with the patient seated (the "neutral" position) and looking straight ahead (to eliminate any gaze factor). Positional testing, including the Dix-Hallpike maneuver, should be done with direct observation (even though the same testing may be repeated later during electronystagmography). The cranial nerves should be examined and cerebellar function assessed.

The audiometric evaluation should be as complete as possible: ideally, this should include at least pure-tone and speech audiometry. Tests for recruitment and tone decay should be performed if indicated. A functional assessment of the vestibular system must be done. An
eltronystagmogram (ENG) should be accomplished if facilities are available. This provides for a graphic record of caloric responses as well as certain other tests (gaze nystagmus, optokinetic nystagmus, spontaneous and positional nystagmus, fixation suppression).

Routine views of the internal auditory meati should be obtained if an acoustic neuroma is suspected. A metabolic work-up should be performed to rule out thyroid disease, diabetes, hyperlipidemia, tertiary syphilis, etc. An MRI with gadolinium is the most sensitive imaging technique, but a CAT scan will suffice if and MRI is not available.

**Inner Ear Conditions**

**Meniere's disease.** This is one of the most common otologic conditions which is manifested by vertigo. In a well established case, the history is typical. The patient will describe recurrent attacks of vertigo which last for several hours (usually 2-6), frequently preceded by an aura, and accompanied by tinnitus, hearing loss and often a feeling of fullness in the involved ear (in most patients, the involvement will be unilateral). The tinnitus is usually described as buzzing or roaring (not as a high pitched ringing sound). During an acute attack there may be nausea and vomiting and a spontaneous nystagmus may be observed. The aura usually consists of a feeling of imbalance or unsteadiness or possibly a return to the feeling of ear fullness, or the tinnitus may recur (or persisting tinnitus may get louder). The typical hearing loss is a flat sensorineural type which involves the speech frequencies. In early stages, the hearing may return to normal between attacks; later, it may improve between attacks. Diploacusis, or double hearing, will usually be noted by the patient and can be demonstrated with a tuning fork. Recruitment will be noted by most patients; it can be suspected if the patient states that loud sounds bother the ear with the poorer hearing. Early in the course of the disease, the caloric response may be normal but the usual picture is one of increasing depression with recurring episodes.

During the initial stages Meniere's disease may be atypical. The hearing loss may be the first manifestation and precede vertigo by months or even years. On the other hand, patients may be seen for vertigo which is fairly typical for Meniere's disease but have no hearing loss. Generally, a patient that presents with vertigo or hearing loss will develop the other manifestation within approximately one year; this premise can be useful in the management of aircrew who are suspected of having Meniere's disease. It is preferable to diagnose the specific manifestation (i.e. vestibular hydrops or cochlear hydrops) and list it as "cause undetermined" rather than use such terms as "atypical Meniere's disease" or "pseudo-Meniere's disease."

**Benign paroxysmal positional vertigo** (BPPV) is a well described entity surrounded by some controversy. Some patients will give a history of a definite relationship between assuming a certain head position and the onset of vertigo. The usual head position is one in which one ear is dependent. The vertigo produced will be sudden in onset, severe, accompanied by nystagmus and last for only a few seconds (not minutes or hours). These patients have normal hearing and normal caloric responses, but the positional tests are generally positive. The Dix-Hallpike maneuver is usually the most useful of the various positional tests; it consists of a combination of movement and the assumption of a certain position. In this maneuver the patient, with head turned either to the right or left, is quickly moved from the sitting to the supine position with the head hanging
over the end of the table (approximately 30 degrees below horizontal). To be considered diagnostic for benign paroxysmal positional vertigo the response should exhibit brief latency, severe subjective vertigo and objective rotary nystagmus and fatigability on repetitive stimulation. The neurological examination will be otherwise normal. Symptoms gradually clear spontaneously within one year in most patients and do not usually recur. The “particle repositioning maneuver may be effective in treating patients on initial presentation with BPPV. Benign paroxysmal positional vertigo may be associated with head trauma, viral labyrinthitis, or may be idiopathic.

**Toxic labyrinthitis.** The most common offenders today are the ototoxic antibiotics. The vertigo is usually not typical since both labyrinths are equally depressed. The primary manifestation is generally difficulty in ambulation with inability to "stay on course." Hearing loss may or may not occur; if present, it is generally accompanied by a high-pitched tinnitus. Since there is no specific therapy for this type of vestibular damage other than withdrawal of the agent, baseline studies are imperative and any evidence of ototoxicity should be closely watched for any time a drug that is suspected of being ototoxic is employed. The patient will gradually "compensate" for the decreased vestibular input but may have difficulty in situations where adequate visual orientation is not possible.

**Serous labyrinthitis** is generally regarded as the labyrinthine response to some type of aseptic irritation, such as direct trauma to the vestibule. The most common cause today is stapedectomy. Management is conservative since this variety usually resolves without residua.

**Infectious labyrinthitis** is the most common viral involvement of the vestibular system. McCabe (13) feels the viral labyrinthitis, vestibular neuronitis and epidemic vertigo are all based on a common pathological process, namely, a viral involvement of the vestibular system. This produces a sudden vestibular crisis which generates acute symptoms for a few days (usually) and then gradually subsides over the next several days to possibly a few weeks. It may occur in "epidemic" form; young adults are primarily affected. Typically, it does not recur; this has considerable aeromedical significance. In the vestibular neuronitis variety, there is a unilateral vestibular deficit after the acute symptomatology clears.

