AVIATION PRESSURE DEAFNESS

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IN his survey of the aural lesions associated with flying, Wing Commander Simpson mentioned a group of symptoms which sometimes occurs owing to closure of the Eustachian tube during descent in aircraft—a syndrome known in America as " aero-otitis ", and which I have called " aviation pressure deafness ".

As I am attached to a Royal Air Force General Hospital which serves many Operational Stations, I have had the opportunity to examine a relatively large series of patients suffering from this disability.

Twenty-six per cent. of the total number of aircrew personnel referred to the ear, nose and throat department of this hospital have complained of aural symptoms which have arisen during loss of height in aircraft ; and for the purposes of the following notes I have reviewed 100 cases.

I propose to deal shortly with :

(1) *The main features of obstruction of the Eustachian tube,* and

(2) *The nature of the obstruction.*

(1) PROOF OF EUSTACHIAN BLOCKAGE has been based on :

A. *Presumptive evidence.* B. *Positive evidence.* (i) *Type of deafness.* (ii) *Otoscopic appearances.* (iii) *Impermeability of the tube to air.* (iv) *Impermeability of the tube to the passage of tympanic catheter or sound.* (v) *Impermeability of the tube to radio-opaque fluids.*

A. *Presumptive Evidence*

(i) *Deafness* alone or in association with other symptoms was the most common complaint of these patients, and this is shown in Table I which is an analysis of their predominant symptoms :

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TABLE I.

PREDOMINANT SYMPTOMS OF 100 PATIENTS

Deafness alone	31
Deafness and pain	55
Deafness and tinnitus	5
Deafness and vertigo	3
Pain alone	6
	100

It will be seen that 94 *patients* complained of *deafness*, and probably had the last six cases been examined sooner after landing a deficiency of hearing would have been found.

I am convinced that some degree of deafness is a constant symptom of the lesion.

In ten cases the hearing was normal at the time of first examination. In the remainder deafness was present, and this was of the "conductive" type with loss of acuity for low tones; and although some patients could hear a whispered voice at 20 ft. they mostly suffered from some loss of hearing for the forks C and C1, and from 10 to 30 decibels by the audiometer. In three patients only was there any loss for high tones, but not one of the series manifested the severe and permanent "inner-ear" type of deafness mentioned by Wing Commander Simpson.

It is noteworthy also that deafness was usually the last symptom to disappear.

(ii) *Otoscopic appearances* (six hours to seven days after onset).

(*a*) *Normal drumheads* were seen in twenty-six patients.

(*b*) *Invagination of the tympanic membrane* of a varying degree was the most common finding and this was noted in forty-seven patients. The appearances of this condition are familiar, but I would call attention to the word "*invagination*" which was used by Mr. Sydney Scott (1919) in his report of similar cases observed during the last war. It is more truly descriptive than "retraction", as the drumhead is *pushed in* by atmospheric pressure around the middle-ear contents. Invagination of the drumhead was always accompanied by some congestion of the region of the handle of the malleus and of the sheaf of vessels which sweep backwards to the posterior malleolar fold. This vascularity varied in intensity and shaded off into the two following changes:

(*c*) *Attic congestion* which was present in six cases, and

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(*d*) *Congestion of the entire drumhead*, which was noted in eight patients. These two conditions simulate, and are often mistaken for, acute bacterial otitis media.

(*e*) *Effusion into the tympanum* was seen in six patients at the time of first examination. Two cases were as usually described, in three patients the effusion was confined to the posterior compartment of the middle ear, and in one there was a localized effusion into Prussak's space. Two other patients developed tympanic effusion after admission to hospital.

(*f*) *Rupture of the drumhead* was present in seven patients. Six of the perforations were secondarily infected and moist when first seen, the remaining one was dry. In three patients the lesion was in the posterior segment and in four it was anterior to the handle of the malleus.

(*g*) *Evidence of old healed middle-ear lesions*—such as atrophic scars, calcareous patches and local or general increased opacity of the tympanic membrane—was present in several patients in addition to the appearances already described, and four patients had submitted to simple mastoidectomy in childhood.

