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The effect of flight on the ear and hearing: a critical review of literature

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Thesis

THE EFFECT OF FLIGHT ON THE EAR AND HEARING:
A CRITICAL REVIEW OF LITERATURE

Submitted by

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(A.B., Cornell University, 1946)

In Partial Fulfillment of Requirements for
the Degree of Master of Education

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CHAPTER I
INTRODUCTION

On December 17, 1903 on the bleak sand dunes of Kitty Hawk, North Carolina, the Wright brothers achieved man's first powered flight. Since that day, the world has witnessed the tremendous technological growth of the aircraft industry, the increasing power and speed of aircraft engines, the shrinking of the earth's vast distances, the impact of the air age on the economic and social growth of underdeveloped areas and the yearly phenomenal increase of the number of passenger air-miles flown.

The air age has brought great benefits to mankind. However, air travel has also created many deleterious effects of flight upon the ear and hearing. They are the most frequent causes of discomfort among air passengers and pilots suffer more from occupational disturbances of the ear than from all other occupational diseases combined.^{1/} People whose ears are easily susceptible to damage from noise may incur permanent damage to their hearing from even brief exposures to aircraft-engine noise.^{2/}

^{1/}H. G. Armstrong and J. W. Heim, "The Effect of Flight on the Middle Ear," Journal of the American Medical Association, (1937), 109: 417-421.

^{2/}Hayes A. Newby, Audiology, Appleton-Century Crofts, Inc., New York, 1958, p. 50.

Statement of the problem.-- It was the purpose of this study to define the areas of past and present research on the effects of flight on the ear and hearing and to report and summarize the findings of such research.

Justification.-- A considerable amount of literature has been published based on research activities which grew out of World War II on the effects of flight upon the hearing mechanism. Two well-defined areas of research have been (1) the condition known as aerotitis media affecting the middle ear and caused by changes in barometric pressure during flight, and (2) acoustic trauma or noise-induced hearing loss.

With increased commercial passenger air travel the problems of aerotitis media and acoustic trauma are rapidly multiplying. Aerotitis media will be seen increasingly often in the private practice of many otologists, particularly now that jet transportation is an actuality. ^{1/} Newby ^{2/} says:

"The entire matter of the effect of aircraft noise on hearing is justifiably receiving the attention of various experts."

Because of the need for background knowledge of such flight-induced hearing loss may become increasingly important

^{1/}Reed W. Hyde, "Aerotitis Media: A Critical Review," *Annals of Otolaryngology, Rhinology, and Laryngology* (1952), 61: p. 937.

^{2/}Hayes A. Newby, op. cit., p. 50.

to audiologists, speech clinicians and speechreading teachers who are involved with the treatment and rehabilitation of those persons so afflicted, a critical and systematic review of the literature is justified.

Scope. -- The significant literature concerning both aerotitis media and the effects of aircraft-engine noise on the hearing mechanism will be annotated and discussed. Emphasis will be given to those areas of knowledge which are of particular interest to audiologists and speech clinicians.

CHAPTER II

REVIEW OF THE LITERATURE

1. Literature on Aerotitis Media

History.-- The first known recorded case of aerotitis media was made in the year 1789 when one of the world's first aeronauts, a physician named Pilâtre de Rozier, reported severe pain in his right ear resulting from an ascent to 10,500 feet in a Montgolfier balloon.

Little investigation was made into this first deleterious effect of flight until about 100 years later when considerable interest in the effects of pressure changes on the human body was stimulated in Europe with recognition of the occupational hazards of the caisson worker.^{1/} In 1879 in the city of Leipzig an investigator named A. Hartmann published a study entitled "Experimentelle Studien uber die Funktion der Eustachischen Rohre" in which he described the egress of air from the middle ear along a normal eustachian tube.^{2/}

During World War I, aircraft had not yet attained extreme ranges of altitude and rates of descent and therefore the problem of aerotitis media did not attract much attention.

^{1/}Reed W. Hyde, "Aerotitis Media: A Critical Review," Annals of Otology, Rhinology, and Laryngology, (1952), 61: 937-975.

^{2/}J.E.G. McGibbon, "Aviation Pressure Deafness," Journal of Laryngology and Otology, (January, 1942), 57: 14-22.

The interest of aviation otologists was concentrated on the vestibular portion of the ear but it was discovered that pilots who had difficulty ventilating their middle ear were greatly handicapped in their flying and regulations were finally adopted requiring candidates for flying training to have patent eustachian tubes.^{1/}

In 1937 Armstrong and Heim first described the condition as a distinct clinical entity and introduced the term aerotitis media. Their original definition is the one most generally accepted:^{2/}

"Aerotitis media is an acute or chronic traumatic inflammation of the middle ear caused by a pressure difference between the air in the tympanic cavity and that of the surrounding atmosphere. It commonly occurs during changes of altitude in airplane flights and is characterized by congestion, inflammation, discomfort and pain in the middle ear and a temporary impairment of hearing."

The impetus of World War II caused a great number of researchers to investigate the problem. In 1941 Wodak, a Flight Surgeon with the RAF in Palestine, listed otitis media and obstruction of the eustachian tubes as rejecting criteria for prospective pilots.^{3/}

^{1/}H. G. Armstrong, Principles and Practices of Aviation Medicine, Williams & Wilkins Company, Baltimore, 1952, p. 251.

^{2/}H. G. Armstrong and J. W. Heim, "The Effect of Flight on the Middle Ear," Journal of the American Medical Association, (1937), 109: 417-421.

^{3/}E. Wodak, "The Auditory Apparatus and Aviation." Lancet, (1941), 1: 8-9.

In the same year Lamport ^{1/} described the Valsalva and Politzer methods for ventilating the eustachian tube. In 1942 Campbell and Hargreaves, ^{2/} Fenton, ^{3/} Carson ^{4/} and McGibbon ^{5/} were among the leading otologists who conducted experimental studies into the problem of aerotitis media.

The literature concerning aerotitis media refers to it by a variety of names, including, among others, aviation pressure deafness and acute otitic barotrauma. The term more commonly used in this country today is aerotitis media. ^{6/}

Anatomy and physiology of the eustachian tube.-- As was pointed out by Armstrong and Heim in their definitive article published in 1937 the deleterious effects of flight on the middle ear depend partly on the peculiar structure and functioning of the eustachian tube and partly on the presence or absence of pathology in this aural appendage. Their

^{1/}Harold Lamport, "Maneuver for the Relief of Acute Aero-otitis Media," Journal of Aviation Medicine, (1941), 12: 163-168.

^{2/}Paul A. Campbell and J. Hargreaves, "Aviation Deafness - Acute and Chronic," Archives of Otolaryngology, (1940), 32: 417-428.

^{3/}Ralph A. Fenton, "Otology and Aviation," Annals of Otology, Rhinology, and Laryngology, (1942), 51: 333-342.

^{4/}Leon D. Carson, "Otolaryngological Aspects of Aviation," Laryngoscope (1942), 52: 704-715. Discussion, 716-717.

^{5/}J.E.G. McGibbon, "Aviation Pressure Deafness," Journal of Laryngology and Otology, (January, 1942), 57: 14-22.

^{6/}Reed W. Hyde, op. cit., p. 937.

description of the anatomy and physiology of the eustachian tube will be briefly reviewed at this time:^{1/}

"The eustachian tube is a slitlike, potential tube extending from the middle ear to the nasopharynx. It is formed of bone, cartilage and fibrous tissue.

The bony portion begins at the upper part of the anterior wall of the tympanic cavity and, gradually narrowing, passes downward, forward and mediad for about 12 mm., ending at the angle of the junction of the squamous and petrous portions of the temporal bone.

The cartilaginous portion of the tube extends from the bony portion to the nasopharynx. This section is about 24 mm. in length and is formed of a triangular plate of elastic fibrocartilage with its apex attached to the bony portion and its base placed directly under the mucous membrane of the nasopharynx where it forms a prominence, the torus tubarius. The upper edge of the cartilage is bent laterally and takes the form of a hook on cross-section, open below and laterally. These walls of the canal are completed by fibrous tissue.

The lumen of the eustachian tube is narrowest at the junction of the bony and cartilaginous portions, the isthmus and expanding rapidly in both directions reaches its largest diameter at the pharyngeal orifice. At rest the lumen of the cartilaginous portion of the tube is a vertical slit with its walls opposed.

The mucous membrane of the eustachian tube is a direct extension of that of the nasopharynx and continues backward to line the middle ear completely. The mucous membrane of the bony portion of the tube is thin but in the cartilaginous portion it is thick and very vascular, contains numerous mucous glands and is composed of ciliated columnar cells. Near the mouth of the eustachian tube is a variable amount of adenoid tissue known as Gerlach's or the tubal tonsil. The pharyngeal ostium of the eustachian tube is located high up on the lateral wall of the nasopharynx. This opening is triangular, bounded behind by the torus tubarius and in front by the nasal cavity."

^{1/}H. G. Armstrong and J. W. Heim, op. cit., pp. 417-421.

