Dizziness and Vertigo

Description
True vertigo is most often associated with a sensation of ‘spinning’ and movement of the surrounding environment. It is important to distinguish this from the more generalised dizziness of disequilibrium.

Epidemiology
Male: Female ratio 1 : 3

Causes and Differential Diagnoses
Once you have established that the symptom is that of true vertigo, it is imperative to ascertain the duration and frequency of attacks, as this is the key to reaching the correct diagnosis and determining if the disorder is most likely peripheral (pertaining to the ear) or central (brain).

There are 3 common causes of vertigo originating from the labyrinth itself:

- Benign Paroxysmal Positional Vertigo (BPPV)- most common cause of true vertigo with typical age of onset 40-60 years
- Vestibular neuronitis
- Meniere’s Disease

These can generally be differentiated by the duration or onset of the vertigo and by the presence or absence of associated audiovestibular symptoms

Another common condition that is seen is vestibular migraine. Symptoms do not always include a headache and/or visual

symptoms and can sometimes overlap (e.g. hearing loss) making it difficult to differentiate between conditions such as Menieres.

	Onset	Hearing loss
BPPV	Sudden	No
Vestibular neuritis	Sudden or Gradual	No
Meniere's	Gradual	Yes-fluctuating
Vestibular migraine	Sudden or Gradual	Sometimes

Signs and Symptoms

- BPPV- Dix-Hallpike test positive. Rotatory vertigo on moving head
- Meniere's – Rotatory vertigo associated with fluctuating hearing loss often with low frequency thresholds affected. Tinnitus usually gets worse during an attack. Patients classically get an aural fullness before onset of vertigo.
- Vestibular neuritis – Rotatory vertigo that is continuous for over 24 hours often associated with nausea and vomiting. Classically they are confined to bed and it takes several days to weeks to recover.
- Vestibular migraine – Rotatory vertigo can last minutes to hours to days. Classically associated with headaches/photophobia/visual disturbance\phonophobia but these are not always present.

Investigations

- Full neurological examination
- Pure tone audiometry
- Dix-Hallpike test
- MRI of internal auditory meatus may be appropriate with asymmetrical sensorineural loss to exclude an acoustic neuroma
- Video head impulse testing (vHiT) – this is performed using specialist equipment and can be used to assess the function of

the semi-circular canals by measuring visual ocular reflex (VOR) function. It takes around 15minutes to perform and is a quick and sensitive measure of labrythine function

Treatment

- BPPV - Epley's manoeuvre can be curative in up to 90% by repositioning of the displaced otoconia crystals. In persistent cases, Brandt-Daroff exercises may be advised. Surgical management is rarely required but posterior semi-circular canal occlusion is useful in resistant cases.
- Vestibular neuronitis – Treatment is expectant with anti-emetics during the acute phase
- Meniere's Disease – There is a hierarchy of treatments depending on the severity of the disease and response to previous treatments. The underlying pathophysiology is thought to be endolymphatic hydrops. Therefore "pressure reducing" therapies include low salt diet, medications such as betahistine and diuretics although the evidence for these treatments is weak. Intratympanic injection of steroid or gentamicin is used for those that fail conservative management. Other treatment options include saccus decompression, labyrinthectomy and vestibular nerve section.
- Vestibular migraine – Common trigger factors include dehydration, foods (classically chocolate, cheese), anxiety and a poor sleep pattern. A symptom diary can help identify these. In those that do not respond to avoidance measures, there are a variety of migraine-preventative medications available.

Hearing Loss

Description

Hearing loss may be unilateral or bilateral.

Causes of Hearing Loss

Conductive Hearing Loss

	Additional Symptoms	Signs	Investigations
Excessive earwax (must occlude canal)	Blocked feeling	Wax on otoscopy	n/a
Otitis media with effusion (OME)	Popping, clicking/ pressure	Dull TM/ Fluid level/ bubbles on otoscopy	Tympanogram will show flat trace
TM perforation	May have middle ear discharge if active infection	TM perforation	n/a
Otosclerosis	Can be unilateral or bilateral	Usually none. Schwartz sign - red tinge to TM on otoscopy due to vessel injection on promontory (cochlear otosclerosis)	CT, PTA - 2kHz raised BC threshold (Carhart notch)
Cholesteatoma	Chronic smelly discharging ear	Deep retraction pocket with keratin collection	CT - assess extent of disease.

Sensorineural Hearing Loss

	Symptoms	Signs	Investigations
Presbyaucusis	Bilateral gradual onset	Normal otoscopy	PTA
Noise induced (NIHL)	Often tinnitus	Normal otoscopy	PTA- raised thresholds at 4kHz
Vestibular schwannoma (acoustic neuroma)	Asymmetrical hearing loss	Normal otoscopy	MRI
Complication of meningitis	Important to exclude in children who have had meningitis	Normal otoscopy	MRI may identify labyrinthine obliteration
Acute sensorineural loss	May have tinnitus and vertigo	Normal otoscopy	MRI, autoimmune screen

History

- Sudden vs gradual onset. Unilateral or bilateral.
- Associated otological or neuro-otological symptoms

Investigations

Pure Tone Audiogram (PTA) & Tuning fork tests (Rinnes & Weber) are complimentary to each other – should always be used together.

Management of Hearing Loss

Audiological

- Hearing aids for mild-to-profound hearing loss

Surgical

- Tympanoplasty - Cartilage or temporalis fascia is used to repair a perforation in tympanic membrane. N.B. This surgery is normally done for recurrent ear infections or to waterproof the

ear; hearing improvement often occurs when a perforation is closed but cannot be guaranteed.

- Stapedectomy - Prosthesis used to bypass fixed stapes/footplate in otosclerosis and allow transmission of sound into inner ear
- Bone anchored hearing aid – a transcutaneous or percutaneous device can be surgically implanted under general or local anaesthesia for a conductive, mixed conductive /sensorineural hearing loss or unilateral dead ear
- Cochlear implantation- There are specific NICE criteria for cochlear implantation which includes profound sensorineural hearing loss. However it may be of benefit in other patients – they require a multidisciplinary team assessment.
- Middle ear implant – suitable for conductive and mixed hearing loss

Management of excessive ear wax

Method	Uses
Topical eardrops - Warm olive oil, sodium bicarbonate.	Soften impacted earwax allowing it to migrate naturally out of the canal
Microsuction	Evacuate softened wax and wax tightly adherent to the ear canal
Jobson Horne wax probe	Useful in slowly coaxing out wax that can be easily manipulated
Syringing	Sometimes performed in primary care setting

Tinnitus

Description

Tinnitus is a term used to describe the perception of sound when no external sound is present. It is sometimes described, as ‘the sound of silence’ because all people, if they are seated in a completely quiet soundproofed room, will hear tinnitus. This noise is usually masked by the environmental sounds. It is said to be objective when apparent to the examiner and subjective when apparent to the patient only (more common).

Epidemiology

No identifiable cause is found in most cases of tinnitus. It is often associated with hearing loss.

Types of Tinnitus

Non- pulsatile tinnitus is typically referred to as a false perception of sound that is heard by the affected individual only (subjective). It is often described as a buzzing, high-pitched tone or a clicking or popping.It can be associated with noise induced hearing loss, presbycusis, Meniere’s disease, head injury, otitis media and drug related causes (e.g. salicylates, nonsteroidal anti-inflammatory drugs, loop diuretics)

Pulsatile Tinnitus (4%) is defined by a sound heard by an individual that is synchronous with their heartbeat and is usually caused by turbulent blood flow that reaches the cochlear. It may be associated with a treatable cause. It can be classified according to the underlying causes which is usually vascular or non vascular.

Causes of Pulsatile Tinnitus

Vascular causes

- Atherosclerosis on the internal carotid artery. The eventual stenosis of the artery may predispose the individual to pulsatile tinnitus due to turbulent blood flow.
- Vascular malformations. Arterio- venous malformations/fistulas are abnormal communications between the arterial and venous system. It may be congenital or acquired- the latter being secondary to trauma or a result of venous sinus obstruction.
- Glomus tumours are rare hypervascular tumours arising from paraganglia cells. Glomus tympanicum are associated with Jacobson's nerve around the promontory in the middle ear. Glomus jugulare are found along the jugular bulb and they involve the skull base and may extend in the middle ear.
- Non-vascular causes

- Paget's Disease
- Otosclerosis
- Myoclonus. Myoclonus of the middle ear muscles or palatal muscles may cause objective tinnitus, which classically presents with a clicking noise rather than a vascular thrill.

Investigations

- If unilateral and associated with hearing loss, MRI should be performed to exclude an acoustic neuroma.
- Pulsatile tinnitus may be investigated using MR or CT angiography. Carotid duplex scanning may also be helpful if carotid artery stenosis is suspected. Arteriography is also helpful in a limited number of cases but is associated with a small risk of CVA.

Treatment

- The vast majority of time, the patient just needs reassurance that tinnitus is very common and that they will adapt to it. It

tends to be worse at quiet times (e.g. at night when trying to sleep) and worrying about it generally makes the tinnitus worse.

- Address any underlying cause of the tinnitus in appropriate cases e.g. hypertension, carotid stenosis, side effect of medications
- For selected patients, behavioural therapy can be provided by audiologist/hearing therapist who introduce coping strategies and tinnitus retraining therapy (TRT) . A noise generator can mask tinnitus if interfering with sleep
- A hearing aid may improve tinnitus if hearing loss is present through a masking effect

Facial Nerve Palsy

Description
Temporary or permanent paralysis of the facial nerve (CN VII)

Epidemiology

- Bell's palsy (idiopathic in origin) is most common but is a diagnosis of exclusion (15-40 cases/100,000)
- M: F, 1:1

Causes and Differential Diagnoses of Facial Nerve Palsy

Idiopathic	Bell's palsy
Trauma	Iatrogenic injury following surgery, temporal bone fracture
Infectious	Bacterial vs viral (Ramsay-Hunt syndrome – Varicella reactivation). Secondary to acute or chronic otitis media, malignant otitis externa
Neoplastic	Malignant parotid or temporal bone tumour, paraganglioma
Congenital	CHARGE syndrome (Coloboma of eye, Heart defects,Atresia of choana, Retardation of growth, Genital and/or urinary abnormalities, Ear abnormalities & deafness)
Systemic/ inflammatory	Sarcoidosis, Gullain-Barre Syndrome, Multiple sclerosis
Other	Cerebrovascular Accident

Symptoms

- Dry painful eye, especially, if eye closure is impaired
- Drooling from side of mouth and difficulties with eating
- Psychological disturbance

Signs

- Differentiate between upper and lower motor neurone (upper motor neuron has sparing of forehead)
- Test strength of each branch of the facial nerve using House-Brackmann classification of nerve palsy - Raise eyebrows, tightly close eyes, wriggle nose, puff out cheeks, show teeth.