B. *Positive Evidence*

(iii) *Impermeability of the tube to the passage of air. Inflation* or "*forced ventilation*" *of the middle ear*, i.e. *passive opening* of the Eustachian tube, leads to a consideration of the pressures that can be created in the nasopharyngeal and nasal cavities by different procedures.

(*a*) *Valsalva's method of inflation* is the generally accepted proof of tubal patency, and its successful performance is insisted on by the Royal Air Force before acceptance for aircrew duties. Twenty-one patients could inflate their middle ears by this method when first examined. The average pressure found to be necessary to cause air-entry through a *normal* tube was found to be 30 mm.Hg. The average maximum pressure that could be produced by 100 unselected patients was 114 mm.Hg. and the maximum achieved by any one patient was 220 mm.Hg.

(*b*) *Valsalva's method combined with swallowing movements* was successful in one case in which Valsalva's manœuvre alone had failed. It has been difficult to measure the pressure created by this combined procedure but estimations have been carried out in five patients with a perforated drumhead and patent Eustachian tube. The manometer was connected by

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an accurately fitting nozzle into the external auditory meatus and whilst the subject maintained a constant pressure by auto-inflation, when he swallowed the mercury rose on an average 20 mm., and then suddenly fell. I think the success of this method was due to two factors :

Increased patency of the tube brought about by swallowing and

A further, though slight, increase of nasopharyngeal pressure caused by the air-content of the pharynx being forced into the nasopharyngeal and nasal cavities during the " closed phase of the pharynx " (Barclay, 1930) whilst the anterior nares were occluded.

(*c*) *With Politzer's bag* the average maximum nasopharyngeal pressure obtained was 210 mm.Hg.

(*d*) *With the Eustachian catheter* in the tubal orifice estimation of the maximum pressure at its tip has been made and found to be 320 mm.Hg. In only one case was inflation by Eustachian catheter successful at the first visit.

(*e*) *Inflation through a tympanic catheter* was successful also in one patient, but the pressure created in the Eustachian tube was not measured.

(iv) *Impermeability of the tube to a tympanic sound and/or catheter.*—An attempt to pass a tympanic sound or catheter was made in fifty-seven patients and *in all* the passage of the instrument was arrested by a firm obstruction at a distance varying from 0·5 cm. to 2 cm. from the tip of the catheter.

(v) *Radiological evidence of obstruction after the injection of radio-opaque fluid* into the Eustachian tube has been demonstrated in thirty-six patients of this series by Squadron Leader Rees-Jones and myself. The technique and results in thirty-four of these have been published (1941), but I would mention that in two patients the obstruction was at the isthmus, in one at 0·5 cm. from its pharyngeal orifice, and in the remaining thirty-three at intermediate situations.

(2) The Nature of the Tubal Obstruction

Commonly the lumen of the posterolateral half of the cartilaginous tube is closed by contact of its mucosal walls and possibly by a film of mucus ; but the extent and degree of closure vary individually, and in a small number of people the tube is constantly open throughout its entire length.

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The question of obstruction of a *normally closed* tube naturally leads to a consideration of the means whereby its *active opening* and *normal ventilation* of the middle ear are brought about.

The cartilaginous tube is cradled by muscles.

From its hamular process and its anterolateral aspect the greater part of the tensor palati muscle takes origin (Fig. 1).

Its postero-medial wall is in contact with the levator palati muscle and some of the fibres may arise from this wall. This muscle gradually comes to lie on the inferior aspect of the tube to form the floor of its pharyngeal opening (Fig. 2).

The slender salpingo-pharyngeus muscle takes origin from the cartilage near its free extremity, whilst below it is the upper margin of the superior constrictor muscle and that portion of the palato-pharyngeus muscle which forms Passavant's bar (Fig. 2).