The most important function of the eustachian tube is ventilation of the middle ear, whereby the pressure on the two sides of the tympanic cavity are equalized.^{1/} The ears and sinuses are gas - or air - containing cavities and the eustachian tube acts both as a ventilating shaft and as a means of drainage for the middle ear. In the resting state, the walls of the eustachian tube are collapsed, but during swallowing or yawning the tube opens and therefore the air pressure on both sides of the tympanic membrane is more or less constantly equalized.^{2/}

In discussing the special physiology of the eustachian tube in aircraft flights with their attendant changes of altitude and atmospheric pressure Armstrong says:^{3/}

"At somewhere between 110 and 180 feet altitude (3 to 5 mm. Hg) there is a slight sensation of fullness in the middle ear and examination will show the tympanic membranes to be slightly bulging. This bulging and the sensation of fullness increase with the decrease of atmospheric pressure until at an average of 500 feet altitude (15 mm. Hg.) there is a sudden annoying "click" in the middle ear as the tympanic membrane snaps back to its normal position. When this occurs it indicates that the eustachian tube has been forced open by the excess pressure in tympanic cavity and that the excess pressure in the ear has been relieved by a sudden rush of air from the ear to the nasopharynx."

^{1/}Reed W. Hyde, op. cit., p. 941.

^{2/}Ross A. McFarland, Human Factors in Air Transportation, McGraw-Hill Book Company, New York, 1953, p. 678.

^{3/}H. G. Armstrong, op. cit., pp. 254-255.

McFarland reported that this sudden release of air is frequently experienced by airmen and passengers and that the process of the building up and release of pressure in the middle ear may be repeated many times during slow ascents.^{1/}

The motion of the cilia and the flutter-valve-like eustachian tubes favor the motion of material from the ear to the nasopharynx and opposes motion in the opposite direction.^{2/} The acts of swallowing and yawning open the tubes easily on ascent and therefore airmen and passengers experience little or no difficulty during this time.

During ascent the eustachian tube is said to open at more or less equal altitude intervals. According to Armstrong^{3/} and Fenton,^{4/} at elevations above 500 feet the tubes open at approximately 425 feet intervals up to 35,000 feet when the air pressure is only 190 mm. Hg.

Therefore the physiology of the eustachian tube functions well on ascent. When the outside pressure decreases, the higher pressure in the ear cavity causes air to escape from the middle ear through the eustachian tube to the nasopharynx easily and automatically.

^{1/}Ross A. McFarland, Human Factors in Air Transport Design, McGraw-Hill Book Company, New York, 1946, p. 54.

^{2/}H. G. Armstrong, op. cit., p. 255.

^{3/}H. G. Armstrong, op. cit., p. 255.

^{4/}Fenton, op. cit., pp. 333-342.

During descent, however, a totally different situation exists and most cases of aerotitis media occur at this time. The ear does not adjust automatically to the increase of air pressure differential. The flutter-valve-like action of the eustachian tube permits the air to pass easily in one direction only, i.e. from the middle ear to the back of the throat. Even a normal eustachian tube remains closed and air does not enter the middle ear unless aided by muscular action or therapeutic inflation. Consequently, a positive pressure develops on the outside of the eardrum and pushes it inward. According to McFarland this results in a relatively greater external pressure being exerted on the eardrum, resulting in decreased auditory acuity, ^{1/} tinnitus, pain, and in extreme cases, rupture of the drum.

Methods of autoinflation such as yawning and swallowing may be used to overcome the negative pressure in the middle ear. However, Armstrong and Heim state that if a negative pressure of 80 to 90 mm. Hg is allowed to develop in the middle ear, the eustachian tube "locks" for it is impossible for the eustachian muscles to exert enough force to open it.^{2/}

Immediate relief of this locking effect can only be obtained by ascending to a higher level. It is also more likely to

^{1/}McFarland, Human Factors in Air Transportation, op. cit., p. 168.

^{2/}H.G. Armstrong and J. W. Heim, op. cit., pp. 417-421.

develop as the rate of recompression is increased.

Since the pressure differential built up in the middle ear depends not only upon the amount of descent but also upon the altitude at which this descent occurs,^{1/} airlines have instructed their pilots to avoid rapid changes of ascent and descent. However, Armstrong^{2/} reported that:

"The restrictions on the rate of aircraft ascent and descent has, in many instances, caused airliners to be operated inefficiently as, for example, when a landing field is in a valley just beyond a range of mountains. In this case instead of gliding directly down to the field it is necessary for the pilot to descend gradually over a long period of time and thus waste both time and fuel. During descent through icing conditions or rough air the airline pilot has to choose between a slow change of altitude with a consequent risk of crashing in the first instance or airsickness among his passengers in the second or a rapid rate of descent with a total disregard for the ear problem."

Commercial aircraft normally descend from 300 to 500 feet per minute. Since the average person swallows involuntarily once a minute, Armstrong states that a rate of climb or descent of 200 feet per minute will usually cause no discomfort, 500 feet per minute slight discomfort and 1000 feet per minute moderate discomfort even though the person makes no voluntary effort to clear his ears.^{3/}

^{1/}Reed W. Hyde, op. cit., p. 943.

^{2/}H. G. Armstrong, op. cit., p. 252.

^{3/}Ibid., p. 263.

Another problem appears to be that of the sleeping passenger who must be awakened every time the plane descends from any considerable altitude in order that his middle ear may not be traumatized. This has proved a most successful measure but one of considerable annoyance to the passenger whose sleep is constantly interrupted.^{1/}

Infants are no more susceptible than adults, but they should be awakened from sleep during descent and given a bottle to nurse or food to eat. If they cry, the pressure in the ears is equalized and there is little or no tendency to develop aerotitis media.^{2/}

The pressurization of modern aircraft has done a lot to reduce the incidence of aerotitis media. Cabin pressure is maintained at 4,000 to 6,000 feet under normal cruising conditions and may be higher when flying at storm-evading level. However, the pressure differential between 8,000 feet and sea level (about 200 mm. Hg) is still enough to cause considerable discomfort.^{3/} McFarland^{4/} agrees that the regulation of pressure changes in supercharged cabins and of rates of descent in unpressurized aircraft will decrease the

^{1/}Ibid., p. 252.

^{2/}Ross A. McFarland, op. cit., p. 679, citing R. L. Fruin, "Aero-otitis Media in Infants," Journal of Aviation Medicine (1950), 21: 150.

^{3/}Reed W. Hyde, op. cit., p. 963.

^{4/}Ross A. McFarland, op. cit., p. 682.

frequency and severity of aerotitis media but will not eliminate all the problems. He states that:

"In planes with pressurized cabins, the practice is to maintain cabin altitudes of 8,000 feet altitude or less while in flight at 20,000 feet. Thus, unless the rate of pressure change in the cabin is kept within safe limits, i.e., a rate of change of 0.1 pounds per square inch/minute, the pressurized cabin will offer only partial relief."

Flight attendants have been instructed to encourage passengers to swallow just before or during the initial stages of descent in order to ventilate the middle ear. However, if the passenger experiences blockage, methods of autoinflation such as grinding the lower jaw or the valsalva maneuver in which the nose and mouth are held shut while air is expelled forcefully through the nose should be explained.

Spealman and Cherry ^{1/} conducted experiments simulating descent from an altitude using subjects who were instructed not to swallow or make other movements that might ventilate the middle ear. They found that:

1. Pressure changes just perceptible to the middle ear were greater at very slow rates of descent than at the more rapid rates.
2. Threshold pressure changes for persistent middle ear pain were about 100 mm. Hg in the two subjects studied irrespective of the rate of descent or the altitude at which the descent was started.

^{1/}C. R. Spealman and J. C. Cherry, "Middle Ear Perception of Pressure and Pain in Descent from Altitude," Journal of Aviation Medicine, (1958), 29: 106-110.

The ability to equalize pressure is blocked by inflammation due to colds, sore throats, or local infections in the upper respiratory tract. During such affections the eustachian tubes become temporarily stenosed. McFarland^{1/} feels that those with upper respiratory infections should delay their flight. If they cannot then a vasoconstrictor should be applied over the orifice of the eustachian tube by dropping a few drops of the material into one nostril while the head is tilted to the opposite side.

Etiology.-- Since aerotitis media occurs as a direct result of failure to ventilate the middle ear and thus equalize the pressure differential between the middle ear and the atmosphere, failure to open the tubes or inability to do so must be considered important etiological factors.

Armstrong and Heim^{2/} stated that failure to open the eustachian tubes is due often to ignorance on the part of inexperienced pilots or passengers but may be due to carelessness or to being asleep; or may arise from the influence of analgesics, anesthetics or from coma on ambulance airplanes.

Some of the more frequent causes of inability to ventilate the middle ear are (1) acute and chronic infections of the upper respiratory tract; (2) nasal obstructions; (3) sinusitis; (4) tonsillitis; (5) tumors and growths of the nose and

^{1/}Ross A. McFarland, op. cit., p. 681.

^{2/}H. G. Armstrong and J. W. Heim, op. cit., pp. 417-421.

nasopharynx; (6) paralysis of the soft palate or superior pharyngeal muscles; (7) enlargement of the pharyngeal or tubal tonsil; (8) inflammatory conditions of the eustachian tube following adenectomy and (9) malposition of the jaws.^{1/}

Armstrong^{2/} is of the opinion that of all the conditions which produce a temporary stenosis of the eustachian tube, and secondarily an aerotitis media, upper respiratory infections are by far the worst offenders. The reasons for this are that upper respiratory infections occur quite frequently in the average person and even those infections of every minor nature, such as a common cold or a slight sore throat, are sufficient to prevent adequate ventilation of the middle ears during aircraft descent.

McFarland^{3/} considered important etiological factors to be inflammation, edema, or hypertrophy of the eustachian tube or surrounding structures.