The vertigo is usually severe for a few days and there may be nausea and vomiting at its height. This gradually regresses over a variable period of time; in most cases this is only a few days. If there is unilateral loss of function, some imbalance and difficulty with sudden head movements may persist until compensation is complete. Typically, there is no evidence of cochlear involvement (no tinnitus or hearing loss). The caloric response is usually depressed or absent in the involved ear in vestibular neuronitis; this is usually permanent. Responses are usually normal following the other varieties of viral involvement of the labyrinth.

**Bacterial labyrinthitis** is somewhat uncommon in this antibiotic era; however, it may follow an acute otitis media, be secondary to chronic otitis media, or may complicate a skull fracture or ear surgery. It is unlikely that purulent involvement of the labyrinth would develop suddenly; the patient would most likely already be grounded and might well be hospitalized.
**Perilymph fistula** is a recently described phenomenon. The usual history is sudden hearing loss following physical stress or trauma (including surgery). It is distinguished from Meniere's in that symptoms are more constant and positional. The fistula test may or may not be positive, depending on whether the fistula is blocked by granulation tissue or other obstructions. The treatment is usually one of waiting. Surgical intervention may be needed. Unfortunately current experience with perilymph fistula in the flying population is limited. Persistent vertigo is disqualifying and middle ear surgery is also disqualifying. Waiver for flying duty will be handled on an individual basis and will, in most cases, require an Aerospace Medicine Consultation Service (ACS) evaluation. A period of observation ranging several months to a year will likely be required.

**Trauma** such as a transverse temporal bone fracture, usually destroys the labyrinth. This end organ destruction results in the vestibular system imbalance and vertigo persisting until compensation occurs. Hearing loss and tinnitus usually accompany the vertigo; facial paralysis may occur with this type of fracture. Concussion can evidently destroy the inner ear because patients are seen with a nonfunctioning ear who give a history of sustaining a blow localized to or near the ear (e.g., baseball injury).

Anyone applying for military flight training with a "dead ear" should be disqualified; however, an experienced flier could be considered for return to flying duties following an injury of this type if there are no significant accompanying residua (such as a facial paralysis).

**Of the eighth nerve conditions** the most important one is the **acoustic neuroma**. Typical vertigo does not ordinarily occur since the vestibular nerve fibers are gradually destroyed. Cochlear manifestations usually appear first but may be very insidious in onset. A unilateral sensorineural hearing loss which involves the higher frequencies should always make one suspect an acoustic neuroma. It is disturbing to realize, as reported by Pulec et al (14) that the initial symptom will be sudden hearing loss in a significant number of these patients (17% in their series); however, in the majority of cases, this lesion should have little aeromedical implication since sudden incapacitation is not likely.

**Central vertigo** is often insidious in onset, will frequently have been present for several weeks or even months, will often be mild and frequently will not be accompanied by nystagmus. It is helpful to remember that there will usually be other signs of central involvement. A spontaneous vertical nystagmus that can be seen by an observer is generally considered to be pathognomonic of a central lesion. Monocular nystagmus and dissociated eye movements are always central in origin. Vertebro-basilar insufficiency is probably the most common central condition that is frequently manifested by vertigo. It is usually regarded as a central condition because ischemia due to decreased blood flow through this system should affect the brain stem earlier than a peripheral structure. However, some authorities disagree with this and feel that symptoms are due to end-organ ischemia. The vertigo is usually intermittent and of brief duration. Common precipitating factors are neck movements (such as hyper-extension by overhead work or extreme rotation as in backing or turning a vehicle) and anything which causes lowering of systemic arterial pressure (such as postural change).
Other neurological conditions which may be manifested by vertigo include vascular accidents, multiple sclerosis, epilepsy, migraine, neoplasms, and head injury. These are discussed in other chapters of this publication.

Aeromedical Implication of Vertigo

An episode of vertigo in an aircrew member has particular significance because of the possibility of recurrence with attendant sudden incapacitation. There are several fairly well delineated vertiginous entities which are sufficiently understood to permit reasonably accurate prediction about the likelihood of recurrence. The better the patient's history and findings agree with an accepted condition, the easier the disposition will be. For example, a patient with a fairly typical history and findings of Meniere's disease should be permanently grounded since symptom recurrence is probable and the interval unpredictable. Conversely, one with a history and findings of vestibular neuronitis can be returned to flying duties after compensation for the labyrinthine deficit occurs since recurrence in this entity is unlikely. In other instances, such as benign paroxysmal positional vertigo and post-traumatic vertigo, a period of observation (usually several months) is generally required before a decision regarding flying duties can be made. Unfortunately, many patients are seen where the history and findings are not typical for any of the generally accepted conditions. If the involvement can be regarded as a single episode, eventual return to flying duties is possible if the patient remains asymptomatic and all studies are normal. Such patients very likely represent atypical presentations of viral labyrinthitis. If the vertigo has been recurrent, return to flying duties is less likely since the possibility of Meniere's disease must be given greater consideration. Vertigo on a central basis may not be accompanied by enough other symptoms and signs to make its origin reasonably clear. In all these atypical cases, an interim period (usually 6-12 months), with re-evaluation, is often helpful and disposition can more confidently be made with due regard for flight safety.