Functionally the muscles of importance are :

(1) *The tensor palati muscle ;*

(2) *The superior constrictor muscle* in its upper part ; and

(3) *The sphincter of the pharyngo-nasal isthmus,* which is formed by :

(*a*) *The soft palate* anteriorly ;

(*b*) *The palato-thyroideus* (posterior pillar) laterally ; and

(*c*) *Passavant's bar* posteriorly.

The mechanism of active opening of the tube has long been controversial, and McMyn (1940) has recently and fully summarized the existing theories. He concluded that the tube is opened by the tensor palati muscle which causes relaxation of its membranous wall, and that other muscles (levator palati, salpingo-pharyngeus, superior constrictor) *may* act synergically.

My own conception is that the tube is opened in a sinuous manner by the combined action of the sphincter of the pharyngo-nasal isthmus and of the upper part of the superior constrictor muscle which drag the postero-medial cartilaginous wall inward : whilst simultaneously the tensor palati muscle braces the antero-lateral wall against the medial pterygoid lamina and draws down the hamular process of the cartilage to relax the membranous wall (Fig. 3).

It is known that the tube is opened by swallowing, yawning and gaping movements ; that opening may occur during phonation depending on the amount of contraction of the

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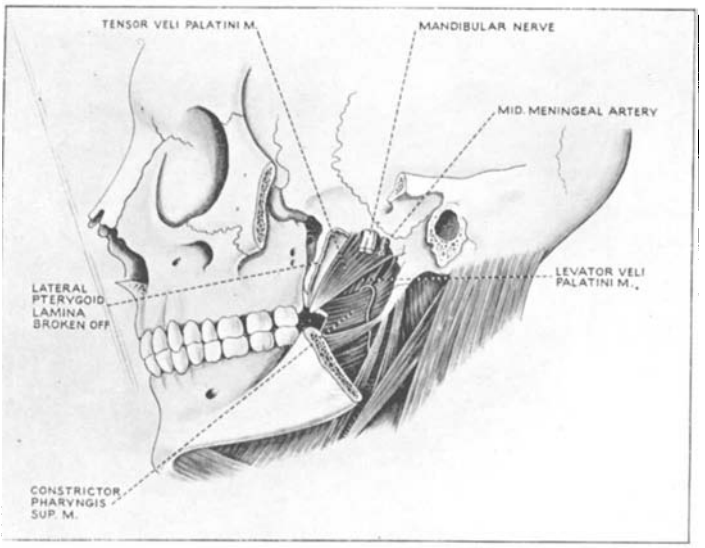


FIG. 1.

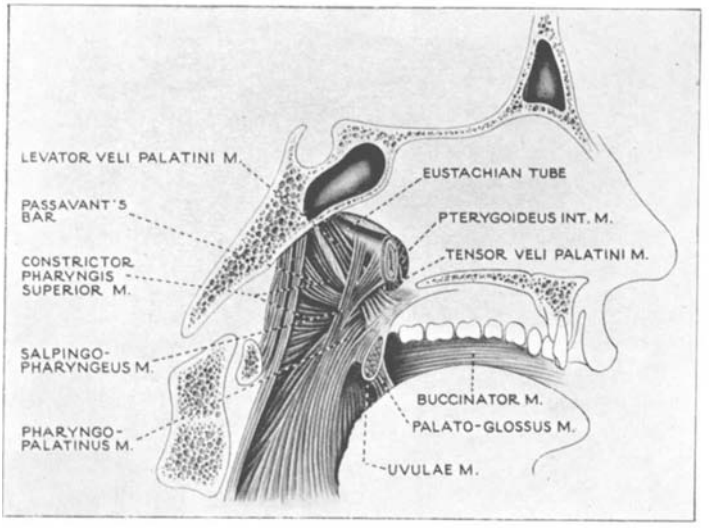


FIG. 2.