Willhelmy^{4/} attributed malposition of the jaws as a causative factor of inability to open the eustachian tubes and advised repositioning of the mandible in such cases.

1/H. G. Armstrong, op. cit., p. 256.

2/H. G. Armstrong, op. cit., p. 256.

3/Ross A. McFarland, op. cit., p. 680.

4/G. E. Willhelmy, "Ear Symptoms Incidental to Sudden Altitude Changes and the Factor of Overclosure of the Mandible," Journal of Aviation Medicine, (1936), 7: 177-181.

Shilling ^{1/} conducted a study of aerotitis media among submarine personnel and chose fifty cases with interference in mandibular function for dental treatment. Following treatment, forty-six cases came through with absolutely normal ears.

Carson ^{2/} and Poppen ^{3/} both stated that a partial vacuum within the middle ear causes engorgement of the capillaries lining the cavity followed by extravasation and actual hemorrhage.

Pothoven and Schuringa ^{4/} were of the opinion that the ossicular chain does not operate properly because of a temporary lower or higher pressure in the middle ear as a result of descending and climbing. This often results in a slight edema with a swelling of the submucous tissue in the middle ear which condition may lead to a permanent conductive deafness.

Campbell and Hargreaves ^{5/} attributed physiologic or pathologic failure of the middle ear to be ventilated properly

1/C. W. Shilling et al, "Aerotitis Media: A Brief Presentation of Its Symptomatology, Prevention and Treatment," Journal of Aviation Medicine, (1947), 18: 48-57.

2/Leon D. Carson, op. cit., pp. 417-428.

3/J. R. Poppen, "The Ear in Flying," Laryngoscope (1941), 51: 974-982.

4/W. J. Pothoven and A. Schuringa, "Aviation Noise Deafness, Hearing Standards and Recruitment," Journal of Aviation Medicine (1948), 19: 380-388.

5/Paul A. Campbell and J. Hargreaves, op. cit., pp. 417-428.

during altitudinal changes in pressure. This ventilation failure leads to acute or chronic changes in the middle ear with the usual picture of conduction deafness.

Incidence.-- Schilling et al.^{1/} examined over 1,000 men per month and found that 25% developed aerotitis media.

During World War II, aerotitis media was encountered in about 11 to 17% of the subjects.^{2/} The incidence was reduced by eliminating subjects with colds or those who developed ear trouble, by using a controlled and constant rate of descent, or by leveling off and reascending when a subject developed painful symptoms.

Stewart^{3/} conducted an extensive investigation of aerotitis media in low-pressure-chamber tests. Approximately 50% of 24,000 RCAF men studied showed reddening of the eardrum. The incidence of aerotitis media varied from 8 to 21% during a 12-month period.

Symptoms.-- Campbell^{4/} noted that changes in altitude resulting in a barometric pressure change of 100 mm. Hg (about

^{1/}C. W. Shilling et al., op. cit., pp. 48-57.

^{2/}Ross A. McFarland, op. cit., p. 680.

^{3/}C. B. Stewart et al., "Acute Otitic Barotrauma Resulting from Low-pressure Chamber Tests," Journal of Aviation Medicine (1945), 16: 385-408.

^{4/}Paul A. Campbell, "Otolaryngological Problems of Aviation in World War II," Annals of Otology, Rhinology, and Laryngology, Chicago, (1945), 51: 293-300.

4,000 feet at near ground levels) can produce a fluctuation of several decibels of hearing in the conversational frequency range between the acts of swallowing. When the negative pressure inside the middle ear reaches 100-120 mm. Hg, the pain gets suddenly severe and 10 or more decibels (db) of hearing are lost, usually from 128-2048 cycles per second (cps).

Fenton ^{1/} also found that repeated exposure to barometric changes will lead to a chronic thickening of the tympanic and eustachian tube linings with eventual formation of connective tissue and considerable reduction of hearing for low tones.

Kos ^{2/} found that persistent aviation pressure deafness associated with tubal obstruction was due to pathologic processes which were prolonged after the precipitating factor and initiating causes ceased to exist. Vital changes in the mucosa of the middle ear and eustachian tube had taken place. If therapy is energetic, the persistent hearing impairment may be reversible; however, if treatment is neglected or unsuccessful, the impairment may be permanent.

Shilling et al. ^{3/} defined the disease as a pathological condition of the middle ear and drum membrane ranging in

1/Ralph A. Fenton, op. cit., pp. 333-342.

2/C. M. Kos, "Effects of Barometric Pressure Changes on Hearing," Archives of Otology, Chicago (1945), 41: 322-326.

3/ C. W. Shilling et al., op. cit., pp. 48-57.

severity from slight erythema of the drum membranes to frank hemorrhage into the middle ear or rupture of the drum which is caused by a difference between air pressure in the middle ear and that in the surrounding atmosphere. They found symptomatology of pain or a feeling of fullness or pressure in the ear, loss of auditory acuity and rarely bleeding from the external canal or spitting of blood. Although auditory acuity is one of the commoner complaints, there is a relatively low percentage of men with loss of any significant degree. With the use of a number of control tests, it was found that the loss is related to the presence of fluid and lack of air in the middle ear. Excess lymphoid tissue in and around the pharyngeal opening of the eustachian tube is significant for disease prediction whereas nasal septum defects, hypertrophoid tonsils and adenoids were non-significant in disease predilection.

Fenton ^{1/} similarly to Shilling ^{2/} noted severe pain, local congestion especially of the drum membrane, transudation of hemorrhage into the middle ear, and low tone deafness of varying degree.

McGibbon ^{3/} found that of one hundred patients: 31 had deafness alone, 55 had deafness and pain, 5 had deafness and

1/Ralph A. Fenton, op. cit., pp. 333-342.

2/C. W. Shilling et al., op. cit., pp. 48-57.

3/J. E. G. McGibbon, op. cit., pp. 14-22.

tinnitus, 3 had deafness and vertigo, and 3 had pain alone.

Treatment.-- Shilling et al.^{1/} felt that no treatment is best in most cases because of the danger of secondary infection. Use of radium applied to the pharyngeal opening of the eustachian tube is the most successful therapy. Fifty milligrams of radium is applied to each side of the nasopharynx for six to ten minutes. This therapy is given for three to eight treatments at monthly intervals. The restoration of normal unstrained activity to muscles attached to the pterygomandibular raphe by dental corrective measures is another form of therapy.

Kos^{2/} advocated the following types of therapy: (1) instillation of a nasal astringent, (2) aspiration of excess secretions, and (3) frequent tubal inflation during the first few hours.

Fenton^{3/} believed that catheterization of Politzerization gives rapid relief, but precautions are necessary to avoid infection of the sterile transudate in the tympanic cavity.

^{1/}C. W. Shilling et al., op. cit., pp. 48-57.

^{2/}C. M. Kos, op. cit., pp. 322-326.

^{3/}Ralph A. Fenton, op. cit., pp. 333-342.

2. Literature on the Effects of Aircraft Noise

The increasing power of aircraft engines has extended the range of man's mobility but has also added to the total amount of noise which surrounds him. Otologists have been increasingly concerned with the effects of the intensity of aircraft noise on the hearing mechanism. If unrecognized and undetected, noise hazards can cause permanent nerve deafness.

In the early days of air transportation, noise levels were very great and passengers experienced considerable discomfort. They were able to converse with each other only by shouting and were bothered with transitory deafness following a flight. Recent years have seen improvements in power plant design and insulation of the cabins has greatly reduced discomfort from this source.^{1/}

Until recently, the jet engine noise problem was associated principally with military air bases and experimental airfields of plane manufacturers. However, since commercial aviation has entered the jet age, noise will effect many other persons: travelers, crew members, and ground personnel. Several million persons living near airports will notice the difference between noise of jet aircrafts and that of conven-

^{1/}Ross A. McFarland, op. cit., p. 699.

tional propellers. Although they may complain, they will not be deaf, since the sound intensity will be lower than the hazard levels.^{1/}

Sources and types of noise.--- The analysis of aircraft noise as a cause of aviation noise deafness is important in various respects. The sound pressure of the higher frequencies is responsible for the damage done to the hearing function. The intelligibility of speech and intercommunication depends largely on the amount of high frequencies.^{2/}

Dickson^{3/} stated that aviation noises are chiefly those from engine explosions, propeller hum, sounds produced by wind and slip-stream effects on the structure and sounds from moving parts. The intensity level of such noises is in the region of 120-130 db.

1/Editorial: "Noise of Jet Engines May Be a Hazard," Journal of the American Medical Association (April, 1959), 169: 1629.

2/W. J. Pothoven, "Some Audiological Aspects of Aircraft Noise," Journal of Aviation Medicine (1950), 21: 140-146.

3/E. D. D. Dickson, "Aviation Noise Deafness and Its Prevention," Journal of Laryngology and Otology, (1952), 57: 8-10.

Poppen ^{1/} found that there was a preponderance of noise in piston aircraft due to propeller tips, explosion cycle of the motor and operation of gears. The sound is of low pitch but of high intensity. High-pitched sounds are those such as whistling through leaking joints in hoods, ventilation systems, etc.

Armstrong ^{2/} noted that the principle sources of jet aircraft noise are from the air intake, the jet exhaust, the slipstream and the turbine wheel. The first three produce a "whooshing" noise while the latter is characterized by a high-pitched whine. The intensities of these noises are subjectively very deceptive for in flight almost everyone feels that the environment is almost perfectly quiet and are amazed to find that they cannot hear the loudest shout or even their own voices.