House-Brackmann Grade	Observation (simplified)
I	Normal
II	Slight weakness
III	Complete eye closure. Obvious weakness but not disfiguring
IV	Incomplete eye closure. Obvious weakness and disfiguring asymmetry
V	Flicker of motion
VI	No movement

- Bell's phenomenon - White sclera visible as eyeball rolls upwards to protect cornea when eyelid does not close
- Otoscopy – cholesteatoma, Acute Otitis Media
- Head and neck examination - parotid tumour

Complications of Facial Nerve Palsy

- Corneal scarring - Blindness if eye care advice not given
- Wasting of facial muscles, synkinesis
- Psychological

Investigations

- Pure Tone Audiogram – Look for conductive hearing loss (cholesteatoma) or asymmetrical sensorineural hearing loss (cerebellopontine lesion e.g. acoustic neuroma)
- MRI scan if suspecting central cause

Treatment

- General: Eye care, Artificial tears/tape eyelid shut. Referral to ophthalmology.
- Medical: Bell’s palsy/Ramsay Hunt syndrome - Oral steroids and oral antivirals although the evidence for oral antivirals in Bell’s palsy is lacking. Treatment needs to be started within forty eight hours to be effective.
- Surgical (rarely indicated): Depends on cause but options include facial nerve grafting, facial re-animation if the function does not recover.

Otalgia

Description

Otalgia is ear pain that can originate from the ear itself or can also be referred from elsewhere in the head or neck (see referred otalgia below)

Epidemiology

This is a very common presentation to primary care especially in young children.

History

Symptoms	Signs	Diagnosis
Child with severe ear pain and preceding URTI	Erythema, bulging drum, febrile	Acute otitis media (AOM)
Severe pain. Often with preceding itch and contact with water (swimming)	Tender, narrow external auditory meatus and mucopus	Otitis externa
Elderly with severe pain and often known diabetes or other causes of immunocompromise	Floor of ear canal showing granulation, +/- cranial nerve palsies	Necrotising otitis externa (malignant otitis externa, skull base osteomyelitis)
Pain anterior to tragus and worse when eating	Normal eardrum, tender over Temperomandibular joint(TMJ) and misaligned/ clicking bite	TMJ dysfunction
Moderate/ severe intermittent pain	Normal eardrum and palpable mass in the head and neck	Referred pain - Look out for red flags

Referred Otolagia

Always ask about other general symptoms – There are several other causes of otalgia, not directly related to the ear (referred pain). It is important to enquire about dental, nasal and throat symptoms in order to identify these. It is particularly important to identify potential indicators of malignancy.

Pain referred to the ear is a well-documented phenomenon. Any pathology involving the cranial nerves V, VII, IX, and X and the upper cervical nerves C2 and C3 can cause the sensation of referred otalgia.

Trigeminal neuralgia is the most common cranial neuralgia linked to referred otalgia. Other causes of referred cranial neuralgia are described below.

Nerve	Possible causes of pain
Cranial Nerve V3 (most common) i.e. mandibular branch of Trigeminal nerve	TMJ Dysfunction Pathology involving the 3 major salivary glands Dental Abscess
Cranial Nerve V (maxillary branch of trigeminal nerve)	Mucosal inflammation involving the sinuses
Cranial Nerve IX (includes the Jacobson nerve branch)	Oropharyngeal pathology e.g. peritonsillar abscess, tonsillitis, oropharyngeal carcinoma
Cranial Nerve X (includes the Arnold nerve branch)	Laryngeal cancer
Cervical Nerve (C2 and C3)	Cervical spine disease e.g. cervical spondylosis

Otorrhoea

Description

The ear can discharge wax, pus, blood, mucus and even cerebrospinal fluid. Remember discharging wax should be reassured as normal.

The common bacterial pathogens in a discharging ear that can cause an infection include:

- Pseudomonas aeruginosa
- Staphylococcus aureus
- Proteus spp.
- Streptococcus pneumonia
- Haemophilus influenza
- Moraxella catarrhalis

In most patients with a discharging ear, the diagnosis can be made based on good history and examination. We will recap on a focused history taking for otorrhoea and then revise the possible differentials of this symptom in more detail.

History

- Duration of discharge – If chronic, think chronic otitis media including cholesteatoma especially if unilateral
- Is there associated otalgia (ear pain)?
- Associated fever or systemic symptoms indicates an infective aetiology
- Is there associated hearing loss or dizziness?
- Do not miss a history of putting foreign bodies in the ear especially in children

- Facial nerve palsy - May occur with acute or chronic otitis media especially if the facial nerve is dehiscent along its course in the middle ear (10% of the population)
- Check for history of trauma - CSF otorrhoea
- Has there been any recent history of topical antibiotics? This can in itself cause discharge or predispose to antifungal ear infections if there is prolonged usage

Differential diagnosis

Symptoms	Signs	Diagnosis
Itchy ear canal	Fluffy whitish yellow or green black coating of the canal	Fungal otitis externa
Recent URTI infection Deep severe ear pain which precedes discharge and improves after discharge appears	Mucoid ear discharge	Acute otitis media (AOM) +/- perforation
Itchy ear canal	Scanty, thin watery discharge External ear canal can be completed occluded with discharged and swelling	Otitis externa

Unilateral Severe ear pain	Foul smelling discharge Cranial nerve palsies	Necrotising otitis externa (malignant otitis externa)
Unilateral Chronic offensive smelling ear discharge & hearing loss	Ear drum retraction/ perforation with keratin accumulation	Cholesteatoma
History of trauma or skull base surgery	Clear, watery discharge	CSF otorrhoea

Otitis Externa

Description
Inflammation of the external auditory canal that can be acute or chronic

Epidemiology
An estimated 10% of people develop otitis externa in their lifetime and there is an increased risk after first episode. Hence, otitis externa is an extremely common presentation when on call for ENT or at the emergency ENT clinic.

Risk Factors

- Swimming
- Warm/humid climates
- Underlying skin conditions e.g. eczema
- Immunosuppression e.g. diabetes
- Trauma e.g. excessive cleaning or scratching
- Hearing aids that reduce ventilation or introduce infection into the canal

Causes

- Mostly bacterial: Pseudomonas aeruginosa, S. Epidermidis and S. Aureus.
- Otitis externa can be fungal. There is an increased risk after prolonged antibiotic courses.

Symptoms

- Otalgia (ear pain) - especially on movement of the pinna or jaw. Can be severe.
- Pruritus (itching)
- Discharge
- Hearing loss

Signs

- Pain on moving the pinna and/ or tragus. Pinna may be very tender indeed.
- If pain is out of proportion with examination findings, there is a history of diabetes or failure to respond to antibiotics, consider necrotising otitis externa which is a severe, potentially fatal progressive form of otitis externa. It is a non-neoplastic infection, which spreads to the bone and results in osteomyelitis of the lateral skull base. It can cause multiple lower cranial nerve palsies. It is almost exclusively unilateral.
- External auditory meatus swelling, erythema with purulent discharge (Figure 29)
- Tympanic membrane not always visible because of swelling
- Complete external auditory meatus obstruction possible
- If possible, examine the tympanic membrane for a perforation. It is possible to have a secondary otitis externa associated with otitis media

Complications

- Peri-auricular cellulitis
- Necrotising otitis externa

Investigations

- Swab the external ear canal for MC&S

- Urgent CT scan is required if necrotising otitis externa is suspected (Unilateral, severe ear pain, elderly and/or immunocompromised)

Treatment

- Aural toilet (microsuction)
- Topical antibiotic and steroid ear drops e.g. ciprofloxacin (provides anti-pseudomonal cover)
- Insertion of a Pope wick helps the antibiotic come into contact with the canal wall and stents open a severely oedematous ear canal. The Pope wick looks like a small thin sponge and expands in the ear canal when wet.
- Admit for diabetic control, IV antibiotics and regular aural toilet if suspecting necrotising otitis externa

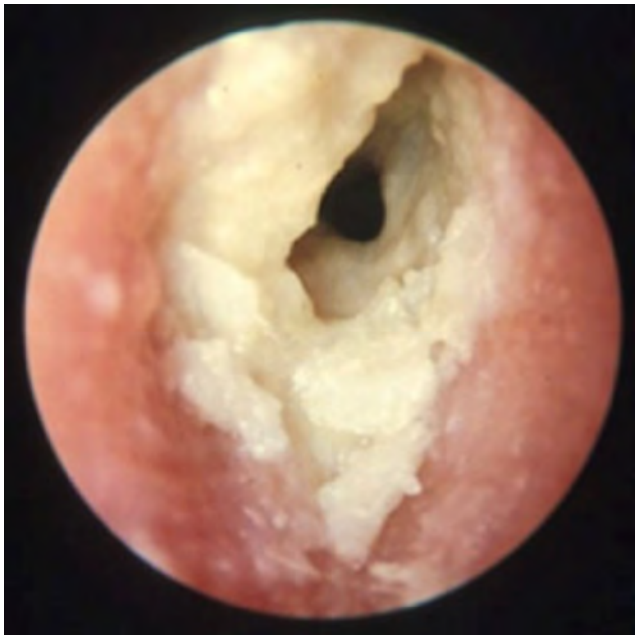

Figure 29: Otitis Externa (Courtesy of Elef-ENT)

Acute Otitis Media

Description
Acute otitis media is acute onset inflammation of the middle ear, usually of an infective origin. Otitis Media with Effusion (OME) may be a sequelea of acute otitis media but is regarded as a different, non-infective condition.

