CHAPTER 8.

FAILURE OF THE EUSTACHIAN TUBE

INTRODUCTION

The middle ear may be compared with a watch, with the drum on the outer face and the cochlea medially. Between lies the air-filled middle ear containing the Ossicular chain. The aeration of the middle ear permits the chain to vibrate freely, transmitting sound to best advantage. The air is soluble, gradually dissipating into the surrounding bloodstream, therefore constant replenishment is required, via the Eustachian tube.

Figure 1: Normal Eustachian tubal function. Air passes into the middle ear and mastoid air cell system and is resorbed into the surrounding bloodstream.

Were the tube to remain constantly open, the patient would be subject to autophony and bodily noises that would impede hearing. Infection would have open access to the sensitive middle ear epithelium. The tube is therefore shut, opening only at deglutition other palatal tension situations. This opening is automatic, as the individual swallows with great frequency through the day to clear saliva from the pharynx.

The tube is normally a sleeve of epithelium, applied to the cartilage. The latter folds over upon deglutition, from the action of the palatal muscles. This opens the tubal lumen and permits air entry into the middle ear.

2. AETIOLOGY OF TUBAL FAILURE

Common origins

a) Idiopathic
b) Cleft palate
c) Infection
d) Allergy
e) Barotrauma
f) Carcinoma
g) Physical obstruction
b) Cleft Palate

Congenital palatal deformity, primarily cleft palate, is a major cause of long-lasting tubal dysfunction. This may arise from several origins:

i. Loss of the midline raphe anchorage of the soft palate musculature

ii. Congenitally absent musculature.

iii. Nerve supply deficient or traumatised during surgery.

iv. Surgical trauma to the pterygoid hamulus pulley mechanism.

Figure 3: The aetiology of Eustachian insufficiency in cleft palate cases. The cleft palate eliminates the inferior anchorage of the tensor palate in the midline raphe. The tensor is deprived of anchorage inferiorly, and is unable to actively open the Eustachian tube by pulling down the tubal cartilage. Tc: tubal cartilage; tp: tensor palate; ph: pterygoid hamulus.

Figure 4: Bifid uvula. Possibly associated with a submucosal muscular dehiscence causing tubal insufficiency.

Substantial numbers of cleft palate cases suffer chronic tubal insufficiency and its consequences. Long term follow-up of the afflicted is prudent, but despite best efforts, the hearing may decline, necessitating surgery or aiding. The new generation of active implants are valuable in many instances.

c) Infection

Infection is a common cause, most likely acting by producing oedema of the tubal lining that impedes adequate opening. A preceding history of coryza, influenza or sinusitis is common. In addition, after acute bacterial otitis media, retained toxins in the middle ear exudate appear to perpetuate similar oedema and dysfunction.
d) Allergy
The role of nasal allergy is less clear, but allergic oedema of the tubal lining also appears to be contributory in some cases.

e) Barotrauma
Barotrauma results from sudden pressure changes in which the pressure in the middle ear fails to adjust to the external increase, e.g. during diving, or descent in aircraft. The drum is stretched under tension, bruising or bleeding. A serosanguinous effusion fills the ear and may take weeks to resolve.

f) Carcinoma
Carcinoma of the postnasal space is a sinister cause of tubal failure, the lesion invading the

Musculature or occluding the lumen. This problem is endemic in people of the southern China area ancestry. Unilateral middle ear effusion in such circumstances dictates thorough evaluation of the postnasal space by endoscopy and radiology.

g) Physical Obstruction
Uncommonly, nasal obstruction due to a deflected nasal septum may cause problems by occluding the tubal orifice during other events, such as viral URTI.
3. SEQUELAE OF TUBAL FAILURE

Patterns of Complications

a) Middle Ear Effusion
b) Adhesive Otitis (Drum Atelectasis)
c) Tympanosclerosis
d) Cholesterol Granuloma
e) Chronic Otitis Media
f) Obliterative Fibrosis
g) Cholesteatoma

3.1. MIDDLE EAR EFFUSION

a) Pathogenesis

Tubal failure isolates the air of the middle ear cleft from re-supply via the tube. With time the air in the cleft diffuses into the surrounding tissues, resulting in reduced pressure in the middle ear. This retracts the drum and causes seepage of honey-coloured serous fluid into the middle ear, gradually filling this space. With time, the linings of the cleft become oedematous, and the epithelium undergoes a partial metaplasia with the development of respiratory epithelium.
Figure 8: Persistent middle ear effusion results in a serous transudate into the middle ear replacing the aerated cleft with a fluid-filled cavity.