Tonndorf ^{3/} discovered still another source of noise referred to as atmospheric static consisting of repeated bursts of noise and covering a wide frequency range similar to those of the jet engine and the slipstream noises. Static differs in that successive bursts both vary greatly in intensity and appear at random levels. Further, the absolute

^{1/}J. R. Poppen, "The Ear in Flying," Laryngoscope (1941), 51: 974-982.

^{2/}H. G. Armstrong, op. cit., p. 275.

^{3/}Juergen Tonndorf, "Auditory Perception in Noise," Journal of Aviation Medicine (1951), 22: 491-500; 529.

intensity of all static bursts is defined by the setting of the radio receiver volume control. Thus the average intensity of static is linked to the intensity at which the signal is presented.

In McFarland's study ^{1/} it was noted that irritating qualities of noise are frequently those high-frequency sounds originating from loose accessory equipment such as ash trays, gallery utensils, metal trim, fresh air ventilators, and pressurization leaks.

Not frequently considered are the important noise sources of the helicopter. Berry and Eastward ^{2/} list the engine, rotor blades, and the motor transmission assemblies as the important noise sources.

Sound spectra of piston and jet engines.-- Among modern aircraft two characteristic noise spectra can be distinguished, that of the conventional-powered and that of the jet-powered airplanes.

Parrack and Eldredge ^{3/} in their study of aircraft noise were of the opinion that propeller engines generate most of

1/Ross A. McFarland, op. cit., p. 701.

2/Charles A. Berry and Herbert K. Eastwood, "Helicopter Problems: Noise, Cockpit Contamination and Disorientation," Aerospace Medicine (1960), 31: 179-190.

3/H. O. Parrack and D. H. Eldredge, "Noise Problems Associated with Aircraft Maintenance," Journal of Aviation Medicine (1951), 22: 470-476.

their sound energy in the narrow frequency band from 75-150 cps. Above this band, relatively little sound energy is produced at the frequencies which are most sensitive to the ear and important for speech since the sound pressure as a function of frequency drops rapidly.

Pothoven ^{1/} also agreed that propeller aircraft shows a peak usually between 50-150 ops. His study revealed that a 3-blade propeller rotating at 2,000 revolutions per minute (rpm) will have a fundamental tone of 100 cps with overtones of 200, 400, 600 cps and so on.

Tonndorf ^{2/} reported that a propeller-type airplane has two components: (1) a low-frequency, discontinuous harmonic line spectrum produced by the engine, propeller, and exhaust, and (2) a continuous band spectrum, similar to that of the jet, which is produced by the slip-stream. The first of these components usually far exceeds the second in intensity so it is the low-frequency portion which defines the over-all noise level.

1/W. J. Pothoven, op. cit., pp. 140-146.

2/Juergen Tonndorf, op. cit., p. 492.

According to Armstrong,^{1/} the B-25 is the noisiest of all aircraft. The over-all intensity of the B-25 noise spectrum is 117 db. The over-all level of the B-25 is determined principally by the frequencies below the 300 to 600 cps band since the intensity levels of the higher frequencies are more than 10 db below that of the peak. The outstanding element in the B-25 spectrum is the low-frequency component which is intense enough to be felt as vibrations.

Berry and Eastward's study of military helicopters^{2/} revealed that as in most reciprocating engine aircraft, the peak levels are in the 110-120 db range in the low frequency bands (below 300-600 cps) with the remainder ranging from 110 db at the 300-600 cps band to 88 db at the 4800-9600 cps band. An exception is the H-37 helicopter which has peaks of 110 db in the 600-1200, 1200-2400, and 2400-4800 bands.

Helicopters are noisy aircrafts. During warm-ups the over-all noise level of the H-19 helicopter at the fire-guard's station to the left of the aircraft varied from 118-125 db. The overall levels at the co-pilot's head with

^{1/}H. G. Armstrong, op. cit., p. 268.

^{2/}Charles A. Berry and Herbert K. Eastwood, op. cit., p. 180.

the door opened ranged from 112-114 db. Closing the door reduced these levels to 110-112 db.^{1/}

Lederer^{2/} measured the over-all noise levels of commercial aircraft and recorded that the Convairliner 340 and 440 reached a peak of over 100 db with two engines under cruise conditions.

Parrack and Eldredge^{3/} reported that in general, jet engines produce sound energy at high levels throughout the frequency range of the human ear. The lower frequencies tend to have the highest level with the sound energy distributed fairly evenly from about 200 cps up to 1,000 or 1,500 cps. The jet engine compressor acts as a siren and produces a high sound pressure level, which is often quite annoying and may be particularly obnoxious from the point of view of hearing, at some particular frequency, usually between 2,000 and 5,000 cps. Above this frequency the sound pressure as a function of frequency decreases up to about 30,000 cps, where it is below the electrical noise level of measuring instruments.

^{1/}Charles A. Berry and Herbert K. Eastwood, op. cit., p. 180.

^{2/}Ludwig Lederer, "The Aeromedical Aspects of Turbo-prop Commercial Aircraft," Journal of Aviation Medicine (1956), 27: 287-300.

^{3/}H. O. Parrack and D. H. Eldredge, op. cit., pp. 470-476.

Propeller noise energy, concentrated at frequencies below 150 cps, tends to be less harmful at levels of 100-110 db than is jet noise of the same level but at somewhat higher frequencies.^{1/}

The cockpit sound spectrum of an Air Force F-80 jet has, like the B-25, an over-all intensity of about 117 db. However, in the F-80 all but two frequency bands are within 10 db of each other so that most of the spectrum contributes to the noise level and no peak frequency stands out. The highest portion of the F-80 noise is at the same range as that for speech. This masks the unprotected ear so thoroughly that the hearing of speech is impossible.^{2/}

The over-all sound levels around an F-84 jet aircraft at idle rpm and at take-off were measured by Parrack and Eldredge.^{3/} At 20 feet from the aircraft, they found the noise level to be 85 db. The authors considered the level of hearing safety to be no more than 85 db for frequencies from 150 cps up to 10,000 cps. Below 150 cps, they felt that one can tolerate sound levels as high as 95 or 100 db.

^{1/}H. O. Parrack and D. H. Eldredge, op. cit., p. 471

^{2/}H. G. Armstrong, op. cit., p. 270.

^{3/}H. O. Parrack and D. H. Eldredge, op. cit., p. 471.

At take-off of the F-84 there was a notable increase in the sound level. At 20 feet from the aircraft the sound level was 144 db, at 80 feet away the sound level was 132 db. The area in which the sound level was 100 db or more became very large. Its boundaries extended roughly half a mile in the rear quadrant of the aircraft.^{1/} Parrack and Eldredge concluded that the sound field generated by a turbojet engine averages 120 db throughout the frequency range from 1,000 to 12,000 cps.

Lederer's^{2/} study of turbo-prop commercial aircraft showed that the Viscount had a peak noise level between 75-150 cps octave bands of 93 db in the cockpit, 101 db at the prop-line level, 90 db in the rear cabin and 88 db in the anterior cabin under cruise conditions.

Rüedi and Furrer^{3/} measured sound pressure levels on stationary "Vampire" jets at idling (3,000 rpm) and at full speed (10,700 rpm). Near the aspiration hole at the front of the plane they found the sound pressure level to be 136 db at the frequency of 850 cps. This was discovered to be 30 db above the general noise spectrum produced by the siren effect of the 17 compressor blades. On the side of the plane to the rear findings were the same except for an additional component

^{1/}H. O. Parrack and D. H. Eldredge, op. cit., p. 472.

^{2/}Ludwig Lederer, op. cit., pp. 287-300.

^{3/}L. Rüedi and W. Furrer, "Special Kind of Acoustic Trauma Produced by Jet Engines," Archives of Otolaryngology, Chicago (1951), 54: 534-541.

of 4,150 cps due to the 83 turbine blades which was 20 db above the general noise level. In the pilot's cabin the special frequencies were only 20 db above the general noise level.

The noise problems of jet-engine operations aboard aircraft carriers were studied by Christy ^{1/} in tests performed aboard the carriers USS Roosevelt, USS Coral Sea, and the USS Midway. Aboard the USS Coral Sea, sound pressure levels of 135-140 db were measured in the catapult areas and aft of the fuselage of the "Banshee" jet aircraft. Sound levels of more than 130 db were measured during the full power turn-up of one Banshee. Communication between personnel was impossible unless it was visual. Earplugs did not afford sufficient protection and light-weight Clark helmets proved to be more effective.

With increasing passenger jet travel, many people will be exposed to the noise generated by a commercial jet airliner during warm-up and take-off operations at airports. In a study of airports and jet noise conducted by Miller, Beranek and Kryter ^{2/} it was found that the noise level 100 feet away from the center of a 4-jet engine array during warm-ups and ramp

^{1/}R. L. Christy, "Jet-engine and Other Noise Problems Aboard Aircraft Carriers," Journal of Aviation Medicine (1954), 25: 485-491.

^{2/}L. N. Miller, L. L. Beranek, and K. D. Kryter, "Airports and Jet Noise," Noise Control (1959), 5: 24-31.

operations may be as much as 110 db. The exhaust from a 4-jet airliner alone produces a noise level of 120 db in the 150-300 cps band.