Vertigo in aircrew will remain a problem until the various entities are more clearly defined and the prognosis as to recurrence is more reliable.

MISCELLANEOUS ENT CONDITIONS

Ear

Acute diffuse otitis externa. This is the most common form of external otitis and it can be a significant problem in military population, particularly in tropical areas. Pseudomonas is the most common organism isolated when material from the ear canal is cultured. However, the infection behaves more like an infection due to streptococcus or staphylococcus than gram negative infection. Fungi frequently are present but are rarely, if ever, the etiologic agent. Infection usually follows a change in pH of the skin from the normal acid range ("acid mantle") to an alkaline one.

Pain is the earliest and most prominent symptom and can vary from mild to excruciating. The most useful diagnostic point is pain on pinnal traction or tragal pressure. The canal may be mildly
inflamed to swollen shut and be exquisitely tender. Periauricular cellulitis may be present with involvement of periauricular lymph nodes. A purulent discharge is usually present. Hearing loss is secondary to obstruction of the external ear canal; it is conductive in type and usually very mild. Of course, an external otitis can complicate an otitis media if the drum ruptures and drainage wets the canal. The tympanic membrane must be visualized to determine whether or not middle ear infection has occurred, unless there is obvious mastoiditis. With mastoid involvement, there will be redness, swelling, and tenderness over the mastoid process (not the entire periauricular area) which may push the ear forward and outward.

Thorough cleaning of the ear canal is the most important part of the treatment regimen and it is certainly the most difficult. This is best accomplished by using normal saline or ear suction. If possible, the drum should be visualized at least as much as the swelling will permit. Initially, Burrow’s solution (1 to 20) is the best agent to restore the normal pH. This reduces the swelling and helps to restore the normal acidity of the skin. A wick fashioned of cotton or an Otowick(R) may be used. The wick should be removed in approximately 24 hours, the canal again carefully cleaned, and the wick re-inserted. Burrow’s solution should be used until the edema has pretty well subsided; then one of the steroid antibiotic preparations in a suspension form can be tried. As the inflammation subsides, wicks can be left in for as long as 48 hours. If the wick comes out, the patient may put the drops directly in the ear until seen again. Medication can be applied without a wick by instilling the drops directly into the ear canal; however, the wick aids medication distribution throughout the canal. Treatment should be continued until the canal has returned to normal size and is no longer tender. A systemic antibiotic is given if there is marked swelling of the canal or periauricular involvement. Cultures should be done routinely, but treatment should be started prior to receiving the report. The choice of antibiotics should be based on the assumption that the agent is a streptococcus or staphylococcus. Hot packs for 15 to 20 minutes every two hours will provide significant pain relief. The individual should keep the ear canal dry and may use Domeboro(R) otic solution or a white vinegar and alcohol solution after showering or swimming to help prevent recurrence.

Any aircrew member with acute otitis externa should be grounded until the involvement has resolved, earphones or helmet can be worn without discomfort, and, if necessary, plugs can be tolerated for noise protection.

**Furuncles** of the external ear canal are a localized or discrete otitis in the outer third of the canal. Management here is the same as for a furuncle elsewhere - hot packs, analgesics and incision if required. For large or multiple lesions an antibiotic should probably be added, especially if there is an associated cellulitis.

**Foreign bodies** in the external ear canal may be classified as animate (living or dead) or inanimate and organic or inorganic. Management consists of removal of the foreign body and treatment of any secondary problem (such as an external otitis or perforated ear drum). Removal can be made more difficult by prior unsuccessful attempts. The safest and easiest way to remove any foreign body is irrigation with water at body temperature, if the physician can be reasonably certain that the drum is intact. If the object is hygroscopic, the physician is committed to removal before appreciable swelling occurs. This aspect can be minimized by using alcohol (95%
preferably) as the irrigant. If the foreign body is a live insect, it should first be killed by filling the canal with alcohol or a light oil. If the foreign body cannot be washed out, instrumentation will be required. It may be possible to suck it out. If the object can be grasped (e.g., a wad of paper), a small alligator forceps can be used. A Buck ear curette may be useful to carefully work the object out. Care should be maintained to avoid traumatizing the canal. Rarely, it may be necessary to resort to surgical removal. Any damage to the canal wall skin or ear drum should be treated as required and the patient followed until recovery is complete.

**Myringitis bullosa** is a condition in which bullae or vesicles form on the external surface of the ear drum. These blebs usually contain serous fluid, but may contain serosanguinous or frankly sanguinous fluid. No specific etiologic agent has been identified. These bullae may occur with viral upper respiratory infections or as the initial phase of an acute otitis media.

Pain is the most impressive manifestation of this entity and is usually described by the patient as very sharp and severe. It usually subsides in 12 hours for most cases. The onset is usually sudden and frequently occurs in the early morning hours. This pain is thought to be due to vesicular fluid dissecting the skin layer of the ear drum; it usually stops when the vesicle stops enlarging. A mild hearing loss, tinnitus and a feeling of fullness may also be noted. Examination usually reveals amber or reddish-purple blebs on the ear drum. These are thin-walled and rupture easily.