The goblet cells of this change gradually thicken the effusion, the fluid becoming more mucoid, and the drum colour changing from the initial yellow tones to a grey or waxy appearance.

If the problem begins with an acute bacterial otitis media, the infection itself induces oedema of the tubal lining and resultant obstruction. In this situation, retained toxins in the effusion may perpetuate the problem.

i. Symptoms

Pain may be present at onset, or a child may have a history of intermittent Otalgia. The pain is due to tension on the drum and is similar to that experienced when diving without equalising. Conversely, the problem may be of silent onset in children, being often missed for some time. After barotrauma stuffiness may be the predominant symptom.

Discharge may occur if the problem is initiated by acute otitis media, or after severe barotrauma.

Deafness is a predominant symptom, sufficient to cause noticeable problems in children.

Tinnitus, usually noted by adults, is popping or gurgling in nature. Physiological or pre-existent pathological tinnitus may be enhanced by the exclusion of everyday environmental noise. Complaint is uncommon in children.

ii. Signs

Accurate diagnosis of tubal failure and its sequelae demands a clear external canal and excellent auriscope or microscope lighting. As the drum may pass through a series of changes, some experience is needed to assess the nuances.

A red and retracted but aerated drum is seen in acute insufficiency. If unilateral, compare the appearance with the contralateral side.

A straw or honey coloured drum follows, as the cleft fills completely with the effusion.

Figure 9: Serous OME displaying the classic honey-coloured drum due to the transudate in the middle ear.

With time, the drum takes on a grey or waxy tone as oedema forms and the effusion becomes more mucoid. “Cartwheel vasculature” may be seen radiating from the umbo in chronic cases.
Figure 10: Grey mucoid OME, cartwheel vasculature present.

Bubbles, coalescing into fluid levels are seen during the initial effusion formation, then later during the resolution phase, when erythema is usually lesser.

Figure 11: Serous OME, resolution phase, showing bubbles and air in the middle ear.

iii. Investigation

Otolaryngological assessment is directed to seeking a cause in the URT or ear.

Radiology (plain films, CT or MRI) may be used to assess adenoidal hypertrophy, sinus infections or postnasal space neoplasms.

Audiology commonly reveals a conductive loss in the region of 20 db, tympanometry a type A flat curve (effusion) or a type C (substantial negative pressure, > -150 mm). Either test may be aberrant, but the combination is more suggestive.

Figure 12: Mild conductive deafness. The 25-30 db conductive losses are typical of the mild losses encountered in middle ear effusions.

iv. Management

Conservative management is initially considered in the absence of more pressing circumstances. Observation alone is recommended for several weeks, as the problem may resolve spontaneously. This may be combined if necessary, with treatment of an identified cause, e.g. sinusitis. The role of Valsalva self-insufflation is uncertain, although tubal cannulation for this role was practiced widely in the past. Antihistamines, topical steroids and decongestants may aid elimination of allergy and infection but are of dubious benefit to clear the tubal obstruction per se.

After failed conservative management, or for more rapid resolution, myringotomy and vent tube insertion offer prompt relief. In adults and some children, this can be done using a microdot of phenol applied to the drum as a topical anaesthetic. The phenol produces a tiny numb patch through which a radial incision and tubal insertion can be performed with very minimal discomfort. Children usually require general anaesthesia.
Figure 13: Middle ear vent tube (grommet) in situ. These alleviate deafness and pain, and stabilise the ear, but do not treat the cause.

Vent tubes are available in a considerable commercial range of models, designed to adapt to the demand of desired duration, effusion consistency, infection, or drum status. All tubes are meant to extrude with time, forming a “cuff” of keratin as this occurs. Tubes left in situ longer than two years risk drum weakening or perforation. This is a problem with some very chronic cases that may require repair and hearing aids in the longer term.

Figure 14: Mouthbreathing due to chronic adenoiditis. Check for OME.

Concurrent with the tube insertion, the patient may undergo adenoidectomy, plus other management such as tonsillectomy or sinus procedures. The role of adenoidectomy is controversial. Adenoiditis is causative in many cases, but the diagnosis of this influence is difficult, and many surgeons opt for their routine removal to ensure cure as best as possible at the one procedure.

Figure 15: Adenoidectomy, commonly undertaken to minimise nasopharyngeal infection.

v. Complications of Ventilation Tubes

Recurrent effusions after extrusion of initial tubes may require re-insertion with longer lasting models. Perhaps 10-15% of cases may require this.