Epidemiology
Occurs at all ages, but much more common in infancy

Risk Factors

- Lack of breastfeeding as a baby
- Attending nursery/day care
- Positive family history
- Age between 6-18 months
- Exposure to smoking

Causes

- An upper respiratory tract infection (URTI) results in inflammation of the upper airways and swelling causes obstruction of the Eustachian tube. Ascending infection results in hyperaemia of the middle ear mucosa with production of a purulent exudate. This is called acute otitis media (AOM)
- Viral infections account for two thirds of cases of AOM. Respiratory syncytial virus, rhinovirus and enterovirus are the most common.
- Bacterial organisms include Streptococcus pneumoniae, Haemophilus influenza, and Moraxella catarrhalis.

Symptoms

- Infants: fever, ear pulling, irritability, vomiting
- Children and adults: otalgia (ear pain), fever, generally unwell, hearing loss. If the tympanic membrane bursts the pain may suddenly improve, but a purulent discharge develops from that ear

Signs

- Bulging tympanic membrane (Figure 30)
- Injected tympanic membrane
- If there is a perforation, there may be purulent discharge coming through. There may also be secondary otitis externa

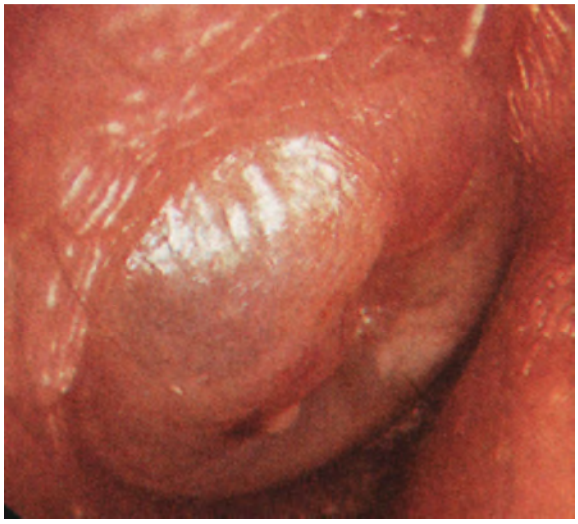

Figure 30: Bulging tympanic membrane in AOM (Courtesy of Elef-ENT)

Complications

- Intratemporal: tympanosclerosis (white patch on the ear drum due scarring), hearing loss, tympanic membrane perforation, mastoiditis, labyrinthitis, facial nerve palsy
- Intracranial: meningitis, intracranial abscess, lateral sinus thrombosis, cavernous sinus thrombosis, subdural empyema.

Investigations

- Consider swab for M, C &S if ear discharging
- Imaging (CT and/or MRI) if complications are suspected

Treatment

- Analgesia (e.g. Ibuprofen) and anti-pyretics (e.g. Paracetamol)
- If failure to improve within 24-48 hours, consider prescribing oral antibiotics. A 10 day course of Amoxicillin is first line. If no improvement, switch to Co-amoxiclav.

Otitis Media with Effusion (OME)

Description

This is a middle ear effusion (fluid) without the signs of infection. Also known as ‘glue ear’.

Epidemiology

- Bimodal distribution at a peak at 2 years and 5 years of age. Prevelance is 20% and 15% respectively.
- 50% of OME resolves spontaneously within 3 months

Causes

- Eustachian tube dysfunction. In children, the Eusatachian tube is smaller and more horizontal than in adults therefore middle ear ventilation is impaired. Thus commoner in cleft palate and other syndromic diseases affecting the face and skull base.
- Beware of the adult with a unilateral middle ear effusion. Nasopharyngeal tumours can block the drainage of the Eustachian tube and result in a middle ear effusion

Symptoms

May be asymptomatic in an infant. Parent may notice the child has hearing loss or behavioural problems.

Signs

- Poor speech development
- Otoscopy: tympanic membrane will appear dull +/- a visible fluid level. If pneumatic otoscopy is performed the tympanic membrane will have poor compliance (Figure 31)

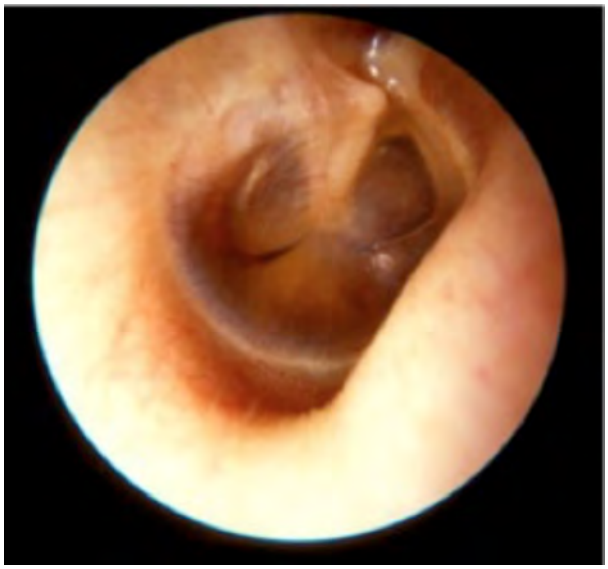

Figure 31: Dull right tympanic membrane with visible fluid level (Courtesy of Elef-ENT)

Investigations

- Pure tone audiogram – this will reveal a conductive hearing loss (i.e. air bone gap on PTA)
- Tympanometry will show a flat trace due to the reduced compliance of the tympanic membrane (i.e. ‘type b’ curve)
- In an adult with a unilateral middle ear effusion ensure that flexible nasoendoscopy (FNE) is performed to rule out a nasopharyngeal tumour

Treatment

- Antibiotics are not advised as they have no benefit in OME

- Watch and wait – 50% of OME will resolve spontaneously within 3 months
- Hearing aid may be useful whilst waiting for the OME to spontaneously resolve
- Myringotomy and ventilation tube insertion. This is a small incision in the tympanic membrane with the insertion of a small ventilation tube. Once inserted, the grommet will usually self extrude after around 9 months. Grommet insertion can lead to tympanosclerosis (scarring of the tympanic membrane) and tympanic membrane perforation (if the TM fails to heal after the grommet extrudes N.B. more common if history of multiple grommet insertion)
- Adenoidectomy may reduce the recurrence rate of OME

Chronic Otitis Media

Description

There are two types of chronic otitis media:

- Mucosal: A tympanic membrane perforation in the presence of recurrent or persistent ear infection.
- Squamous: Gross retraction of the tympanic membrane with formation of a keratin collection. (cholesteatoma)

The disease may be active (infection present) or inactive (no infection present):

- Inactive mucosal: Dry perforation
- Inactive squamous: Retraction pocket, which has the potential to become active with retained debris (keratin)
- Active mucosal: Wet perforation with inflamed middle ear mucosa and discharge
- Active squamous: Cholesteatoma

Mucosal Chronic Otitis Media

Epidemiology

- May occur in children and adults
- Causes
- Chronic infection following development of a post-infective, traumatic or iatrogenic perforation
- Pathogens: most commonly Pseudomonas aeruginosa, Staphylococcus Aureus

Symptoms

- Hearing loss
- Otorrhoea

Signs

- Mucosal inactive: Dry perforation (Figure 32)
- Mucosal active: Wet perforation with middle ear inflammation
- Otorrhoea: May be present in active disease
- May develop secondary otitis externa due to the discharge

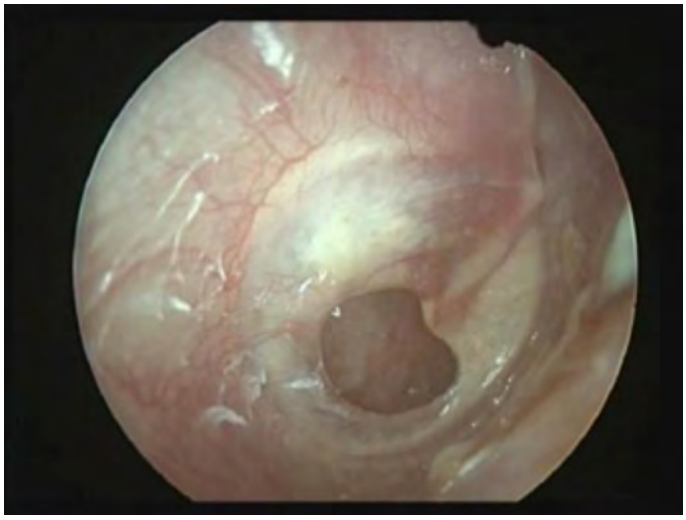

Figure 32: Right TM perforation (Courtesy of Elef-ENT)

Complications

As for acute otitis media.

Investigations

- Pure tone audiometry
- Ear swab for MC&S

Treatment

- Aural toilet with microsuction ensuring that the whole tympanic membrane is visualised

- Antibiotic and steroid combination ear drops if infection is present
- Myringoplasty (also known as Type 1 tympanoplasty). This is the surgical repair of the tympanic membrane perforation using cartilage or fascia. It is indicated to prevent recurrent otitis media.

Cholesteatoma

Description

Despite the name, a cholesteatoma is neither a tumour nor does it consist of cholesterol. Instead, it is an accumulation of benign keratinizing squamous cells which most commonly involves the middle ear. The squamous cells are hyperproliferating and secrete enzymes, which can be locally destructive (with potentially serious complications). It is commonly described as ‘skin in the wrong place’.

Epidemiology

Can occur in both children and adults but it usually has it’s origins in childhood chronic Eustachian tube dysfunction although congenital cholesteatoma accounts for around 5% of cases (see below). It is more common in males.