If the patient is still in pain when first seen, rupturing the bleb will bring immediate relief. The bleb may already have ruptured by the time the patient is seen and only a raw area, or possibly an early encrusted area, will be seen on the surface of the drum. These vesicles can be ruptured with a small suction tip or with a myringotomy knife. Of course, eliminating the bleb will permit much better visualization of the ear drum. If desired, an antibiotic powder can be instilled on the raw surface of the drum. If an associated otitis media is present, this must be adequately treated.

**External ear canal polyps** or polypoid masses that present in the ear canal may be a tumors of the ear canal; however, these usually result from chronic middle ear and mastoid disease (i.e. cholesteatoma) and will, in most cases, be attached somewhere within the middle ear space and will be protruding through a perforation in the ear drum. The flight surgeon should not attempt to remove one of these polyps, even if it appears to be quite accessible, since the stapes may be avulsed, resulting in a purulent labyrinthitis. These patients should be grounded and then referred to an otolaryngologist for management.

**Traumatic perforation** of the ear drum may result from a slap or blow to the auricular area, or by insertion of a cotton tip applicator or other object into the external ear canal with accidental perforation of the tympanic membrane. Perforation of the ear drum can also result from proximity to any type of explosion. The patient will frequently mention noting a momentary sharp pain when the perforation occurred, followed by a dull, aching pain. The hearing loss is minimal unless there is dislocation of an ossicle (usually the incus) in which case a moderate conductive loss will be noted.
Examination will reveal perforation of the tympanic membrane, typically located on the posterior half of the eardrum, though any part of the drum may be involved. These perforations vary from small to very large. The margins of the perforation may be jagged.

Treatment of a traumatic perforation consists primarily of watchful waiting and prevention of moisture gaining entrance to the middle ear. Topical treatment will tend to irritate the middle ear mucosa. Antibiotic therapy is necessary only when the perforation has definitely resulted from direct contact with a contaminated foreign body. The middle ear should be regarded as contaminated when the perforation resulted from diving into water. These perforations usually heal over a period of several days to several weeks. As long as the perforation is closing, the flight surgeon should manage the patient. If it becomes evident that the perforation is not closing or if the perforation is complicated by infection, the patient should be referred to an otolaryngologist for management. Periodic audiometry is a mandatory part of the routine care during the healing phase.

**Chronic otitis media.** Most physicians outside the specialty of otolaryngology have little knowledge of chronic otitis media and many would experience difficulty in establishing the diagnosis and instituting proper therapy.

The most common complication of acute otitis media is the development of chronic infection in the contiguous recesses of the middle ear, the antrum, and mastoid air cells. Continuation of recurring drainage from the middle ear is the most constant symptom of a chronic suppurative otitis media; it may vary from foul-smelling to nearly odorless, depending on the organism involved. If pain should occur in the chronic stage it is either due to an acute exacerbation of a chronic infection or the development of a complication. The conductive hearing loss which accompanies a chronic otitis media varies from minimal when there is no ossicular chain defect to a moderate degree of deafness when there is ossicular discontinuity.

If chronic otitis is associated with ear drum perforation, the site and characteristics of the ear drum perforation may help assess the extent of disease present. Unfortunately this may be difficult to ascertain without an operating microscope, as the perforation may be small or covered by granulation tissue or scabbing. An anterior central one is usually referred to as a tubal perforation and the discharge is usually mucoid. The source of the pathology is frequently chronic infection in the nasopharynx or tonsils with secondary extension along the eustachian tube. The central type is frequently seen following an acute otitis media with perforation of the tympanic membrane. A long standing central perforation may be associated with caries of the ossicles and an ossicular defect may have developed (this is most frequently absence of the long process of the incus). Occasionally, a central perforation will be obscured by granulation tissue filling the middle ear. Marginal perforations should be regarded with suspicion since they may indicate the presence of cholesteatoma. Foul otorrhea from a perforation may be a cholesteatoma. Two common locations of this type of perforation are posterior-superior and in the epitympanum or attic. Both locations provide an opportunity for epithelium to invade the tympanic cavity. Here the squamous epithelium proliferates to form a cyst and casts off squamous debris until a mass is formed. This cholesteatomatous mass is capable of expanding with continued growth. As the mass enlarges, it can destroy bone. This may extend intracranially with resultant brain abscess or meningitis or
through the bony canal of the facial nerve producing facial paralysis. Cholesteatoma usually presents as whitish debris which can sometimes be seen in the perforation.

Any crew member in whom the history and findings suggest chronic otitis media should be grounded, placed on systemic antibiotics, maintain hygiene of the canal, and referred to an otolaryngologist for management. Conservative therapy will be tried initially; there may be a contributory factor (such as hypertrophic lymphoid tissue in the nasopharynx or the presence of allergic rhinitis) which may require attention. Frequently, definitive treatment will be surgical and can vary from a simple myringoplasty to more complicated procedures. The therapeutic aim will always be twofold: to eliminate infection and to maintain or improve hearing.

If a crew member with a dry central perforation is seen, the flight surgeon may question why this should be considered cause for grounding. These individuals are subject to recurrent drainage, particularly with an upper respiratory infection or if moisture is introduced into the ear. Also, they are vulnerable to the development of any of the complications of chronic middle ear disease, including formation of a fistula into the inner ear with vertigo (which could be suddenly incapacitating).