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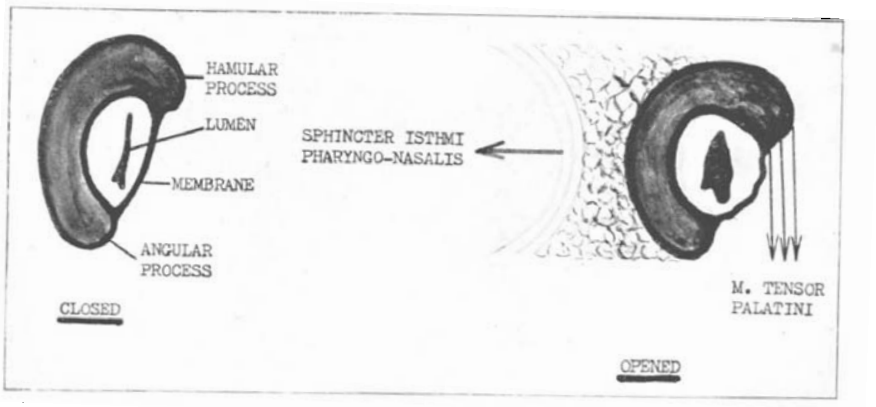


FIG. 3.
Mechanism of tubal opening.

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pharyngo-nasal sphincter; and that certain physiological factors such as posture and exercise influence its patency.

Closure of the tube in the majority of individuals is passive and due to the elasticity of the cartilage.

Ventilation of the Middle Ear During Flight

During ascent in aircraft or decrease of pressure in a chamber, as the extratympanic pressure becomes less, egress of air from the middle ear along a normal Eustachian tube is *easy and automatic*. Most aviators are aware of periodic and involuntary escape of air from their Eustachian tubes. This was first described in 1879 (Hartmann) and more recently by Armstrong and Heim (1937). As a result of experiments they state that at 500 ft. (15 mm.Hg.) there is a " click " and air escapes from the Eustachian tube leaving a residual positive intratympanic pressure of 3·6 mm.Hg. and that this escape recurs once at an average ascent of 425 ft.

During loss of height in aircraft or increase of pressure in a chamber, the Eustachian tube behaves in an entirely different manner; for, even with a normal tube, air does *not* enter the middle ear automatically. In the majority of aviators, however, the tube is opened and ventilation of the middle ear brought about by involuntary swallowing movements which occur at the rate of once every sixty to seventy-five seconds in normal individuals; and in addition all aircrew personnel are taught to practise auto-inflation at every 1,000 ft. loss of height.

Non-ventilation of the middle ear during descent results from either:

(1) *Failure to open the tube*, or (2) *Inability to open the tube*.

(1) *Failure to open the tube* may be due to:

(*a*) *Ignorance* as to the correct means to achieve patency.

(*b*) *Lack of opportunity* to open the tube, and this has been responsible when energetic evasive action to escape searchlights or anti-aircraft shells has been necessary. *Rapidity of descent per se* is *not a cause of non-ventilation* and is of importance only in this respect. In a large number of patients of this series symptoms became manifest during a *gradual* loss of height from 14,000 ft. or 12,000 ft. when returning from an operational flight.

(2) *Inability to open the tube* owing to the presence of some existing tubal abnormality may have occurred, but in two

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patients only was œdema of the tubal ostium observed and in one an old injury of the torus.

Table II is an analysis of the upper respiratory abnormalities noted in the present series.

TABLE II.

EXISTING LESIONS OF UPPER RESPIRATORY TRACT.	
"Cold"	6
Deviated nasal septum 18:	
Operation performed	8
Operation not advised	10
Chronic infection of adenoid	2
Chronic tonsillitis	7
Antral infection	10
Dental lesion	1
REMOTE LESION OF UPPER RESPIRATORY TRACT.	
History of earache and/or otorrhœa ..	10
History of myringotomy	1
Previous cortical mastoidectomy ..	4
Previous injury of Eustachian orifice ..	1

It will be seen that of these—six patients were suffering from coryza; eight showed gross septal deformities; nineteen had infected tonsils, adenoid or antra; and one an old injury—i.e. one-third only of 100 patients were suffering from lesions which *might* have affected the lining of the tube.