Causes

Congenital cholesteatoma results from persistent epithelial cell rests left within the middle ear during growth of the embryo and is diagnosed in children who have an intact tympanic membrane and no history of ear surgery

Acquired cholesteatoma develops after birth. Eustachian tube dysfunction results in tympanic membrane retraction and once the retraction is deep enough keratin migration from the tympanic membrane to the external auditory canal can no longer occur and the trapped keratin develops into a cholesteatoma. Keratin debris can become infected and leads to a chronic ear discharge

Symptoms

- Persistent/recurrent ear discharge despite topical antibiotics

- Unilateral hearing loss

Signs

- Otoscopy should be performed and micro suction should be used to clear any wax to ensure that the whole TM is visualised paying particular attention to the superior part of the TM (attic area)
- There is a deep retraction pocket in the TM with keratinous debris within it. There may be granulations around the margins of the retraction pocket and it is very common for adjacent bony erosion to occur
- If there is secondary infection there will be discharge in the ear canal
- In congenital cholesteatoma, the tympanic membrane is usually intact but it may be bulging and a white pearly mass will be visible through the tympanic membrane

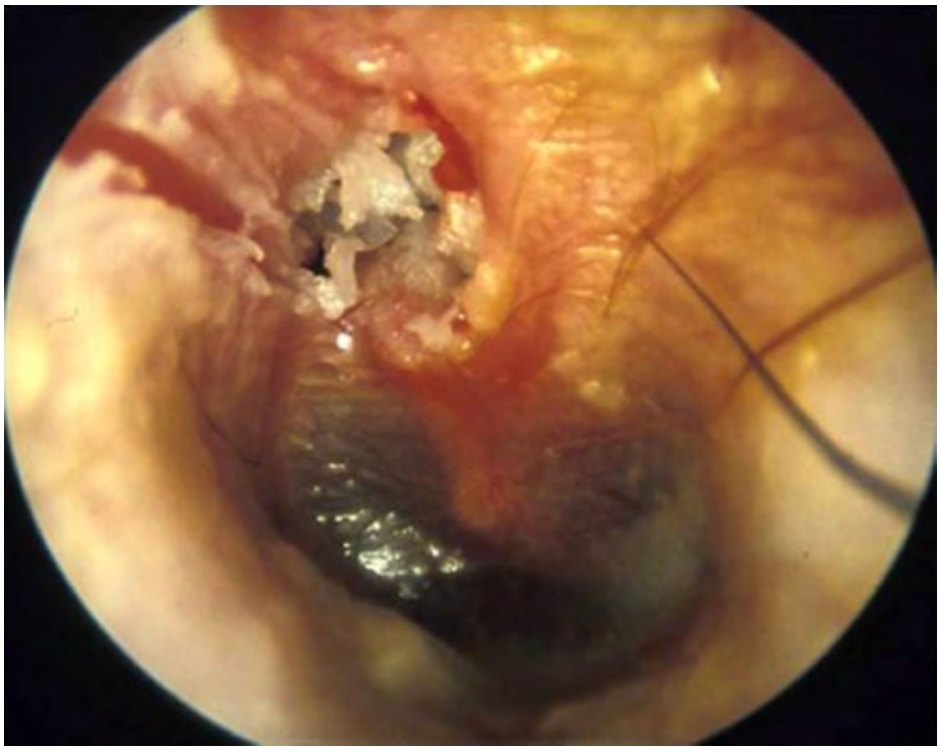

Figure 33: Active squamous chronic otitis media in left ear: attic retraction with retained squamous epithelial debris (cholesteatoma) (Courtesy of Elef-ENT)

Complications

As per acute otitis media (see above)

Investigations

- Pure tone audiometry
- CT scan of the temporal bone

Treatment

- Surgical management unless the patient is not fit for surgery
- The aim of surgery is to remove the cholesteatoma sac and repair the tympanic membrane and any adjacent bony defect, to prevent recurrent discharge. The mastoid cavity is drilled to allow access to the middle ear and all of the cholesteatoma is removed. If there has been destruction of the ossicles, hearing can be reconstructed (ossiculoplasty) using a variety of techniques and the tympanic membrane is replaced with a graft

Epistaxis

It is not a trivial condition and can potentially be life threatening. If the patient is actively bleeding, see them in A&E Resus, not in an isolated treatment room without resuscitation facilities/help.

History

You may have to take a focused history whilst simultaneously resuscitating and stabilising the patient (establishing intravenous access, sending off the pertinent bloods and setting up IV fluids).

Important points to remember are:

- Unilateral Predominance- which side did it start or more commonly comes from, which can then focus your assessment for the likely source.
- Anterior/Posterior – often patients can tell initially if it runs out the front first or down the back of the throat, which may guide further management
- Frequency – how often is it troubling them?
- How much blood loss has occurred – estimating blood loss can be notoriously difficult and bleeding from the nose can be very distressing. By focusing on common measures such as a teaspoon, a cupful, or a kidney dish full of blood can help prioritise your patient's resuscitation requirements.
- Co-morbidities such as hypertension, cardiac history, anti-coagulant use and previous nasal surgical history can all have an effect on patient management.
- Antecedents – traumatic bleeding can have a different anatomical source to other forms of bleeding
- Management techniques, risk factors, smoker, occupation, allergy to nuts

Examination

Most epistaxis occur in Little's Area (otherwise known as Kisselbach's plexus), located in the anterior part of the septum where an anastomosis occurs between the branches of the internal and external carotid arteries.

Initial management options for epistaxis include leaning forwards and pinching the soft part of the nose to apply pressure to the septum (not the bony part of the nasal bridge - Figure 37 and Figure 38). Sucking on ice cubes or applied to the forehead may help slow bleeding by causing vasoconstriction of the feeding vessels. The next step should ideally be identification of the bleeding point for cautery (see Cautery in Practical Procedures Chapter)

Figure 37: Incorrect method of pinching the nose.

Figure 38: Correct method of pinching the nose

Surgical Management of Epistaxis

If these measures fail to control the bleeding (or bleeding from more posteriorly in the nasal cavity is responsible), an endoscopic sphenopalatine artery ligation under general anaesthesia is undertaken (see Figure 39).
Traumatic epistaxis (for example from a sports injury) is usually anterior and related to the anterior ethmoidal artery. Ligation of this is often performed through an external incision at the medial aspect of the orbit.

When dealing with acute traumatic injuries of the nose, it is important to rule out a septal haematoma (Figure 40), which can starve the underlying septal cartilage of oxygen and cause ischaemic necrosis and cartilage loss, resulting in a saddle nose. It is therefore important to drain these at an early opportunity.

Trauma to the front of the nose may cause one or both nasal bones to be displaced. Assessment of any nasal vault deformity should be

made after 5 days once the swelling has subsided. There is a window of opportunity in the weeks after the injury for the nasal fracture to be reduced under local or general anaesthetic.

When the cartilaginous septum is fractured, deformed or displaced it may be corrected with a septoplasty (cartilage remodelling) procedure.

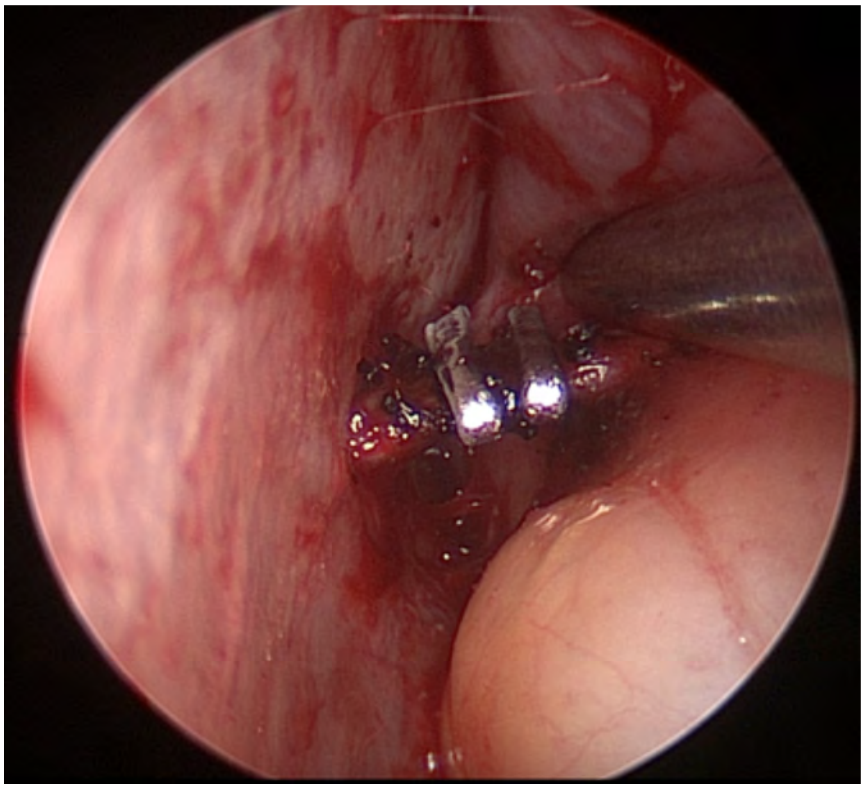

Figure 39: An endoscopic clinical photograph of the right Sphenopalatine artery being clipped in the back of the nose under general anaesthetic for epistaxis. 2 clips have been applied to the main branch.

Nasal obstruction

Causes of Nasal Obstruction

There are a number of causes for nasal obstruction, many of which are beyond the scope of this chapter. More common, acquired causes of nasal obstruction can be classified as seen below. A thorough history, good clinical examination and tailored investigations will often reveal the underlying aetiology:

Infectious – viral; bacterial or fungal infections causing rhinitis/rhinosinusitis
Allergy – inflammation with or without nasal polyps
Developmental - resulting in septal deviation, bony deviation or both; cleft lip
Traumatic – same as above (+/- septal haematomas, perforations, etc)
Iatrogenic – previous surgery (eg septoplasty) causing scar tissue (adhesions); residual septal deformities; mucocoeles
Drugs – chronic use of decongestants (rhinitis medicamentosa); side effects of medication (eg. Beta-Blockers, oral contraceptive pill); cocaine abuse resulting in vasculitis or septal perforation
Neoplastic – benign or malignant masses
Inflammatory/Systemic Diseases – eg. Granulomatosis with Polyangiitis (GPA); Eosinophilic Polyangiitis (EPA/Churg-Strauss); Sarcoidosis; Cystic Fibrosis; Kartaganer’s Disease

Fractured Nose

Nasal fractures do not need to be X-rayed.