**Otosclerosis** is one of the osteodystrophies in which formation of abnormal bone occurs in the petrous portion of the temporal bone. The site of predilection is the area just anterior to the footplate of the stapes. If the proliferation of abnormal bone encroaches of the oval window area, it can interfere with movement of the stapes and produce a conductive hearing loss. This hearing loss is usually accompanied by tinnitus. Symptoms usually begin in early adult life and will typically be progressive until the process becomes inactive. The hearing loss can vary from mild to severe. The involvement is usually bilateral. The hearing loss may exceed the limits of serviceability in one or both ears.

An aircrew member with otosclerosis may develop enough hearing loss to have difficulty in the performing ground duties and off duty activities. The individual will usually not have any difficulty while airborne since discrimination is good and the conductive deficit can be eliminated by increasing the volume in the communication system. Additionally, the conductive loss acts as a built-in barrier against noise damage.

Any aircrew member with otosclerotic hearing loss should be strongly advised against any immediate surgical treatment since any procedure that involves surgical violation of the oval window or the emplacement of any prosthesis is disqualifying for flying at this time. A waiver for substandard hearing can usually be obtained since the crew member will likely have no difficulty while airborne. Such individuals can be satisfactorily rehabilitated with a hearing aid which can be worn as desired when not flying. Surgery can always be considered when flying status is no longer a consideration.

**Nose**

**Foreign bodies** of the nose usually present with a history of the onset of a unilateral nasal discharge which soon becomes purulent. Occasionally the individual may have a foul smelling
breath or others may complain of unpleasant odors. The duration is usually days to weeks but may be even longer. The patient is usually a child and the parents often have no knowledge of any foreign body. The usual finding is a unilateral purulent nasal discharge and some type of foreign body impacted in the nasal vestibule of the anterior portion of the inferior meatus. Flattened or thin foreign bodies (such as a coin) may be more posterior in the nose. Granulations may be present and may bleed excessively. Sometimes the granulations will be so extensive that it will be difficult to determine that a foreign body is present. Removal of the foreign body requires a good light source, strong suction, and proper instruments. This can best be done with the patient in a sitting position; the patient should be restrained if necessary. The nose should be cleaned as thoroughly as possible and a decongestant spray used to maximally shrink the mucosa. If the object can be grasped (such as a wad of paper or a piece of plastic), a small alligator forceps may be used. Do not try to grasp a hard object with such forceps since it can easily be driven farther back into the nasal cavity. A hard object (such as a small rock or a bead) can frequently be removed by passing a bent wire loop over and behind it and working it out anteriorly. An antibiotic may be indicated if there is appreciable associated infection. It is essential that the patient be followed until the nose has healed completely and is functionally normal.

**Bleeding.** The anterior septal area is the most common site of nose bleed and the most common etiologic factor is trauma, though the type of trauma may be obscure. Nose picking is very common in children and the anterior septal area is the usual site. Forceful nose blowing can cause nose bleeds, particularly with upper respiratory infections when the mucosa is edematous and more easily traumatized. Drying and cracking of the mucosa during periods of low environmental humidity (as found during the winter heating season or desert summers) may lead to bleeding. Epistaxis can complicate a maxillofacial injury. Arteriosclerosis and hypertension frequently coexist in elderly patients. Hereditary hemorrhagic telangiectasia occur in the nose and, even though the lesions are located throughout the nasal cavity, those in the anterior septal area are vulnerable to trauma and will generally be the ones that will bleed. These patients may bleed spectacularly. Patients with blood dyscrasias frequently develop epistaxis; the bleeding may be diffuse and bilateral. Any inflammatory process in the nose can produce granulation tissue. Trauma usually initiates the bleeding. Neoplasms may present as epistaxis. The hemangioma or "bleeding polyp" is usually located in the anterior septal area where it is easily traumatized.

Prior to instituting any treatment for epistaxis, you should assemble all that will be required. This includes a good light source, strong suction, an angled Frazier sucker, a nasal speculum, and a bayonet thumb forceps. Also available should be half-inch Vaseline(R) gauze for anterior packing, previously prepared postnasal packs, catheters, and a large forceps and a mixture of topical 4% lidocaine and neosynephrine (1/2 %). It is good practice to have all this pre-assembled as an "epistaxis tray." Patients should be examined sitting up unless the patient is very weak or in shock. It is easier to look in a nose with the patient sitting up. This also decreases the venous congestion and blood pressure to the head and thus helps slow the flow. As soon as possible, the patient should be given an injection of an analgesic to reduce pain and anxiety. This will also be beneficial if the patients blood pressure is elevated.

The nose must be cleaned as thoroughly as possible by suctioning the clots. Pack the nose with the neosynephrine/lidocaine mixture on cotton for 5-10 minutes. Remove the pack and attempt to
locate the bleeding point. In this regard, certain generalities may be helpful. Bleeding will usually be from one side only and usually from only one spot, even though some blood may come out of the opposite nostril. Bilateral nosebleeds sometimes occur in nasal fractures and in patients with blood dyscrasias. The opposite side can be stirred up by instrumentation. Always inspect the anterior septal area first; in most cases, this is all that will be required. If no bleeding is found there, it can be assumed that the bleeding is posterior, particularly if blood continues to go down the back part of the throat. An attempt should be made to determine if the blood is coming from high in the nasal vault (anterior ethmoid origin) or from lower down (sphino-palatine source). Bleeding from the posterior half of the nose is not likely to be due to external trauma; it is usually caused by splitting or rupture of a sclerotic blood vessel. The bleeding is always worse if the patient is also hypertensive. Posterior bleeders are larger vessels and the bleeding is usually more severe. This type of bleeding can usually be controlled only by adequate control of the bleeding point.