With a "safe" noise level considered to be no more than 85 db, prolonged exposure to such aircraft noise spectra as described above can cause serious injury to the unprotected ear. Initiation of a noise exposure control program and hearing tests for those exposed to this noise has been suggested by many otologists and others concerned with hearing conservation.

Effect of prolonged exposure: aviation deafness.-- The biological effects of high intensity sound have been the subject of a large number of studies in recent years, most numerous are those relating to acoustic trauma. The questions to be considered were: (1) adequate quantification of noise exposure in terms of spectrum of noise, (2) intensity, and time necessary to produce what degree of hearing loss.^{1/}

Only when these variables can be accurately assessed will it be possible to judge to what extent hearing can be preserved against the insult of modern noise-producers, how much acoustic trauma must be accepted as the price of operating noise-making machinery, and how much compensation is proper

^{1/}Harlow W. Ades, et al, "Threshold of Aural Pain to High-Intensity Sound," Aerospace Medicine (1959), 30: 678-684.

for such acoustic trauma.^{1/}

One of the earliest articles to appear in the literature on the effects of vibration and noise on the human ear was written by F. Koelch in 1935.^{2/} He noted that the workers in German shoe factories and air-drill operators suffered from angioneurosis of the extremities close to the points of trauma; blanching; numbness; palsy; and a neuromuscular asthenia.

C. C. Bunch^{3/} in 1937 demonstrated that deafness produced by noise is a nerve deafness and is most marked immediately after excessive stimulation, followed by a variable period of recovery and an audiogram that would reveal its typical characteristic sharp loss in the frequency area of 4096 cps.

In 1938 Firestone^{4/} recorded audiograms of 109 pilots and found great losses in their bone conduction. He compared this with the similar type audiograms of otosclerotics and interpreted them as effects of vibration of the aircraft. He concluded that (1) noise and vibration incidental to flying appear to be injurious to the ear, and (2) the amount and extent of the injury would be proportional to the flying time.

1/Harlow W. Ades, et al., op. cit., pp. 678-684.

2/C. Firestone, "Bone Conduction in the Experienced Pilot and Probable Interpretation," Laryngoscope (March, 1938), 48: 168-175.

3/C. C. Bunch, Symposium: Neural Mechanism of Hearing; "Nerve Deafness of Known Pathology or Etiology," Laryngoscope (September, 1937), 47: 615-691.

4/C. Firestone, op. cit., pp. 168-175.

Firestone described this condition as "aero-otosclerosis."

In 1941 Poppen ^{1/} considered hearing loss incidental to pressure changes in the middle ear of little significance. He was more concerned with the question of permanent impairment of hearing or damage to the auditory mechanism by the fatiguing effects of the noises encountered in flight.

Hallowell Davis ^{2/} suggested that there are three distinguishable signs that noise may be approaching dangerous levels: (1) the threshold of discomfort is about 120 db, (2) there is an unpleasant tickle in the ear at 130 db, and (3) at 140 db and above the noise is intense enough to cause pain and severe injury.

Persons working near turbojet engines may be exposed to intensities of 140 to 160 db and should always wear ear defenders or work in specially shielded rooms to avoid aural damage. ^{3/} Impaired hearing, buzzing, and diplacusis were noted by airport personnel after a short time exposure to "Vampire" jets despite ear defenders introduced into the meatus. ^{4/}

^{1/}J. R. Poppen, op. cit., pp. 974-982.

^{2/}Hallowell Davis, Hearing and Deafness, Rinehart, New York, 1947.

^{3/}Ross A. McFarland, op. cit., p. 479.

^{4/}L. Ruedi and W. Furrer, op. cit., pp. 534-541.

McFarland ^{1/} described an experience by Parrack who exposed his left ear to a turbojet engine having a sound field of 6,500 cps at a level of 159 db for 5 minutes:

"The initial pain was unbearable, and he moved out of the field somewhat. After about 3 minutes, he had the sensation of warm fluid in the external canal. At the end of the experiment, blood was found in the canal, and the eardrum was ruptured. There was generalized hearing loss at all frequencies at a stage when the middle ear was filled with fluid. Recovery was not complete after a lapse of about 1 year; some hearing loss is still present at frequencies between 9,000 and 12,000 cps."

The estimates for the maximum noise levels in each octave that will not, in time, produce permanent damage to an ear which is exposed daily and continuously for 8 hours out of every 24 hours vary considerably. According to Parrack and Eldredge, ^{2/} the most liberal of these estimates would allow as much as 110 db for frequencies between 75 and 150 cps and about 95 db for all higher octave bands. Sound levels as high as 95 or 100 db may be allowed for the frequencies below 150 cps, but the over-all level for all other frequencies up to 10,000 cps must be no more than 85 db.

Willis and Hoffman ^{3/} agreed that noise levels of 85 db or higher constitute a noise hazard area on the flight line.

^{1/}Ross A. McFarland, op. cit., p. 479.

^{2/}H. O. Parrack and D. H. Eldredge, op. cit., p. 470.

^{3/}H. S. K. Willis and I. Hoffman, "Hearing Loss from High Intensity Sound of Jet Engines," Aerospace Medicine (1959), 30: 764-772.

Both McFarland ^{1/} and McGirr ^{2/} believed that hearing conservation measures should be adopted if the sound pressure level in any of the four particular octaves of 300-600, 600-1200, 1200-2400, and 2400-4800 reaches or exceeds 85 db. This applies only to continuous sound. If the sound is intermittent, then the danger line might be at 75db instead of 85 db.

Hearing loss due to high intensity sound exposure makes its first appearance on audiograms at about 4,000 cps. If the exposure continues unchecked the loss spreads laterally along the frequency spectra--upwards from 4,000 and downwards into the 500-2,000 cps range, when disability occurs. The lesson here is that noise-induced hearing loss first affects hearing at frequencies higher than those necessary for communication by speech, therefore most early occupational deafness passes unnoticed by the workmen and will not be noticed until the disability is so extensive that it is all too obvious. ^{3/}

^{1/}Ross A. McFarland, "Psycho-physiological Problems of Aging in Air Transport Pilots," Journal of Aviation Medicine (1954), 25: 210-220.

^{2/}P. O. M. McGirr, "Effects of Noise," Reprinted from the British Encyclopedia of Medical Practice, Interim Supplement 198 (March, 1959).

^{3/}P. O. M. McGirr, op. cit.

Dickson ^{1/} also suspected a high-tone hearing loss to represent the effects of acoustic trauma. He felt that repeated and continuous exposure to noise may involve frequencies below the level of 4,096 cps. The individual is unaware of any auditory defects and probably notices no disability so long as loss is restricted to 4,000 cps and is not very marked.

Analysis of aircraft noise showed that the frequencies of the highest intensity were at the low end of the acoustic spectrum. Why, then, does prolonged exposure to aircraft noise produce a high-tone hearing loss?

Malone ^{2/} reasoned that it was a result of damage inflicted on the basal coil by excessive sound pressure of a complex noise of high over-all intensity. Malone, ^{3/} Armstrong ^{4/} suggested the theory that the external auditory meatus acted as a resonator and intensified a frequency in a complex sound in the region of 3,000 cps and thus caused a dip at 4,096 cps.

1/E. D. D. Dickson, op. cit., pp. 8-10.

2/P. W. Malone, "Aviation Deafness," Archives of Otolaryngology, Chicago (1944), 40: 468-474.

3/Ibid., p. 469.

4/H. G. Armstrong, op. cit., p. 277.

Graebner ^{1/} stated that the high tonal dip due to auditory fatigue and deterioration was once believed to occur at or near the pitch frequency of the greatest noise intensity. However, he discovered that although the over-all noise level is highest in the low range, greatest auditory deterioration occurs in the high frequencies. He believed that damage to the high-tone range may be due to its anatomical location in the basilar coil of the cochlea and its close proximity to the oval window, the point of greatest impact of undue stimulation.

Glorig ^{2/} advanced another concept of fatigue of the peripheral organ. He believed that most evidence points to an electrochemical change which renders the hair cell on the basilar membrane or its connecting structures incapable of firing or which blocks the normal conduction of the nerve impulse.

Armstrong ^{3/} expressed two other theories explaining why a high-tone loss is produced by great noise intensities in the lower frequencies: (1) the blood supply of the basal turn of the cochlea is less efficient than that of the other areas and

1/Herbert Graebner, "Auditory Deterioration in Airline Pilots," Journal of Aviation Medicine (1947), 18: 39-47.

2/H. Glorig et al, "Observation on Temporary Auditory Threshold Shifts Resulting from Noise Exposure," I, II, Annals of Otology, Rhinology, and Laryngology, (1958), 67: 824-827.

3/H. G. Armstrong, op. cit., p. 277.

it is possible that there is a nutritional factor involved, and (2) there is a natural island of hearing depression in some of the higher mammals (chimps) in the 4,000 cps area and following the adage that the functions last to appear in the phylogenetic scale are most vulnerable it may be possible that this area is most vulnerable to environmental phenomena.

Exposure to high intensity sound may produce aural pain. However, the threshold of aural pain does not serve as a danger signal for injury to the organ of Corti or for hearing loss. The threshold for aural pain is much too high to allow pain to serve as a warning against acoustic trauma. By the time the noise is loud enough to produce pain, hearing loss is well on its way.^{1/}

Von Gierke et al.^{2/} measured the threshold for aural pain in the frequency range from 0-2,000 cps. The Auditory Range Pain Threshold was determined to be roughly 140 db. Below 15 cps it increased to 179 db for static pressure. They concluded that aural pain most probably arises from a region of the middle ear which included the drum membrane and ossicles and not from the organ of Corti.