If ventilation of the middle ear does not take place *from any cause* during loss of height a positive pressure, depending on the amount of loss and the altitude at which it occurs, develops outside the middle-ear cleft. Armstrong and Heim (1937) have shown that an extratympanic positive pressure of 90 mm.Hg. or more will cause the tube to "lock", as the muscles concerned are unable to overcome this force (Fig. 4).

When one considers the cartilaginous portion of a Eustachian tube it is realized how easily its cartilaginous and soft membranous walls can be pressed together, completing the impermeability of what is already only a potential lumen.

This occlusion due to pressure I have regarded as *Primary (Pressure) Occlusion*; and this partially explains the obstruction. If this were all, however, it could be relieved by one of the recognized forms of inflation, and in many patients such relief has been achieved when the treatment has been applied within a *short time* of the onset of symptoms.

If, however, "forced ventilation" of the middle ear is not available or is neglected for one to two hours, the tube becomes

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impermeable to air even at high pressures, fluids and sounds as mentioned earlier.

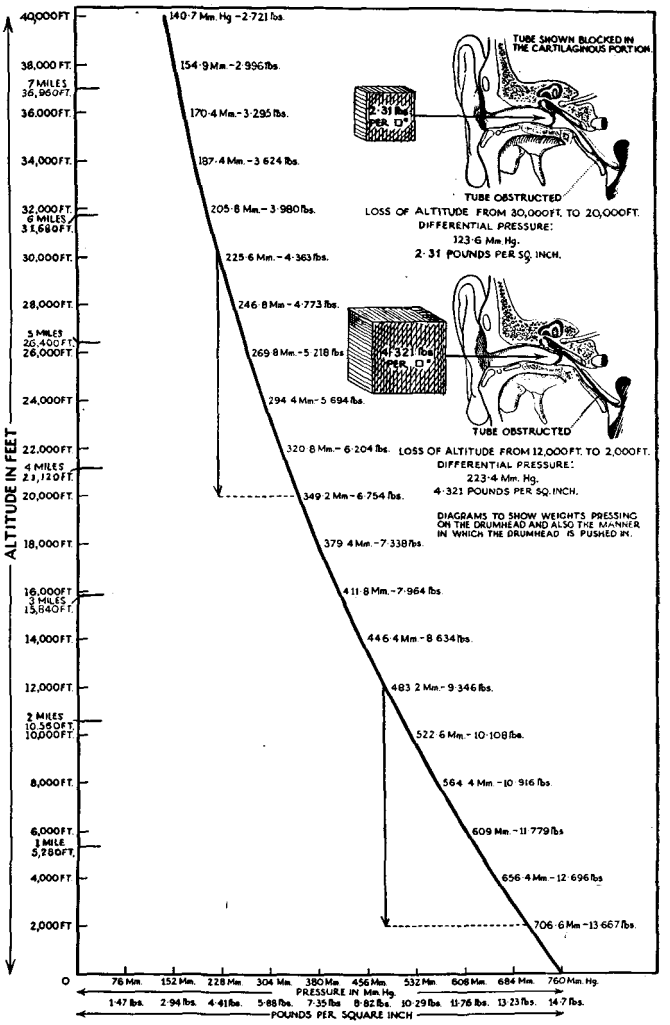


FIG. 4.

To demonstrate the amount of positive extratympanic pressure developed by equal loss of height at different altitudes.

It is reasonable to assume therefore that some biological change has taken place as a result of the primary (pressure) occlusion, and this I have called *Secondary (Vital) Occlusion.*

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The nature of these secondary changes is not known but it can be surmised when one views the thickness of the submucosa and the large numbers of mucous glands and goblet cells which are present in the epithelial lining of the cartilaginous tube.

Secondary occlusion is a *very real* entity and it may take as long as fourteen weeks to resolve, as happened in one patient of this series.

The ætiology of this type of aural defect appears to be :

Failure or inability to open the tube → Primary Pressure Occlusion → Secondary vital occlusion.

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