If the patient is well and there is no epistaxis and no septal haematoma (a boggy swelling of the septum which is usually seen bilaterally and insensate when probed with a jobson-horn- see Figure 40) they can be sent home.

If there has been no change in shape and no new nasal obstruction since the injury the patient does not need to be seen by ENT and can be discharged with advice.

However, if the swelling and bruising are such that any deformity is obscured, then they should be reviewed in 5-7 days (once the swelling has subsided) in the ENT emergency clinic for assessment of the injury and discussion as to whether the patient wants to proceed to manipulation under anaesthetic.

The patients will need to be consented and listed for an MUA (manipulation under anaesthesia- Figure 41) after seeing them in the SHO emergency clinic. The common risks for the procedure include pain, bleeding, the need to pack the nose, the need to wear a splint, bruising and failure to get the nose back to its original shape.

If the clinician is confident, however, and the patient will tolerate it, a manipulation under local anaesthetic can be undertaken. Using a dental syringe infiltrate over the nasion down to bone and then either side of the nasal bones. Then firm pressure over the deformity will usually result in the bones being realigned into the midline. Patient selection is key to a successful procedure. They will need to wear a splint for 1 week which can be removed by the patient themselves or their GP practice nurse.

If a septal haematoma is present, the patient must have this drained (in theatre) as soon as possible to prevent infection and subsequent

destruction of the septal cartilage. This will lead to saddle deformity of the nose. They will need to be started on antibiotics to prevent secondary infection of the haematoma causing a septal abscess.

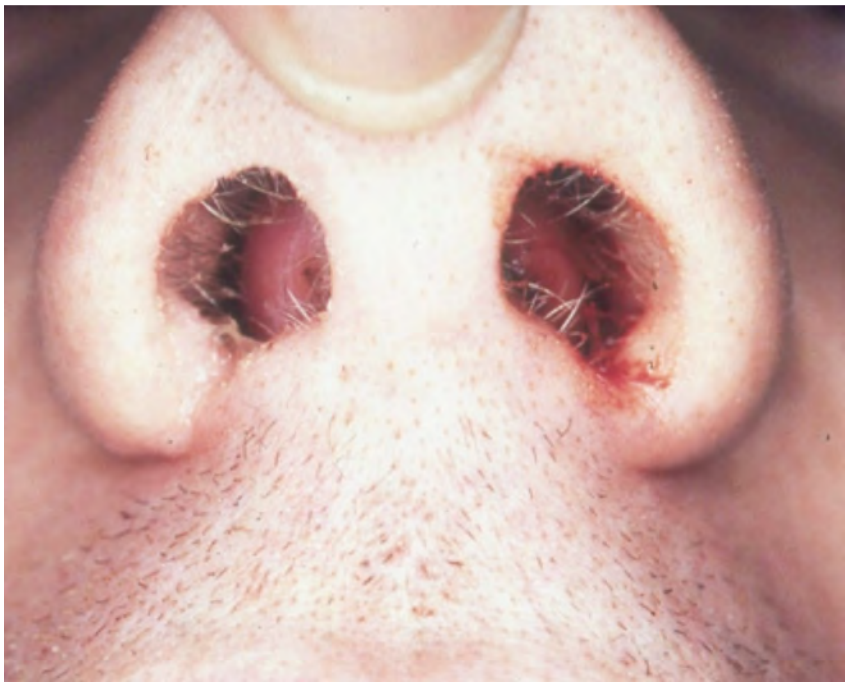

Figure 40: Septal Haematoma after nasal trauma. This requires urgent drainage to avoid complications such as abscess and septal cartilage necrosis.

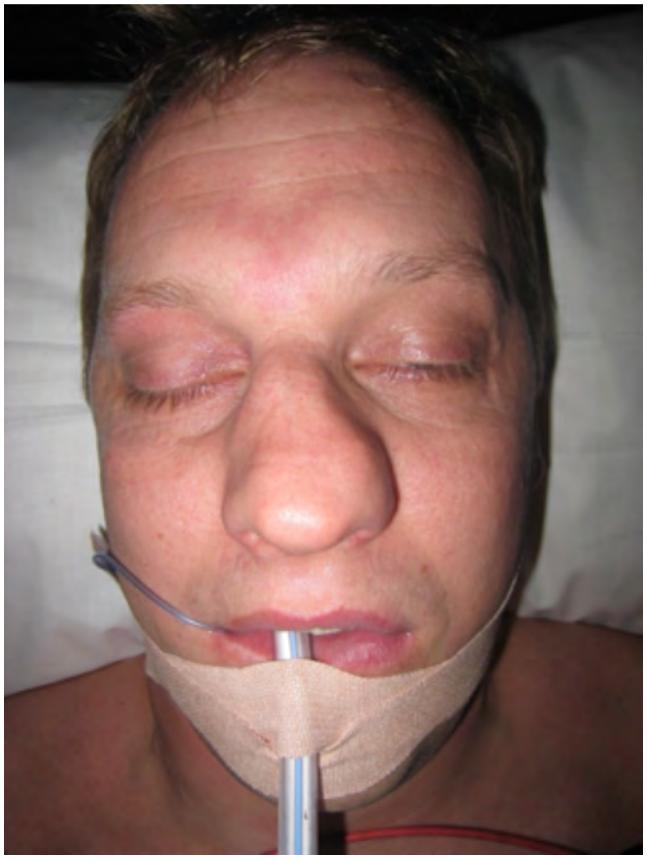

Figure 41: Pre-operative image of trauma resulting in significant nasal deformity.

Septoplasty/Septorhinoplasty

When the underlying midline cartilaginous or bony septum is deviated, a septoplasty can be undertaken to remodel and improve the functional nasal airway. It is often performed under general anaesthesia, as a day-case procedure. It can also be performed to gain better access to the nose for other endoscopic procedures e.g. limited access for epistaxis surgery or for septal cartilage harvest for graft harvesting.

A septorhinoplasty may be indicated if the septum and bony vault are deviated. This operation can be carried out via an external approach (via an incision in the columella) or endonasally. It can be performed for functional and cosmetic indications, and has important implications for both, so patients require appropriate pre-operative counselling.

Both of these procedures, if indicated, are not done acutely after the nasal injury and would be done on an elective list often 6 -12 months after the initial injury.

Rhinitis and Rhinosinusitis

Description

The term ‘rhinitis’ describes inflammation of the lining (mucous membranes) of the nose, characterized by nasal congestion, a runny nose, sneezing, itching and post-nasal drip. It can be usually divided into allergic and non-allergic causes.

The term ‘rhinosinusitis’ describes inflammation of the lining of the nose and paranasal sinuses. It has a number of manifestations, the commonest symptoms being anterior or posterior rhinorrhea (runny nose or post-nasal drip), nasal blockage/congestion or obstruction, and facial headache or reduction in sense of smell.

Allergic Rhinitis

Description

IgE-mediated Type 1- hypersensitivity reaction of the nasal mucosa. This condition can significantly affect quality of life with negative impacts on activities of daily living, school and work attendance.

Epidemiology

Allergic rhinitis (AR) is common and increasing in western populations. It can be associated with atopic disease such as eczema and asthma (1 in 3 patients with AR have asthma). There can be a family history noted.

Causes

- Intermittent (previously known as seasonal)- e.g. grass/tree pollen (hay fever) – late Spring/Summer.
- Persistent (previously known as perennial)- e.g. house dust mite, moulds, dogs, cats
- Food allergens causing rhinitis – this is a controversial area and not fully proven but may be a contributory factor

Symptoms

- Rhinorrhoea (seasonal)
- Nasal irritation /itching (seasonal)
- Sneezing (seasonal)
- Nasal obstruction (seasonal and perennial)
- Ocular symptoms e.g. itchy/watery eyes

Signs

Inflamed nasal turbinates and mucosa- bluish and pale hue to turbinate mucosa, watery nasal discharge and gross turbinate hypertrophy

Investigations

- Clinical diagnosis can be made with a combination of history & examination
- The mainstay of investigation is the skin-prick allergy test (SPT), which can be done cheaply and quickly in clinic. Positive (histamine) and negative (saline) controls are inserted into the skin (dermis), along with solutions of the various common inhaled aero-allergens (Figure 42), eg grass and tree pollen, dog and cat dander. A positive result is a wheal response after 20 minutes (Figure 43). Resuscitation equipment should be ready in case of anaphylaxis. Contra-indications include severe eczema/dermatographism or anaphylaxis. Patients should avoid taking anti-histamine tablets for 72 hours prior to the test in case they mask a response.
- Serum RAST (Radio-allergosorbent test) is a blood test to find specific IgE to an allergen. There is no risk of anaphylaxis, but this is more expensive and takes time for the results. This may be also useful in children who may not tolerate SPT.