^{1/}H. E. von Gierke et al., "Aural Pain Produced by Sound," in Benox Report: An Exploratory Study of the Biological Effects of Noise (December, 1953), 29-36. ONR Project NR 144079, University of Chicago.

^{2/}Ibid, p. 30.

The results of Ades ^{1/} study of the threshold of aural pain to high intensity sound indicated that a degree of interdependence may exist between pain perception and hearing. This explains the fact of aural pain disappearing coincidentally with the development of hearing loss and why workers will ignore ear protection once acoustic trauma has proceeded far enough.

There have been many studies made to determine the amount and type of loss resulting from exposure to aircraft noise.

Simonton ^{2/} conducted a study of the hearing of 47 airline pilots taken at intervals of 10 years each for each pilot. He found that the hearing loss of 36 out of 47 pilots was 15 or more db in one or more frequencies for frequencies of 4,096 cps or higher.

Rüedi and Furrer ^{3/} studied 4 persons with normal audiograms who were exposed to "Vampire" jet noise of 136 db for ten minutes at the front of the plane. Two subjects wore close-fitting hard rubber caps over the ears. The unprotected subjects had a clear hearing loss which began at 500 cps;

^{1/}Harlow W. Ades et al, op. cit., pp. 678-684.

^{2/}K. M. Simonton, "Hearing of Airline Pilots: A Ten-Year Study," Journal of Aviation Medicine (1949), 20: 418-429.

^{3/}Rüedi and Furrer, op. cit., pp. 534-541.

curves dropped sharply at 8,000 cps. Subjects recovered from the test loss in 24 hours. Those protected by the ear defenders had no hearing loss.

Senturia ^{B/} conducted a study of the influence of airplane noise on auditory thresholds. He obtained pure-tone auditory threshold tests on 100 enlisted trainees who had had no recent exposure to airplane noise. Threshold determinations were repeated on 74 subjects who had successfully completed 9 weeks of primary training and who had had 4-6 hours of freedom from aircraft noise before retest. 18.9% of the re-examined ears showed an elevation of threshold of 15 db or more at 1 or more of the frequencies tested. This elevation occurred most often at the frequencies 2896, 4096, and 5792 cps.

Following 135 hours of flying, 19.5% of the ears showed threshold elevations of 15 db or more at one or more tested frequencies. The greatest elevation occurred at the higher frequencies 2048-5792 cps.

Following advanced training with exposure to 123 db noise level, subjects who had 24 hours' rest showed partial or complete recovery. Subjects who had only 1-8 hours freedom from noise had widespread hearing loss, involving frequencies of 1024-5792 cps inclusive.

1/Ben H. Senturia, "The Influence of Airplane Noise on Auditory Thresholds," Annals of Otology, Rhinology, and Laryngology, (1952), 61: 331-349.

E. S. Mendelson et al.^{1/} tested 9 volunteer subjects, aged 18-21, wearing ear defenders and exposed to turbo-jet engine noise operated in an open-end test cell at cruising speed (15,000 rpm) for periods of 1-2 hours. Exposures extended over 26 days. Hearing loss was found in 4 subjects ranging from 20-60 db threshold elevation between 512 and 1024 cps. The effects were reversible, however and auditory acuity returned to normal in two or three days.

Rosalie Noble^{2/} obtained audiometric data over a 5 to 8 year period for 76 men employed as engine test laboratory personnel. The frequency range extended from 128 cps to 8192 cps in octave intervals. There was no apparent change attributed to exposure to the high-intensity noise environment.

Five hundred flight-line mechanics who were exposed to aircraft engine noise for periods up to ten years were tested by Barron.^{3/} These men were engaged in servicing reciprocating, turbo-prop and turbo-jet engines for Lockheed

1/E. S. Mendelson et al, "Turbo-jet Engine Noise. Vibrational Frequencies and Intensities Encountered in Engine Test Cells," Journal of Aviation Medicine (1948), 19: 365-374.

2/Rosalie Noble, "Effect of Noise Environment of an Engine Test Laboratory on Auditory Acuity," Journal of Aviation Medicine (1956), 27: 452-489.

3/Charles I. Barron, "Audiometric Studies of Flight Line Mechanics," Journal of Aviation Medicine (1957), 28: 295-302.

Aircraft. He found no significant changes in the mean or median tests for the group at 1,000 or 4,000 cps other than those generally associated with presbycusis. Individual threshold changes revealed only 6 cases of hearing loss of 20 db and higher at 4,000 cps with a maximum loss of 45 db.

Threshold changes appear to be unrelated to ear protection. Daily exposure of this group was limited to a few minutes. Auditory nerve damage and hearing loss of a significant degree could not be frequently demonstrated.

Berry and Eastwood ^{1/} studied the effects of helicopter flights of average duration of 1.4 hours on 33 marines. All exhibited objective and subjective hearing loss. Those marines who had no ear protection showed a hearing loss of 22 db. Within 24 hours the hearing had returned to normal in 56% of the cases. Approximately 60% of the flights produced symptoms of tinnitus, headache, and "ear plugging." There were less frequent complaints of drowsiness, nausea and nervousness.

The negative results of these studies support the fact that there are very marked individual differences in susceptibility to auditory fatigue. ^{2/} The existence of marked

^{1/}Charles A. Berry and Herbert K. Eastwood, op. cit., pp. 179-190.

^{2/}R. Plutchik, "The Effects of High Intensity Intermittent Sound on Performance, Feeling and Physiology," Psychological Bulletin (1959), 56: 133-151.

individual differences are attested by the fact that the least susceptible subjects may return to normal in less than 7 minutes from stimulation which the most susceptible do not recover from in more than 24 hours.

Hearing loss, according to Harris,^{1/} tends to be a linear function of both stimulus duration and intensity-- but for fatigue resistant individuals, recovery from 10 minutes of exposure to noise levels of 120 db takes only about 7 minutes.

McGirr^{2/} stated that after a high intensity sound exposure there is normally a period of recovery. The more intense and prolonged the stimulus, the longer the recovery period. This transient hearing loss is called the threshold shift. With each succeeding severe exposure, the period of recovery increases until finally recovery becomes less and the deafness a permanency.

Glorig^{3/} determined the feasibility of using this temporary threshold shift data to predict the amount of permanent hearing loss resulting from years of exposure to any given noise. He stated that if a noise does not produce a permanent hearing loss in 1 day of exposure then it will not

1/Ibid., pp. 133-151.

2/P. O. M. McGirr, op. cit.

3/H. Glorig et al, op. cit., pp. 824-827.

produce a permanent hearing loss after many years of exposure.

He exposed 99 normal hearing subjects to 100 db sound pressure level for 2 hours. Some were exposed for 1 hour only, some for 6 hours. The control group had no noise exposure. Growth of and recovery from temporary threshold shifts were determined from auditory thresholds measured at 30 minute intervals during 2 hours of exposure and 2 hours of recovery following exposure.

Glorig's results indicated that (1) the threshold shift produced at 4,000 cps was significantly larger than the shift at 1,000 cps; (2) at 4,000 cps temporary threshold shifts of more than 50 db recover more slowly than do shifts of less than 50 db; (3) the maximum noise shift produced by exposure to octave bands of noise occurs, on the average, one-half octave to one octave above the nominal high cut-off frequency of the exposure band.

Kryter ^{1/} has suggested that tones of 85 db may cause some deafness following exposure to noise applied intermittently over months or years. Intensities of 100 db over the entire frequency spectra can cause deafness after brief exposures lasting up to an hour.

1/K. D. Kryter, "The Effects of Noise on Man," Journal of Speech and Hearing Disorders (1950), Monograph Supplement Number 3, 95 pp.

The Effects of exposure to intermittent and continuous sound.-- According to Plutchik,^{1/} there are three aspects to the problem of the effects of intermittent sound on feelings: (1) the nature of the feelings that are associated with high intensity noise; (2) the effect of such noise on threshold adaptation and auditory acuity; and (3) the special subjective characteristics of intermittent, repetitive, or pulsed sounds.

Plutchik considered the effects of irregular or intermittent sound on performance, feeling, and physiology more disturbing than those of steady sound sources. High intensity noise, even when it may have no effect on performance, will generally produce symptoms of discomfort, irritability, and distraction.

Armstrong^{2/} stated that irregular noises, such as those from non-synchronized propellers on multi-motored airplanes, and from the radio beam or radio static tend to produce irritability as well as fatigue.

The Benox Report^{3/} revealed that a steady noise of 115 db heard for three hour periods produced fatigue and discomfort.

1/R. Plutchik, op. cit., pp. 133-151.

2/H. G. Armstrong, op. cit., p. 271.

3/Benox Report: An Exploratory Study of the Biological Effects of Noise (December, 1953), pp. 29-36. ONR Project NR 144079, University of Chicago.

Glorig ^{1/} maintained that although the total acoustic energy of intermittent and continuous sound may be the same, if the end organ is allowed to rest between assaults, then it is given a chance to recuperate its losses or to recharge its battery. This explains why industries' riveters whose ears are exposed continuously to noise develop more permanent hearing loss than do jet flight-line mechanics. Results of Glorig's study of temporary auditory threshold shifts resulting from noise exposure indicated that temporary threshold shifts produced by intermittent sound was significantly less than the shift produced by continuous sound exposure.