Anterior septal bleeding points can usually be readily controlled by a small cotton pack. The cotton should first be moistened with a suitable topical anesthetic agent (1% lidocaine with epinephrine 1:100,000). This pack can usually be removed in a few minutes and the site of bleeding cauterized, either chemically (50% trichloracetic acid, silver nitrate) or electrically and then packed over night. If the bleeding is severe and the point cannot be visualized, or the bleeding is diffuse, an anterior nasal pack can be installed. Half-inch Vaseline gauze is a good material to use for this. Insert the Vaseline gauze with a bayonet thumb forceps, with the nasal speculum opened as widely as possible for maximum visibility. Pack toward the bleeding point in an effort to bring it under control as quickly as possible. Use suction as necessary in order to keep the nose as clear as possible so you can see what you are doing. Firm packing will usually be required. If the bleeding cannot be controlled by anterior nasal pack, it should be removed and both the posterior and anterior packing installed. A balloon device may be utilized as a posterior pack. Whenever anterior, or both anterior and posterior packing is required, it should be left in place for 3-5 days from the time the nose bleed is controlled. The patient should be on a systemic antibiotic while the nose is packed and it is a good practice to continue the antibiotic for 1 or 2 days after the nasal packing is removed. If the patient presents with brisk bleeding, packing will usually have to be resorted to as an initial measure; if this is successful, further measures are not required. The most common reason for failure to control a nose bleed with packing is improper installation of the packing. Since most nosebleeds are anterior and not severe, most patients with epistaxis do not require transfusion; however, severe bleeding will frequently require transfusion. Hemoglobin determinations made immediately after a nose bleed may be misleadingly high because of hemoconcentration. It should be checked again when the patient has been adequately hydrated. Continued uncontrolled bleeding may require more drastic intervention such as arterial ligation. If a posterior pack must be placed, evacuation of the patient to an otolaryngologist should be considered. Immediate resolution of the bleed will not usually occur in these cases and there may be slow leaking for several days.

The aeromedical implication of epistaxis is obvious; the crew member must be grounded until recovery is complete and the nose is functionally normal. Any predisposing or contributory condition must be fully evaluated and its aeromedical significance carefully considered.
Paranasal sinuses

**Acute sinusitis.** Maxillary sinusitis is the most common, followed by frontal involvement. Acute sinusitis usually follows an upper respiratory infection. Individuals with nasal allergy are more susceptible to sinus infections. Patients with acute sinusitis may have minimal symptomatology; with severe involvement, symptoms are more impressive. These patients may be febrile and quite ill, even prostrate. Nasal discharge is usually noted, frequently unilateral. Patients will usually say that this drainage is different from what they had noted earlier with the head cold. With maxillary involvement, the patient may mention a fullness in the cheek with a dull, aching pain. The individual may also have noted pain in the upper teeth on the involved side. There may be a history of recent extraction of an upper molar on the involved side. In frontal involvement, patients usually mention that one or both frontal areas are tender. Occasionally, a patient with frontal sinusitis will tell you of improvement in symptoms as the day goes along; this is due to gravity aiding drainage in the upright position. In acute ethmoid disease, pain may be localized over the involved side or may be felt in the frontal area or eye. Tenderness can often be elicited by palpation with the finger along the inner aspect of the orbit over the region of the lacrimal bone of the lamina papyraceas of the ethmoid. With sphenoid involvement, there will not be pain or tenderness over the involved sinus since this is not a subsurface cavity. The headache which may occur with acute sinusitis is generally secondary to an acute febrile state. As a general rule, localized pain and tenderness are more reliable historical points than headache.

Examination will usually reveal redness and swelling of the nasal mucosa of the involved side. If pus is found in the nasal cavity, its source may or may not indicate which sinus is involved. Drainage from the frontal sinus is usually far anterior in the nose and comes down across the anterior tip of the middle turbinate. Drainage from the maxillary sinus usually presents below the middle turbinate or will be seen dripping off the posterior end of the inferior turbinate into the nasopharynx. Anterior ethmoid drainage will present in the middle meatus; posterior ethmoid and sphenoid drainage will be seen on the lateral wall of the nasopharynx, coming out of the sphenoid recess. Subjective tenderness may be demonstrable, either over or under the eye or the cheek area. There may be detectable swelling in the frontal area. With marked frontal involvement, this swelling may extend inferiorly to involve the medial aspect of the orbit. Swelling of the medial aspect of the orbit without frontal swelling would very likely be due to acute ethmoid involvement.