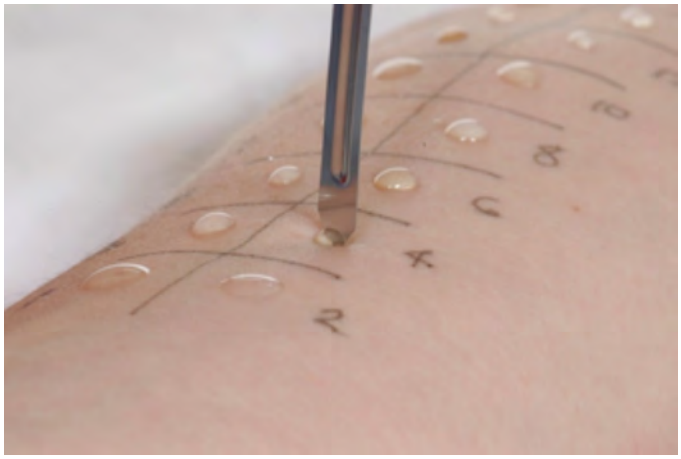

Figure 42: Skin Prick Allergy Testing (SPT) Droplets of various allergen solutions are placed onto the forearm and the skin is punctured

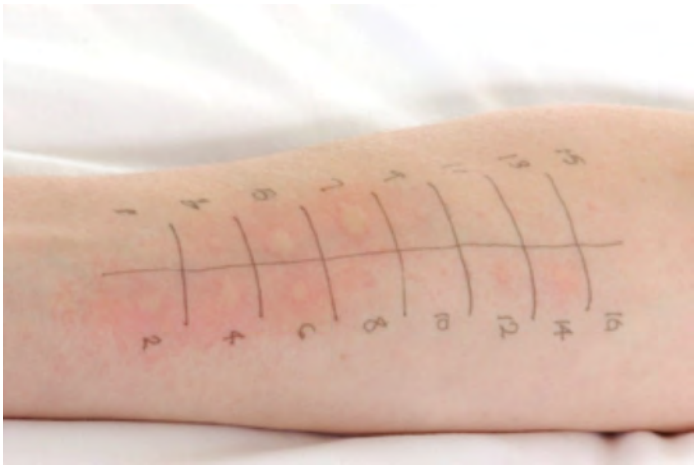

Figure 43: After 15-20 minutes the results can be shown by the positive wheal reaction

Treatment

- General advice- avoiding allergen exposure. E.g. washing bed linen at high temperature to reduce house dust mite allergen burden
- Nasal douching and barrier ointments/creams
- Oral non-sedating antihistamine, eg. Loratidine, Cetirizine, Fexofenadine
- Intra-nasal steroids e.g. Fluticasone (Flixonase or Avamys nasal spray), Beclomethasone (Beconase Nasal spray), or Mometasone (Nasonex Nasal spray) - 1 – 2 sprays, once or twice a day titrated to the patient’s symptoms for a minimum of 4-6 weeks. Check formulary for appropriate prescribing ages. Intra-nasal steroid sprays are suitable for long-term use if tolerated. Escalation to steroid drops or oral steroids can be considered on rare occasions for severe symptoms, but caution must be taken over longer courses due to the numerous systemic side-effects.

- Combination steroid and anti-histamine nasal sprays (eg. Dymista manufactured and distributed by Mylan®) – prescribed by specialists as 2nd line topical management for more effective relief in allergic rhinitis due to the synergistic combination of medications.
- Leukotriene antagonists e.g. montelukast, (orally) useful if patients also have asthma
- Immunotherapy- Desensitisation therapy works by gradually increasing the exposure to an antigen. It can be given sublingual or via subcutaneous injection. Immunotherapy to grass pollens and house dust mite is available in a few specialist centres.

Non-Allergic Rhinitis

Description
Inflammation of the nasal mucosa.

Epidemiology
Very common. Up to 50% of all cases of rhinitis in adults

Causes

- Irritants: tobacco, pollution, cleaning products (occupational)
- Vasomotor: temperature changes- especially cold, dry air
- Gustatory: Spicy food
- Pharmacological: rhinitis medicamentosa (rebound nasal congestion following prolonged use of topical decongestants), substance abuse- cocaine
- Infection
- Systemic- Granulomatosis with polyangitis (GPA), sarcoidosis.
- Physiological- exercise, positional, hormonal
- Atrophic rhinitis

Symptoms
Nasal congestion, rhinorrhea, post-nasal drip, hyposmia

Signs
Nasal congestion and hypertrophic turbinates

Investigations
Allergy testing to exclude allergic cause

Treatment

Non-surgical treatment. Non-surgical treatment should always be tried and is the mainstay of treatment.

- General advice- avoidance of trigger, eg smoking cessation.
- Nasal douches and intranasal steroids
- Other: Ipratropium. Decongestants (short-term only)
- Surgical treatment

- Surgery is not a permanent, curative procedure as the underlying pathology is to do with the lining of the nose. However, surgery may help improve the nasal airway and allow better delivery of topical nasal steroids, which the patient will need to continue postoperatively.
- Turbinate reduction surgery for obstructing inferior turbinates.
- Vidian Neurectomy – cutting the parasympathetic nerve supply to the nose (rarely indicated).

Acute Rhinosinusitis (ARS)

Description

Acute rhinosinusitis (ARS) is caused by symptomatic inflammation of the mucosal lining of the nasal cavity and paranasal sinuses.

There is sudden onset of two or more symptoms, one of which should be either nasal blockage/obstruction/congestion or nasal discharge (anterior/posterior nasal drip):
± facial pain/pressure
± reduction or loss of smell; for <12 weeks

Epidemiology

An average child is likely to have 6-8 colds (ie, upper respiratory tract infections) per year, but only approximately 0.5-2% of upper respiratory tract infections in adults and 6-13% of viral upper respiratory tract infections in children are complicated by the development of acute bacterial sinusitis. Therefore, antibiotics should be prescribed sparingly (see below for indications).

Causes

- Most commonly ARS is viral i.e. a common cold caused by Rhinovirus, Coronavirus, Parainfluenza virus or Respiratory Syncytial Virus (RSV).
- Streptococcus pneumonia, Haemophilus influenzae and Moraxella Catarrhalis account for the majority of bacterial causes.

Symptoms

- Main symptoms are nasal obstruction and coloured discharge

- Facial pain occurs more commonly in the acute presentation of rhinosinusistis
- Hyposmia/ anosmia (reduced/absent smell)
- Ask about pain elsewhere. e.g. dental infections can spread into the sinus
- Systemic features of fever and malaise
- Double sickening (deterioration after an initial milder phase of illness), which may indicate acute post-viral rhinosinusitis or bacterial rhinosinusitis.

Signs

- Facial tenderness can occur but is uncommon
- Anterior rhinoscopy- inflammation, discharge
- Nasendoscopy - pus discharging from sinus openings e.g. middle meatus (Figure 44)

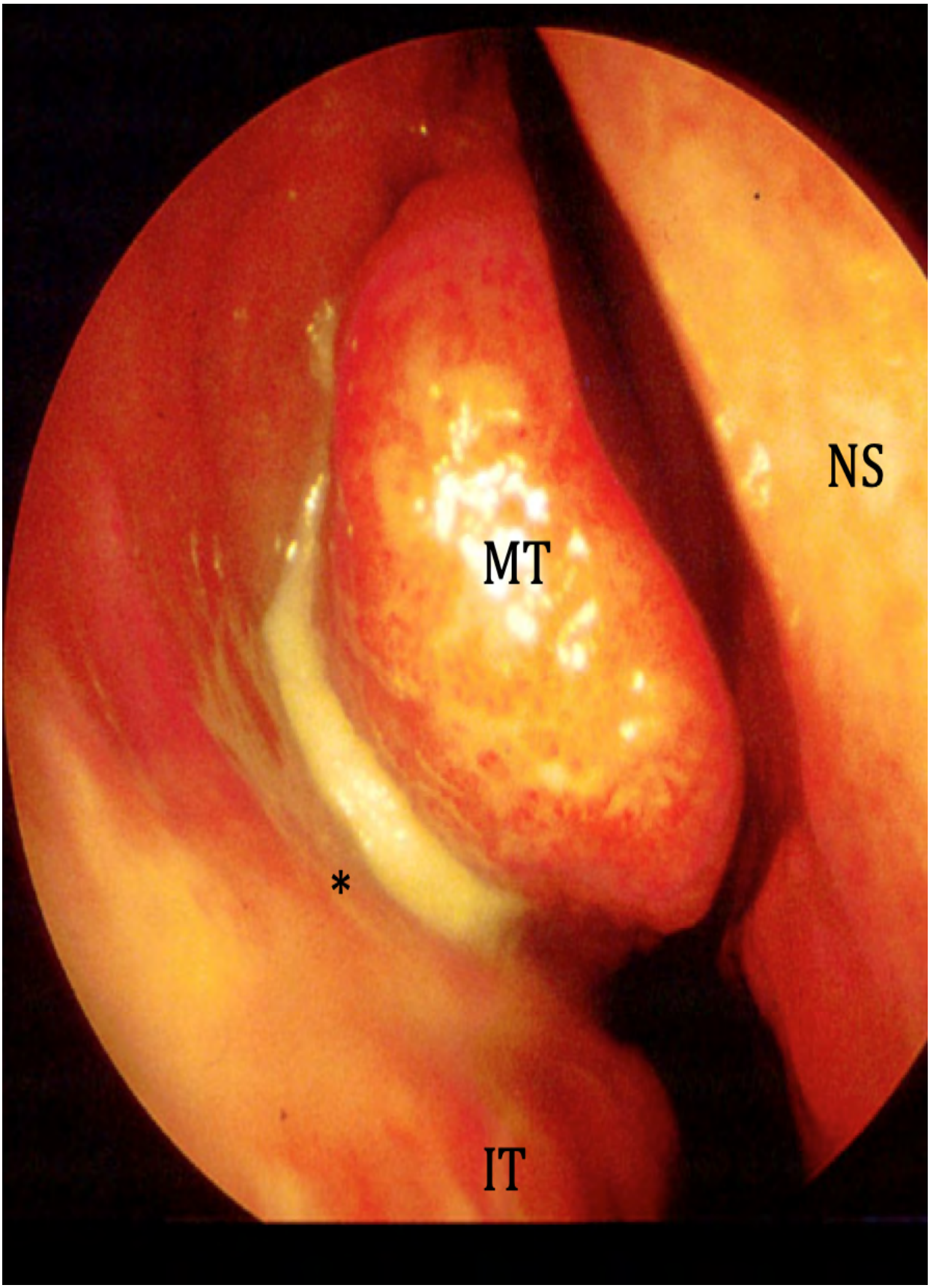
NS
MT
*
IT

Figure 44: Mucopurulent discharge from the Right Middle Meatus (Sinus Drainage pathway) may be indicative of bacterial infection. (NS) Nasal septum, (MT)Middle Turbinate, (IT) Inferior Turbinate.