Methods of assessing hearing loss for airmen.-- The intense noise associated with aviation and the efficient hearing required of individuals in aviation are conflicting factors.

In setting up hearing requirements for airmen, it is necessary to go beyond the tests that otologists use and to establish requirements that are specifically related to auditory performance in flight. ^{2/} McFarland stated that:

1/H. Glorig et al., op. cit., p. 827.

2/Ross A. McFarland, Human Factors in Air Transportation, op. cit., p. 123.

"Clinical tests for the ability to hear pure tones are helpful in diagnosing individual differences, changes due to age, and the effects of noise and of changes in barometric pressure but do not accurately appraise the ability to hear in the presence of noise on aircraft."

Hinchcliffe ^{1/} and Wheeler felt that an aviation otologist is often confronted with the problem of whether a man may continue as an air crew member when he has developed a traumatic hearing loss. An airman's ability to use telecommunications cannot be assessed solely by his pure tone audiogram. They maintained that the test material should be a form of speech audiometry in which the words are presented against a background of aircraft noise.

Present audiometric methods used by the Federal Aviation Agency (FAA) are based primarily on the ability to hear the whispered voice at 20 feet. This test is notoriously poor because of the uncontrolled ambient noise present at the time of testing. A survey of the medical records for airline pilots indicated that about 98% of them pass such tests. ^{2/} The FAA recommends an audiometric examination for all those failing to pass the whispered-voice test at less than 20 feet for either ear.

1/R. Hinchcliffe and L. J. Wheeler, "An Investigation of the Effect of Flying on Speech Intelligibility in Noise," Journal of Aviation Medicine (1957), 28: 277.

2/Ross A. McFarland, op. cit., p. 124.

The International Civil Aviation Organization standards state that a candidate applying for an airline transport license should not have a loss in either ear of more than 20 db at any one of the four frequencies, 500, 1,000, 2,000 and 3,000 cps.^{1/} If an older, more experienced pilot cannot reach this standard then he is given a more functional test consisting of the receipt of radio telephone signals in a complex noise background of an intensity level of not less than 100 db with the intensity level of the signals being 8 db above the intensity level of the background noise.^{2/}

In selecting flying officers for the military services and candidates for West Point, The Committee on Hearing of the National Research Council has recommended that not more than a 15 db loss should be allowed at 250, 500, 1,000 and 2,000 cps and not more than 30 db at 4,000 to 8,000 cps.^{3/}

The need for audiometric tests at the time of testing can be demonstrated by Senturia's^{4/} study of the auditory acuity of 500 aviation cadets. These cadets, aged 18 to 27, had

^{1/}Pothoven and Schuringa, op. cit., p. 381.

^{2/}Ross A. McFarland, op. cit., p. 125.

^{3/}Ross A. McFarland, op. cit., p. 126.

^{4/}Ben H. Senturia, "Auditory Acuity of Aviation Cadets," Annals of Otology, Rhinology, and Laryngology (1944), 53: 705-716.

little or no flying experience and had not been exposed to aircraft noise for 30 days. He found that almost 15% of the group had an average of more than 45 db hearing loss at frequencies of 250 to 4,000 cps in both ears.

A distinction must be made between clinical audiometry in the laboratory and operational audiometry, or the hearing requirements of pilots on flight duty. Certain pilots with marked high frequency hearing losses caused by age or continued exposure to noise are able to hear the necessary signals in flight. Since such individuals show improvement with a noise background, actual or simulated tests in aircraft are indicated.^{1/}

The hearing requirements of a pilot while in flight involve (1) an understanding of speech in a noise background, the most important frequencies being 500 to 3,000 cps; (2) the four-course radio range or radio beam, operating at 1,020 cps; (3) the fan and cone markers at 3,000 cps and inner and outer markers at 1,300 and 400 cps, respectively; (4) the perception of coded messages; and (5) the detection of irregularities in engine noise, warning signals, and many other areas. Test frequencies between 400 and 3,000 cps would give a satisfactory frequency over which the pilot's hearing should be measured.^{2/}

^{1/}Ross A. McFarland, op. cit., p. 126.

^{2/}Ross A. McFarland, op. cit., p. 126.

A pilot with a hearing loss of 30 db might require about 1-watt amplification of the 1,020 cps radio beam-- an intensity level that leads to distortion and possible misinterpretation of the signals. Similarly, at 3,000 cps, a loss of more than 25 db would mean that the aural signal of the fan and cone markers would not be heard. Fan and cone markers, however, operate a visual signal on the instrument panel, as well as give an aural signal.^{1/}

Hinchcliffe and Wheeler devised a hearing efficiency test for the Royal Air Force which used a recorded phonetically-balanced word list against a background of aircraft noise. The records were replayed to reproduce noise at a sound pressure level of 100 db and the speech at 106 db at the ears. They found that a long operational flight in a Shackleton aircraft had no deleterious effect on the crew's efficiency over the telecommunications channels, as distinct from any effect on auditory acuity.^{2/}

Simonton^{3/} also stated that threshold audiograms taken in quiet surroundings were not a good index of efficiency of of pilot's hearing in flight. He suggested that hearing tests

^{1/}Ibid., p. 126.

^{2/}Hinchcliffe and Wheeler, op. cit., p. 277.

^{3/}K. M. Simonton, op. cit., pp. 418-429.

for the spoken voice and directional radio beam be delivered by earphones and conducted against simulated aircraft noise.

The recruitment phenomenon, i.e., an apparent improvement in hearing in the presence of noise, has been studied extensively by Pothoven and Schuringa ^{1/} who believed that this factor should be considered in the hearing requirements for older pilots. They felt that the recruitment factor is responsible for the fact that experienced flyers with a considerable hearing loss do not have the slightest difficulty in their flying duties although their hearing acuity does not come up to present hearing standards. They stated that:

"If we want to bring the hearing standards for pilots in accordance with the practical requirements, then we can no longer neglect the very important recruitment phenomenon. In the absence of acute and chronic diseases of the ear, one must realize that a hearing loss of 30 db in the speech frequency has an entirely different meaning in it due to a conduction deafness or whether it is caused by a perception deafness. Increasing the intensity up to 70 db will have the result that a conduction deafness hears such an intensity always 30 db less, whereas a perception deafness with recruitment, hears the noise or the human voice with normal or nearly normal intensity and loudness."

Armstrong ^{2/} believes that because of the recruitment phenomena, one should not arbitrarily ground older pilots on the basis of a supposed lack of ability to use their radios successfully unless by actual test in flight it can be proved otherwise.

^{1/}Pothoven and Schuringa, op. cit., p. 386.

^{2/}H. G. Armstrong, op. cit., p. 278.

Simonton ^{1/} agreed that recruitment, while in flight, enables the pilot to discriminate between sounds of 80 to 100 db of intensity and that it is more important than his threshold of hearing. Thus the general increase of auditory threshold will be compensated by an improvement in the intelligibility of speech under high noise levels.

Methods of protection.-- Ear protectors can reduce noise level at the ear by 10-45 db and occasionally to 50 db, depending on their make and the sound frequency. ^{2/}

There are four types of ear protectors, classified according to their position relative to the ear: (1) ear plugs, (2) semi-inserts, (3) ear muffs, and (4) helmets.

Ear plugs are inserted into the ear canal and usually remain there without any additional means of support. When inserted correctly, they provide high sound attenuation, are unobtrusive, and do not interfere with head covers, masks, goggles, or other devices worn on the head. Because they are small, they can be carried in a pocket. Ear plugs are the least expensive ear protectors. However, they are often uncomfortable and may cause pain in the auditory canal, or even, in extreme cases, inflammation, especially in a tropical

^{1/}K. M. Simonton, op. cit., pp. 418-429.

^{2/}Josef Zwislocki, "Ear Protectors," Chapter VIII in Handbook of Noise Control, Cyril M. Harris, Editor. McGraw-Hill Book Company, Inc., New York, 1957.

climate.

Semi-inserts are supported by a headband and are not used generally as ear protectors, but rather as part of the ear-phone system. They close the entrance to the ear canal without being inserted into it.

Ear muffs cover the entire ear in the same way as do earphones mounted in an earphone cushion.

Helmets are not commonly used for ear protection alone. Since they cover most of the head surface, they combine ear protection with protection of the head against cold or injury.

Ramp attendants and service crews are required to use ear plugs and ear muffs during engine warm-ups. When jet engines are serviced, they should be placed in acoustically treated test cells.^{1/}

Thiessen and Shaw explained that vibration of the whole ear protector is the main mechanism by which low-frequency sound reaches the ear when a well-scaled protector is used. This vibration can be minimized by means of a cushion which contains a high-bulk filler.^{2/}

Intelligibility of speech in the presence of ambient cockpit noise may be improved by the use of ear defenders since they reduce not only over-all noise levels but specific

^{1/}Editorial: "Noise of Jet Engines May Be a Hazard," op. cit., p. 1629.

^{2/}G. J. Thiessen and E. A. G. Shaw, "Ear Defenders for Noise Protection," Journal of Aviation Medicine (1958), 29: 810-814.

levels in certain frequency components.^{1/}

Berry and Eastwood's^{2/} study of helicopter noise problems revealed that hearing loss and symptoms could be reduced by providing protection in the form of standard ear defenders or even cotton. Combat helmets alone offered no protection. The percentage of subjects wearing ear defenders who had post-flight hearing loss was reduced to 37% and 93% had recovered within 24 hours. The average db loss per frequency was also reduced to 6 db by cotton and 3 db by ear defenders.