Transillumination is a crude but useful screening procedure. It may be reduced or absent for the frontal and maxillary sinuses. It can be misleading early in an inflammatory process. In general, failure of a frontal or maxillary sinus to transilluminate in a patient suspected of having acute sinus infections should be regarded as an indication for sinus x-rays. X-rays are the best diagnostic tool for evaluating the paranasal sinuses, and in most cases, sinus film have the last word. The usual finding is some degree of clouding or opacification of the involved sinus, or a fluid level may be seen. The clinical picture must be correlated with the radiographic findings in order for the x-rays to be properly interpreted.
The basic premise in the therapy of paranasal sinus disease is to establish and maintain drainage. Sinusitis complications are unlikely and uneventful recovery is the usual outcome. It is a fairly reasonable assumption that many patients with sinusitis are never seen by a physician and clear spontaneously without any specific treatment. Maxillary sinusitis is seen most commonly in clinical practice; these patients are not usually seriously ill and can generally be managed on an out-patient basis. Patients with frontal sinusitis must be watched closely and may require hospitalization. ENT consultation should be considered. An adequate course of an appropriate antibiotic should be given. When selecting the antibiotic agent, it should be borne in mind that the most common bacteria responsible for this entity are streptococci, staphylococci, pneumococci and Hemophilus influenzae. Both local and systemic vasoconstrictors should be employed; it is a good idea to combine the systemic decongestants with a suitable antihistamine. Supportive therapy includes bed rest, local wet heat, moist environment and analgesics, if needed. Irrigation may occasionally be necessary in acute sinusitis, but it is wise to wait until the patient has been under treatment for several days, if at all possible. Cannulation of the maxillary sinus may be done by any physician after proper instruction in the technique; however, the flight surgeon might prefer to refer the patient if irrigation of a sinus cavity may be required. Surgical procedures are rarely necessary in acute sinusitis; trephining an acute frontal empyema is sometimes required.

Intracranial extension with brain abscess or meningitis is probably the most serious complication which can occur with acute sinusitis; this is most likely with frontal involvement. Orbital cellulitis is usually of ethmoid origin.

Aircrew members with acute sinusitis must be removed from flying status until the infection has cleared completely and the nose and sinuses are functionally normal. Follow-up x-rays should always be obtained to rule out the possibility of persisting infection.

Cystic lesions of the paranasal sinuses are very common. In one study (8) an incidence of approximately 10% was found. The maxillary sinus is the most common site; however, soft-tissue densities are also found less frequently in the frontal and sphenoid sinuses. Most of these lesions are pseudocysts or interstitial cysts and consist of a submucosal accumulation of clear, yellow fluid without a definite epithelial lining. The usual radiological appearance is a smooth, rounded mass located inferiorly in the sinus; however, the site may be lateral, medial, or even superior in the sinus. A smooth, rounded soft-tissue density located superiorly in the maxillary sinus may be a cyst or polyp; the distinction cannot be made radiographically. Some of the cystic lesions are "soft"; these may look much like air-fluid levels with the patient upright. The rounded configuration typical of a cyst can sometimes be demonstrated in the lateral decubitus position.

These patients are usually asymptomatic and the cystic lesion is an incidental finding. No treatment is required in most cases; however, follow-up by periodic x-rays is indicated. Hanna(8) revealed that the most likely course for a cystic lesion is to remain essentially unchanged for long periods of time. These lesions can increase in size, decrease in size and even clear spontaneously. Soft-tissue densities located superiorly in the maxillary sinus (and probably those in the frontal sinus also) should be followed more closely initially and until it can be determined what their course will be.
These cystic lesions of the paranasal sinuses are not causally related to flying and should not affect flying status since the lesion is asymptomatic and is usually an incidental finding. These cysts should, however, be followed by serial x-rays, at least annually. Systematic follow-up should make it possible, in most instances, to detect in time any soft-tissue density which proves to be a mucocele. Surgical intervention is necessary for a mucocele since erosion of bone may occur.

Larynx

Hoarseness is generally accepted as one of the common manifestations of a laryngeal abnormality. Hoarseness can be produced by extra-laryngeal causes (e.g., paralysis of the recurrent laryngeal nerve) or by a lesion within or immediately adjacent to the larynx. No attempt will be made to discuss all of the various causes for hoarseness. The primary purpose here is to emphasize the importance of any hoarseness which is progressive or persistent. It is true that most chronic and persistent hoarseness will prove to be a benign basis. However, due to the frequency of cigarette smoking, with its attendant chronic irritation of the larynx, consideration must be given to the possibility of neoplasia. The most common site for neoplasia is the anterior vocal cord. Since the cord proper is usually involved, hoarseness is frequently an early development. In any aircrew member with hoarseness that persists longer than can reasonably be explained by an acute catarrhal laryngitis (2-3 weeks) carcinoma of the larynx should be suspected, particularly if the individual is a cigarette smoker, and should be promptly referred to an otolaryngologist for examination of the larynx (which must include adequate visualization of the anterior commissure).

The most frequent cause of injury to the larynx is blunt trauma to the anterior neck area. The specific injury may consist of hematoma formation, mucosal lacerations, fractures of the thyroid and cricoid cartilages, and dislocation of the cricothyroid or cricoarytenoid joint. Frequently, more than one type of injury will be present.

Any patient who has received trauma to the anterior neck area should be suspected of having injury to the larynx. There may or may not be symptoms which suggest this. Any change in the patient's voice or any alteration in the airway (particularly increasing airway obstruction) should strongly suggest injury to the larynx. The patient with a laryngeal injury may also mention pain in the neck, difficulty swallowing and cough (sometimes blood-tinged). Examination may reveal an obvious deformity of the neck, the presence of subcutaneous emphysema, and laryngeal tenderness.

Initial management of the patient with a laryngeal injury requires maintaining an adequate airway, which can require a tracheostomy once a cervical spine injury is ruled out. Direct laryngoscopy will usually be required to determine the presence of edema, hematoma, mucosal tear, cartilage displacement and vocal cord paralysis.