Complications

- Intracranial: meningitis, cavernous sinus thrombosis, intracranial abscess (rare)
- Associated with severe, sudden onset headache, vomiting & photophobia
- Extracranial: Osteomyelitis, orbital or pre-septal cellulitis, orbital abscess

Investigations

- Bloods: FBC for WBC, CRP,blood culture only if very ill or other comorbidity
- If complicated/not responding…
- Swab for microscopy, culture and sensitivity
- High definition CT scan with contrast, of sinuses and brain if above complications occur. Intra orbital complications are a surgical emergency as vision can be threatened in the presence of intra orbital sepsis (see Figure 45)

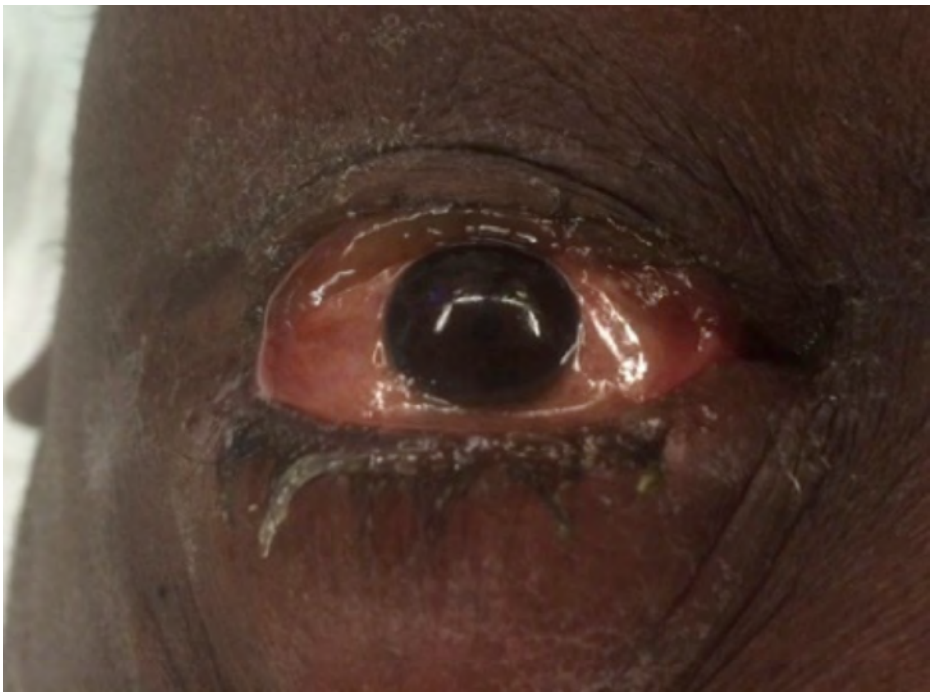

Figure 45: Evidence of a sub-periosteal abscess secondary to sinusitis causing right eye proptosis, peri-orbital swelling, chemosis and loss of the pupillary reflexes. Urgent surgery is required to reduce pressure on the optic nerve.

Treatment

If symptoms less than 5 days and mild:

- Analgesia and nasal saline irrigation
- Fluid rehydration
- Nasal decongestant e.g. xylometazoline or Pseudo-ephidrine for period of 1 week only.

If symptoms are persistent after 10 days or worsening after 5 days;

- As above
- Topical intranasal steroids

If severe (at least 3 of: discoloured discharge, severe local pain, fever, elevated ESR/CRP, double sickening)

- Broad-spectrum antibiotics for 7days. e.g. amoxicillin

- Topical intranasal steroids

If recurrent or chronic symptoms, refer to an ENT specialist for further management, and possible endoscopic sinus surgery.

Chronic Rhinosinusitis (CRS)

Description

- Inflammation of the nasal mucosa and paranasal sinuses for >12 weeks.
- Can be divided into CRS with polyps or CRS without polyps.

Epidemiology

- Common. CRS accounts for 85% of outpatient visits for rhinosinusitis in adults.
- Can follow ARS.

Causes

- Multifactorial and still not fully understood:
- Allergic: Intermittent or Persistent.
- Other non-allergic causes – Occupational, Hormonal, Granulomatous/ Inflammatory, Infective (Viral, Bacterial, Fungal), Anatomical, Iatrogenic, secondary to medication (eg. Rhinitis Medicamentosa/Cocaine abuse)

Symptoms and Signs

The European Position Paper on Rhinosinusitis and Nasal Polyps (EPOS 2012) defines the diagnostic criteria for CRS below:

- Inflammation of the nose & sinuses causing 2 or more symptoms
- Nasal blockage/obstruction/congestion

- Nasal discharge (ant/post nasal drip)

+/- facial pain/pressure
+/- reduction/loss smell

And either:

- Endoscopic signs of polyps, middle meatal oedema or mucopurulent discharge
- CT mucosal changes of the ostiomeatal complex/sinuses

Investigations

CT Sinuses. Can assess the extent of disease and provide anatomical detail for pre-operative planning (see Figure 46).

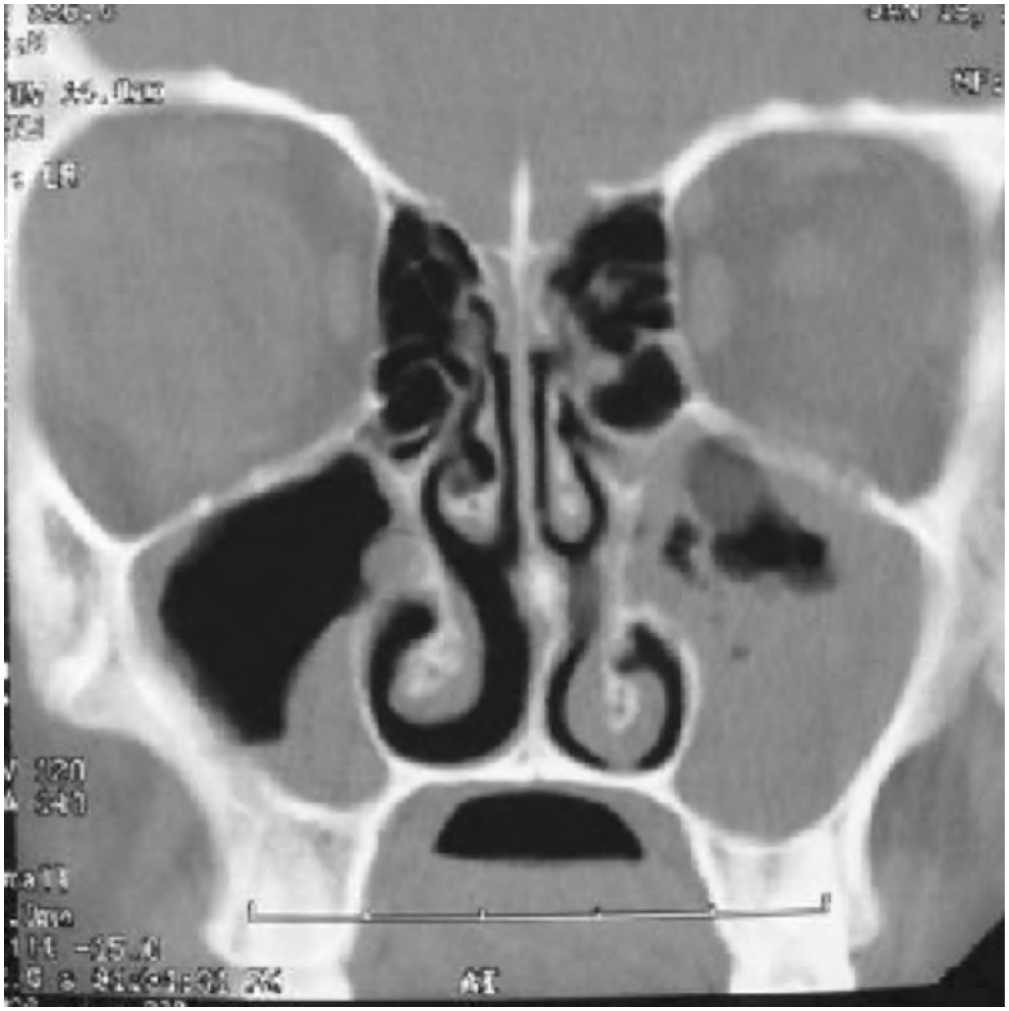

Figure 46: CT Scan in coronal section showing bilateral maxillary sinusitis. R = Right side. Disease is worse on the left hand side mostly in the maxillary sinus and there is hypertrophy of the left inferior turbinate (reproduced with permission from Otolaryngology Houston, www.ghoryeb.com)

Treatment

Non-operative. Appropriate medical management should be attempted primarily, including:

- Saline Nasal Irrigation
- Nasal decongestants (short course only)
- Anti-histamines (if there is an underlying allergic component)
- Oral steroids with CRS with polyps – (caution with side effects), followed by topical therapy

- Topical Steroids with CRS without polyps-
    - Drops include: betamethasone or fluticasone (A typical regime might be for a period of 4-6 weeks)
    - Then switch onto maintenance intra-nasal cortico-steroid spray (INCS) such as fluticasone or mometasone for 3 months until review.
- Antibiotics – Certain antibiotics may help by their anti-bacterial and anti-inflammatory mechanisms. The true benefit of antibiotics in CRS is undergoing evaluation by ongoing trials.
    - The EPOS guidance suggests macrolides are useful in non-polyp CRS when the IgE levels are not raised and the cardiac history has been taken into account due to possible effects on prolongation of the QT interval (e.g. Clarithromycin 500mg OD PO for 3 weeks).
    - In polyp patients, the antibiotic of choice is Doxycycline (50-100mg OD PO for 3 weeks) if tolerated.
    - Local formulary guidance should be sought if patients are allergic to first line options.

Operative: Functional endoscopic sinus surgery (FESS).

- The principles of FESS are to remove diseased tissue, relieve obstructions and to restore the normal function and anatomy of the paranasal sinuses. Often it can involve removing the bony septae obstructing the sinus outflows whilst preserving mucosa, to widen the sinus drainage pathways and increase access for subsequent topical medical therapies.
- Sinus surgery should be seen as an adjunct in the management of CRS where medical treatment alone has failed, rather than stand alone definitive treatment.
- Patient selection is key, and quality of life screening tools such as the SNOT-22 (Sino-Nasal Outcome Test of 22 questions) can help identify appropriately symptomatic patients who may gain benefit from surgery.