Metcalf and Witwer^{3/} found that without ear protection, there was a resulting hearing loss of 18 to 22 db among men who flew in military helicopters. Cotton plugs and rubber ear defenders gave almost complete protection against this loss.

Mendelson et al,^{4/} found that protective assemblies of ear plugs, ear cushions, and headphones raised the threshold 25-35 db less at the lower frequencies and more at the higher frequencies when tested with white noise and pure tones.

^{1/}Ross A. McFarland, op. cit., p. 188.

^{2/}Berry and Eastwood, op. cit., pp. 179-190.

^{3/}C. W. Metcalf and R. G. Witwer, "Noise Problems in Military Helicopters," Journal of Aviation Medicine (1958), 29: 29-65.

^{4/}E. S. Mendelson et al., op. cit., pp. 365-374.

The masking effects of aircraft noise on passenger conversation are fairly well controlled if the intensity level of the 500 to 4,000 cps frequency band and particularly the 1,000 to 2,000 cps bands are approximately 70 db.^{1/}

The noise inside the cabins of commercial airliners has been successfully suppressed by soundproofing.^{2/} Noise as a source of passenger discomfort has become of negligible importance with the introduction of jet engines.

^{1/}Ross A. McFarland, op. cit., p. 700.

^{2/}H. E. Whittingham, "Aero Medical Problems of Jet Passenger Aircraft," Journal of Aviation Medicine (1954), 25: 440-450.

CHAPTER III
SUMMARY AND CONCLUSIONS

Summary of aerotitis media.--- From the year 1783 when the first case of aerotitis media was reported to approximately 100 years later, little investigation was made. During World War II regulations were adopted requiring candidates for flying training to have patent eustachian tubes. In 1937 Armstrong and Heim were the first to denote the condition as a distinct clinical entity and introduced the term aerotitis media. With the onset of World War II, a great amount of research was initiated. Aerotitis media became a rejecting criterion for prospective pilots. In the same year the Valsalva and Polizer methods of eustachian tube ventilation were reported.

The most important function of the eustachian tube is ventilation of the middle ear thereby equalizing air pressure on both sides of the tympanic membrane. The physiology of the eustachian tube functions well on ascent; however, most cases of aerotitis media occur during descent since the ear does not automatically adjust to the increase of air pressure differential. Therefore a positive pressure develops on the outside of the eardrum, pushing it inward, resulting in decreased auditory acuity, tinnitus, pain or rupture of the ear drum. Since pressure differential built up in the middle ear depends

not only upon the amount of descent but also upon the altitude at which descent occurs, pilots are instructed to avoid rapid changes of ascent and descent thereby causing inefficient operations of airliners at times.

Another problem is the awakening of passengers when the plane descends from any considerable altitude to prevent traumatization of the middle ear. Even with pressure changes in super-charged cabins and regulation of the rate of descent, all of the problems will not be eliminated. Since the ability to equalize pressure is blocked by inflammatory processes in the upper respiratory tract causing the temporary stenosing of the eustachian tube, passengers with upper respiratory infections should delay their flight.

A few of the most important causes producing this disease are infections of the upper respiratory tract, nasal obstructions, sinusitis, tonsillitis, growths of the nose and nasopharynx, paralysis of the soft palate or superior pharyngeal muscles, enlargement of the tubal tonsil, and malposition of the jaws.

Studies on incidence have revealed that of 100 men examined per month, 25% developed aerotitis media. Otolologists have noted that repeated exposure to barometric pressure changes may lead to a chronic thickening of the tympanic and eustachian tube lining with eventual connective tissue formation resulting in a considerable reduction of hearing for low tones. If therapy is soon initiated, the impairment may be

reversible, but may not be so if therapy is neglected or unsuccessful.

The disease may be defined as a pathological condition of the middle ear and drum membrane ranging in severity from slight erythema of the drum membrane to frank hemorrhage into the middle ear or drum rupture caused by a pressure differential between the middle ear and that in the surrounding atmosphere. Symptoms of pain, a feeling of pressure in the ear, loss of auditory acuity or bleeding from the external canal may occur.

Various methods of treatment are advocated although at times the best therapy is not at all because of the danger of introduction of a secondary infection. When therapy is advocated, the following methods are used: (1) radium applied to the pharyngeal opening of the eustachian tube, (2) dental corrective measures, (3) instillation of a nasal astringent, (4) aspiration of excess secretions, (5) frequent tubal inflation, (6) catherization, and (7) politzerization.

Conclusions and Implications.-- With the increasing use of air transportation as a means of saving time and linking distant nations closer together, aerotitis media may become a condition with an ever increasing incidence. Since many audiologists and speech clinicians may not be familiar with the conditions and symptoms of aerotitis media incorrect or partial symptomatic therapy may be initiated thereby causing low tone conductive deafness at minimum or bleeding into the

middle ear and drum membrane rupture at maximum. Not only can the passengers of airliners be affected, but pilots as well. It is, therefore, extremely important for all individuals dealing with persons afflicted with hearing problems to recognize the symptoms and to be cognizant of the etiological factors of aerotitis media so that prompt referral for appropriate therapy can be made.

Summary of the effects of aircraft noise.-- Otologists have been increasingly concerned with the effects of aircraft noise intensity on the hearing mechanism so that permanent nerve deafness as the result of acoustic trauma may be avoided.

The sound pressure of the higher frequencies is responsible for the damage done to the hearing function. Intelligibility of speech and intercommunication depends largely on the amount of high frequencies. Therefore the analysis of aircraft noise as a cause of deafness is important. The intensity level of aviation noises is in the region of 120-130 db. Since the intensities of these noises are very deceptive for in flight everyone feels that the environment is almost perfectly quiet, one is amazed to find the loudest shout or even one's own voice cannot be heard immediately after landing.

With a "safe" noise level considered to be no more than 85 db, prolonged exposure to various types of aircraft noise spectra can cause serious injury to the unprotected ear.

In 1937 it was demonstrated that deafness produced by noise is a nerve deafness and is most marked immediately after excessive stimulation followed by a variable period of recovery. An audiogram revealed a sharp loss in the frequency area of 4,096 cps.

Noise and vibration cause injury to the ear, the extent of which is directly proportional to the flying time.

The three distinguishable signs that noise may be approaching dangerous levels are: (1) the threshold of discomfort is about 120 db, (2) unpleasant tickle in the ear at 130 db, and (3) at 140 db or above the noise is intense enough to cause pain and injury.

Persons working close to turbojet engines may be exposed to intensities of 140 to 160 db and should take precautions to avoid aural damage. Impaired hearing, buzzing and diplacusis were noted by airport personnel after short time exposure to jet-propelled planes despite precautions taken.

Most early occupational deafness passes unnoticed by workmen and will not be noticed until the disability is so extensive as to be all too obvious. The individual upon exposure is unaware of any auditory defects so long as the loss is restricted to 4,000 cps and is not very marked.

It was shown that frequencies of 110-115 db were at the low end of the acoustic spectrum and suggested that a low-tone was causing a high-tone loss.

The Auditory Range Pain Threshold was determined to be

roughly 140 db. The threshold of aural pain does not serve as a danger signal for damage to the organ of Corti or for hearing loss. The Threshold is too high to allow pain to serve as a warning against acoustic trauma.

A study conducted on the hearing loss of pilots showed that the loss was 15 db or more in one or more frequencies for frequencies of 4,096 cps or higher.

There are very marked individual differences in susceptibility to auditory fatigue.

It was shown that after a high intensity sound exposure there is normally a period of recovery. The intensity and prolongation of the stimulus is directly proportional to the length of the recovery period.

In assessing hearing loss for airmen one group of investigators felt that the test material should be such that the words are presented against a background of aircraft noise.

The present methods used by the Federal Aviation Agency are poor because of the uncontrolled ambient noise present at the time of testing.

The International Civil Aviation Organization standards state that a candidate for license should not have a loss in either ear of more than 20 db at any one of four frequencies which are 500, 1,000, 2,000 and 3,000 cps.

The need for audiometric testing at the time of pilot selection is a must.

A distinction between clinical and operational audiometry should be made.

Test frequencies between 400 and 3,000 cps would be satisfactory for measuring a pilot's hearing.

The recruitment phenomenon must be considered in the hearing requirements for older pilots especially.

Ear protectors can reduce the noise level at the ear by 10-45 db. The four types of ear protectors are: ear plugs, semi-inserts, ear muffs and helmets. All of the various studies involving the use of ear protectors as a means of protection against hearing loss proved the essentiality of such devices to be worn by pilots and ground personnel.

Conclusions and implications.-- The studies on aircraft noise are extremely effective in stressing the importance of the initiation of a noise exposure control program.

The amount of hearing preservation and the amount of acoustic trauma acceptable along with the amount of compensation needed for such trauma constitute important knowledge for audiologists.

The preservation of the hearing acuity of airline pilots is an important factor in air safety. With the better understanding of the problems involved, greater precautions can be taken to prevent hearing loss, and most important, nerve deafness.

Audiologists and speech clinicians must be well aware of the possible hearing loss or deafness of aircraft and ground

personnel so that early pathological conditions may be noted and corrective measures taken wherever possible.

Audiologists and speech clinicians can and will play an important role in methods of prevention and preservation of hearing loss due to acoustic trauma caused by exposure to aircraft noise.

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