Definitive management of laryngeal injuries is the province of the otolaryngologist; however, since the flight surgeon may be the first physician to see such a victim of trauma, it is imperative that the possibility of injury to the larynx be considered and the patient be referred if an injury is at all likely. Unrecognized and untreated injuries frequently result in stenosis of the larynx, an extremely serious complication which is very difficult to manage. Amazingly, minimal injuries can
result in stenosis if proper treatment is not carried out. This complication is largely preventable provided the possibility of injury to the larynx is considered.

**Facial Nerve Paralysis**

*Facial nerve palsy (Bell's palsy)* is an idiopathic paralysis of the facial nerve and is generally unilateral. Historical findings may include an antecedent viral infection or severe emotional shock. Symptoms may include a loss of taste along with the inability to whistle and smile. Vertigo may be a transient occurrence. The severity of pain may correlate with the severity of paralysis. The pathogenesis is believed to be edema of the nerve due to vasospasm or viral neuronitis. Treatment modalities vary and may depend on the severity of the symptoms. Incomplete paralysis without pain will usually have a complete recovery in 7 to 14 days. Individuals with complete paralysis may be placed on corticosteroids. Those who do not respond to two courses of steroid therapy or in whom steroids are contraindicated may need surgical decompression. Artificial tear solutions should be provided and the individual instructed to patch the involved eye at night.

Approximately 86% of patients without signs of degeneration will have a full recovery, 56% of those with complete degeneration will have residua. Nerve conduction studies can be helpful in this instance. A constellation involving hearing loss, vertigo, and trigeminal involvement should raise the level of suspicion and CAT scans should be performed to rule out a cerebello-pontine angle tumor or facial nerve neuroma.

The individual should remain grounded until there is evidence of complete recovery. Any permanent residual should be stable and determined not to interfere with performance of flight duties before return to flying.

**EQUIPMENT**

Flight surgeons may find themselves working in places ranging from a major medical facility with all ancillary equipment and all supporting specialties to a small tent in the middle of nowhere. When the former condition exists there is little need to have a well stocked otolaryngologic tray. However, when the latter exists, what you bring is what you have. Compact trays for things such as epistaxis and cricothyroidotomy/tracheostomy should be prepacked and sterilized for easy and quick access. Adequate lighting should always be available. Various sized laryngeal and pharyngeal mirrors should be readily available for physical examination of the patient. A cerumen loop should also be kept nearby whenever examining ears if needed to remove cerumen. Various other equipment may be needed during treatment of office otolaryngologic problems. A minimum of equipment for meeting most contingencies is listed in table 6-2.
### TABLE 6-2. MINIMAL EQUIPMENT FOR ENT EXAMINATION AND TREATMENT UNITS.

<table>
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<th>Amount</th>
<th>Item</th>
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<tr>
<td>1</td>
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<tr>
<td>1</td>
<td>Headband, mirror</td>
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<tr>
<td>1</td>
<td>Mirror, headband</td>
<td>6515-347-5200</td>
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<td></td>
<td>Laryngeal mirrors</td>
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<td>4</td>
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<td>6</td>
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<td>2</td>
<td>Kelly curved forceps</td>
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<td>2 pkg.</td>
<td>4 x 4 gauze pads</td>
<td>6510-203-8448</td>
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<td>4</td>
<td>Nasal applicators, Turnbull</td>
<td>6515-303-3600</td>
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<td>6</td>
<td>Frazier suction tips 7 or 8 Fr. (nose)</td>
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<td>4</td>
<td>Bayonet forceps</td>
<td>6515-333-6600</td>
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<td>1</td>
<td>Knight nasal forceps</td>
<td>6515-336-3300</td>
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<td>Nasal speculum (Vienna)</td>
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<td>Atomizer set, medicinal</td>
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<td>1</td>
<td>Vaporizer, medicinal (Politzer)</td>
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<td>Dental syringes</td>
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<td>Intubation tubes</td>
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<td>1 can</td>
<td>2% Lidocaine with Epinephrine carpules</td>
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<td>Tuning fork set</td>
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<td>Toynbee tubing</td>
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<td>Ear speculum set, Gruber</td>
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<tr>
<td>1</td>
<td>Ear basin, Goldnamer</td>
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<tr>
<td>1</td>
<td>Pneumatic otoscope set</td>
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</tr>
<tr>
<td>2</td>
<td>Billeau loop (small &amp; medium)</td>
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<tr>
<td>1 dz.</td>
<td>Applicator, Turnbull 5 in</td>
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<tr>
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<td>Hook and spoon, ear, gross</td>
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<tr>
<td>1</td>
<td>DeVilbiss syringe</td>
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</tr>
<tr>
<td></td>
<td>Disposable spinal needles</td>
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6-30
<table>
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<th>Item Description</th>
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</thead>
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<td>Baron ear suction 5 Fr.</td>
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<td>Ear forceps, alligator, jaw size 2 x 8 mm</td>
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<tr>
<td>Buck ear curette, sizes 1 and 2</td>
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<tr>
<td>Neosynephrine, 1/2 %</td>
</tr>
<tr>
<td>Ear syringe, Reiner-Alexander 4 oz</td>
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</tbody>
</table>
REFERENCES

1. AFI 48-123. Medical Examination and Standards. 1994.