Nasal Polyps

Description

Nasal polyps are very common and are typically bilateral. They typically present with symptoms of nasal blockage with or without a change in smell perception. They are benign. They include:

- Inflammatory/Allergic Polyps – sino-nasal polyposis, often multiple grey, oedematous polyps associated with CRS (see Figure 47)
- Antro-choanal polyp – single polyp arising from maxillary sinus extending out towards nasopharynx causing unilateral nasal obstruction.

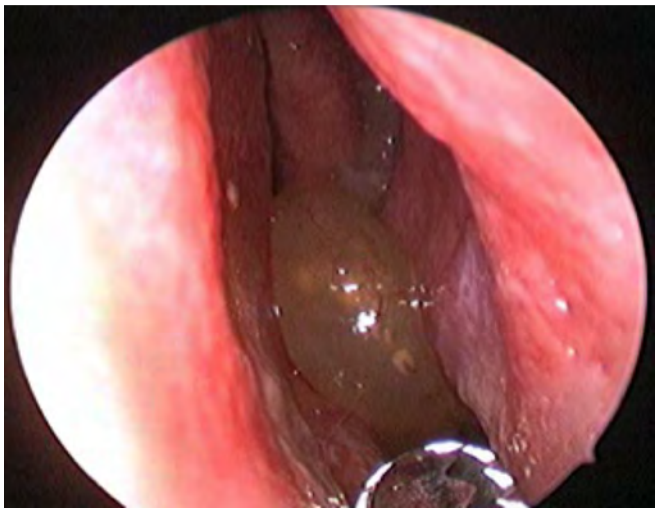

Figure 47: Clinical photograph of left nasal cavity showing inflammatory nasal polyps arising from the sinus drainage pathway (Middle Meatus) blocking nasal airway

Treatment

Medical

Medical treatment involves some form of steroid-based treatment combined with nasal saline rinses. If there are no contra-indications, a typical regime might consist of a short course of oral steroids (Prednisolone 0.5mg per kg PO for 7 days with PPI cover), followed by intra-nasal steroid drops for 4-6 weeks, and subsequently a maintenance intra-nasal corticosteroid spray (INCS) such as Mometasone until further review.

There is some evidence from the EPOS guidance that certain antibiotics are of added benefit if the levels of IgE in the serum are not raised (eg. Doxycycline).

Surgical

The principles of surgery for CRS with polyps are to establish good access for further medical treatment, when medical therapy alone has failed. Simple endoscopic nasal polypectomy or Functional Endoscopic Sinus Surgery (FESS) is usually performed under general anaesthesia and aims to remove the polyps and re-establish the natural drainage pathways of the paranasal sinuses. The extent of surgery will be governed by disease factors, patient factors and the experience of the operating surgeon.

When consenting patients for surgery, certain risks should be included such as pain, infection, bleeding, the need to pack the nose, intra-cranial complications (CSF leak and meningitis), intra-orbital complications (double vision/blindness) and possible recurrence of polyps/sinus disease.

Other Sinonasal Lesions

Benign lesions

- Papilloma/Wart – verrucous lesion, commonly in nasal vestibule, often multiple and painless, presents with bleeding. Local excision is mainstay of treatment
- Pyogenic Granuloma – friable lesion that bleeds, usually from trauma often arising on septum. Commoner in pregnancy
- Other rarer pathology can have the appearance of a “unilateral nasal polyp” during nasal examination. Examples include
- Inverted Papilloma – benign but locally aggressive polyp that has a predisposition for recurrence if not completely cleared including its site of origin. Can rarely transform into malignancy over time (see Figure 48)
- Juvenile Nasopharyngeal Angiofibroma (JNA) – vascular benign tumour exclusively present in adolescent males (may have hormonal element). These are extremely rare but classically present with nose-bleeds and nasal obstruction in teenage boys. Embolisation and subsequent surgical removal (endoscopic or open depending on extent) is the mainstay of treatment.
- Meningoencephalocoele/glioma – herniation of intracranial contents through a weakness in skull-base containing meninges, brain (rarely functioning) or support tissue. Imaging with MRI needed before biopsy.

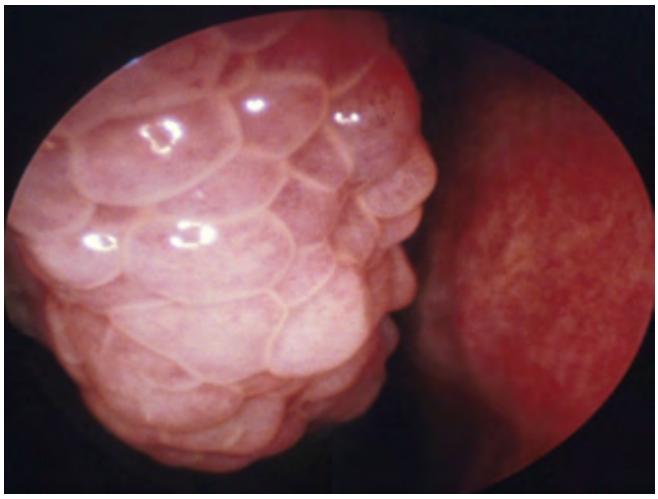

Figure 48: Inverted papilloma right nasal cavity arising from lateral wall of nose

Malignant

Sino-nasal malignancies are rare but present late, resulting in a poor prognosis. Presenting symptoms include unilateral nasal obstruction, unilateral glue ear, bleeding, pain, neck lumps, unexplained weight loss, eye symptoms, headaches or cranial nerve deficits. The commonest are squamous cell carcinomas (see Figure 49 and Figure 50), adenocarcinomas (associated with wood-working) and nasopharyngeal carcinomas (arising from the nasopharynx)

Imaging and histology are essential and the results are discussed in a multi-disciplinary team meeting, where recommendations for which treatment is best suited for the patient can be made.

Treatment can be curative or palliative and may include surgery, chemotherapy (drugs), radiotherapy (ionizing radiation) or a combination of these. Nasopharyngeal cancers are commonly treated with radiotherapy or chemoradiotherapy.

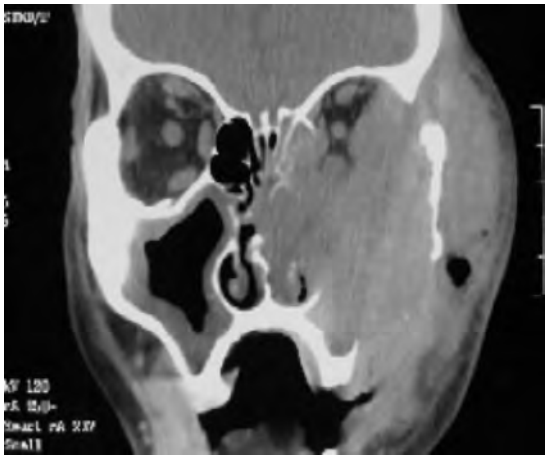

Figure 49: SCC of the Left Maxillary Sinus

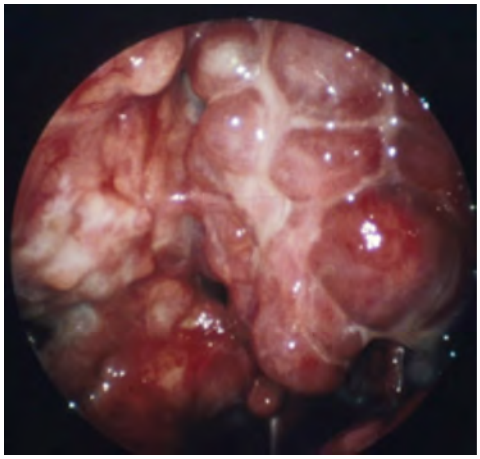

Figure 50: Endoscopic image of SCC Left Maxillary Sinus

Cleft Lip and Palate

Description

A cleft is a gap or split in the upper lip and/or roof of the mouth (palate) resulting in an abnormal connection between the oral and nasal cavity. A cleft lip and palate is the most common facial birth defect in the UK, affecting around one in every 700 babies.

It can range in severity from a submucous cleft (the muscles of the soft palate not fully joining) which may be relatively asymptomatic to a bilateral cleft lip and palate with immediate concerns at birth regarding airway and feeding.

It can be an isolated occurrence or associated with other genetic and developmental abnormalities.

Important Considerations

- Feeding – poor suction, lengthy feeds, nasal regurgitation, excessive air intake, poor airway protection and expending too much energy can cause failure to thrive. Bottles with special nipples can help improve this.
- Otologic – high incidence of glue ear and delayed resolution (poor Eustachian tube function secondary to abnormally developed palate muscles predisposes to middle ear effusions) means hearing assessment early on is a priority with onward referral for grommets or hearing aids.
- Speech/Swallow – speech and language therapy to combat problems with palate dysfunction and reflux
- Cosmetic – lip and nasal deformities will require addressing within 1st year of life and revision often required later as patient grows
- Dental – restoration / prosthesis may be required for normal function and cosmesis

- Psychology, Social Work & MDT – parental counselling and assistance is important starting pre-natally and ongoing throughout childhood. Paediatricians are often well placed to help co-ordinate global development.

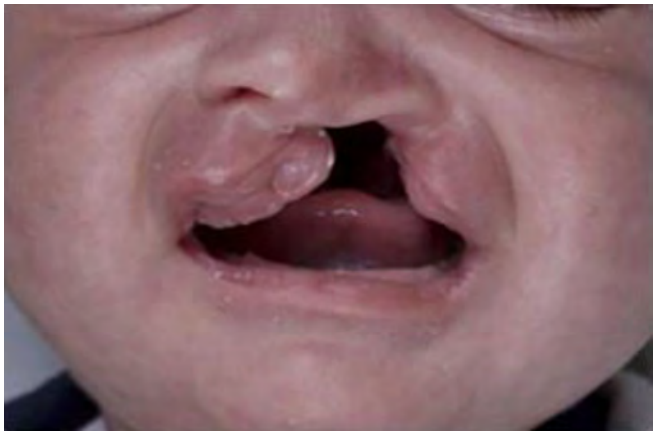

Figure 51: Unilateral left cleft lip and palate (Courtesy of Elf-ENT)