Otorrhoea from vent tubes occurs frequently. Immediate problems may result from infection that antedates the insertion, or from surgically initiated infection.

Later onset may come from several sources. Water sports or bathing may permit soiling of the middle ear via the tube. Alternatively, a further virulent URTI may occur. The two may be difficult to separate, or a mixed URT (streptococcus, haemophilus, moraxella) and EAC (gram negatives) origin infection may be present. Given this possibility, it is prudent to treat for both with oral amoxicillin-clavulanic acid and ciprofloxacin drops.

A chronic otorrhoea from a vent tube frequently results from granulation formation around the tube. Treat with the above antibiotics, plus a
ciprofloxacin-impregnated wick for several days, to absorb discharge, then similar drops.

Stage 1: Collapse without substantial conductive loss. These cases require no care, or vent tube insertion if an effusion is present.

Occasionally, chronic mastoiditis may be present or occur later. Persistent otorrhoea may require mastoidectomy to achieve resolution.

Drum atrophy or perforation may complicate repeated tube insertions. Small areas of atrophy are common but usually asymptomatic. Advanced damage may require surgical repair with cartilage-reinforced grafts.

3.2 ADHESIVE OTITIS MEDIA
(COLLAPSED EARDRUM, DRUM ATELECTASIS)

Gradual collapse of the drum into the middle ear is a common outcome of chronic insufficiency. The collapse may be graded according to the clinical and management implications:

Stage I: Collapse, no intervention indicated.

Stage II: Collapse with significant conductive loss. Drum repair with a cartilage-perichondrial graft is needed to restore hearing.

Stage III: Collapse with Ossicular necrosis or fixation. Collapse of the drum is often followed by chain necrosis, initially the long process of the incus. The cause is uncertain, possibly pressure effects. Fixation may be due to scarring, tympanosclerosis or secondary ossification. Chain and drum repair will be needed.
3.3 TYMPANOSCLEROSIS

Calcific deposits in the drum or middle ear are a common occurrence. In the drum they represent past tubal or infection conditions, and often result from vent tube insertion. Small deposits are asymptomatic, but longer term tubal problems may cause substantial drum thickening and immobility. In chronic insufficiency or long term drum perforation cases, the calcified deposits may fix the Ossicular chain, causing advanced deafness. Repairs may involve myringectomy, difficult chain mobilisations, then tympanoplasty.

3.4. CHOLESTEROL GRANULOMA

Tubal insufficiency may cause repetitive bleeding in the middle ear and mastoid. This may result in the accumulation of blood breakdown products that excite a granulomatous reaction typified by blackish debris, cholesterol crystals and a mucoid effusion.

Figure 19: Stage III* atelectasis. Collapse, with necrosis of the long process of the incus. Effusion seen.

Stage IV: Cholesteatoma. Marked retraction may impede the normal self-cleaning ability of the drum skin. Failure results in accumulation of keratin, then further invagination into the middle ear and mastoid, forming an infected sac that erodes the surrounding tissues. This requires more advanced surgery, often mastoidectomy, drum and chain repair.

Figure 20: Impending Stage IV collapse. Early Keratin accumulation indicates the onset of cholesteatomatous disease.

If any stage has an effusion present, the long term hearing prospects may be poor. To indicate this, an asterisk* is appended; Stage III* will therefore indicate drum collapse, chain damage, plus an effusion that may indicate a poor hearing prognosis if a tympanoplasty is considered.

Figure 21: Black drum due to blood breakdown debris in the middle ear. Cholesterol granuloma possible.

Inspection shows a blackened drum (idiopathic haemotympanum) and myringotmy, the mucoid
effusion. The problem often requires mastoid surgery to eliminate the deafness, but recurrence is common.

3.5 CHRONIC OTITIS MEDIA

Chronic drum tension from the dysfunctional tube stretches the pars tensa producing atrophic areas that are prone to perforation. Chronic infection and further damage to the middle ear commonly follows. Repairs with cartilage-perichondrial grafts are prudent when evidence of pre-existent drum collapse is present. To prevent re-collapse should tubal dysfunction continue.

Figure 22: Chronic Otitis Media. Collapse and perforation of the drum has been managed with a cartilage and perichondrial graft to prevent re-collapse and re-perforation.

3.6 OBLITERATIVE FIBROSIS

Chronic tubal failure is frequently associated with repeated middle ear infections that resolve slowly. Gradual obliteration of the middle ear cleft may result.