51 T_h e ear, nose and sinuses

- Acute otitis media
- Acute rhinosinusitis
- Balance disorders
- Benign tumours
- CONDITIONS OF THE EXTERNAL EAR Congenital anomalie
- CONDITIONS OF THE EXTERNAL EAR Congenital anomalies
- CONDITIONS OF THE INNER EAR Congenital sensorineur
- CONDITIONS OF THE INNER EAR Congenital sensorineural hearing loss
- CONDITIONS OF THE MIDDLE EAR Congenital anomalies
- Chronic otitis media
- Chronic rhinosinusitis
- EPISTAXIS
- EXAMINATION OF THE EAR
- EXAMINATION OF THE NOSE AND
- IMAGING OF PARANASAL SINUSES
- Introduction
- Inflammation and infection
- Learning objectives
- Malignant tumours
- Management of epistaxis
- Neoplasms
- Otitis media with effusion (glue ear)
- Otosclerosis

- PARANASAL SINUSES
- Presbycusis
- RHINOSINUSITIS
- Radiological investigation
- Septal deformity
- Septal injury
- Septal perforation
- Sudden sensorineural hearing loss
- THE EAR
- THE NOSE AND SINUSES BASIC ANATOMY OF THE NOSE AND PARANASAL SINUSES
- THE NOSE AND SINUSES BASIC ANATOMY OF THE NOSE AND
- TRAUMA TO THE NOSE AND PARANASAL SINUSES Fracture of the nasal bones
- TRAUMA TO THE NOSE AND PARANASAL SINUSES Fracture
- TUMOURS OF THE NOSE AND SINUSES
- The external ear
- The inner ear
- The sensory nerve supply
- The tympanic membrane and middle ear
- Tinnitus
- Trauma

Acute otitis media

Acute otitis media

Acute otitis media (AOM) is one of the most common child - hood illnesses with a peak incidence between 6 and 18 months of age. It has occurred in 70% of children by the age of 2 and in 90% by the age of 6. It is characterised by purulent fluid in the middle ear. The tympanic membrane bulges because of pressure from the pus in the middle ear (Figure 51.17). The child su ff ers pain, fever and lethargy . The most common infecting organisms are Streptococcus pneumoniae and Haemoph - ilus influenzae . Treatment is with analgesics and antipyretics. Systemic antibiotics should be reserved for children under 2 /uni00A0 years with bilateral disease or those with other risk factors for complications.The most common complication is mastoiditis because the mastoid air cells connect freely with the middle ear space. Mastoiditis (Figure 51.18) requires hospital admission for intravenous antibiotics, for consideration of CT scanning - and to monitor for complications such as facial nerve palsy , lateral sinus thrombosis and meningitis. If infection does not resolve quickly abscess aspiration and myringotomy (with/ without grommet insertion) is performed. A cortical mastoid - ectomy is carried out if complications arise. Acute otitis media

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Acute rhinosinusitis

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ARS is thought to result from bacterial superinfection of virally damaged mucosa. The commonest bacteria involved are S. /uni00A0 pneumoniae , H. influenzae and Moraxella catarrhalis . Upper dental sepsis may also predispose to acute maxillary sinusitis. Patients with maxillary sinusitis have a mucopurulent discharge, facial pain and nasal obstruction. Irritation of the superior alveolar nerve may give rise to referred upper toothache. In ARS nasendoscopy reveals inflamed and swollen nasal mucosa with mucopurulent secretions in the middle meatus. Dental sepsis from anaerobic organisms causes around 10% of cases of maxillary sinusitis. The resultant mucopurulent nasal secretion has a foul taste and smell. Plain sinus radiographs may show a fluid level in the antrum or complete opacity (Figure 51.47). However, plain radiographs are now seldom used and have been superseded by CT scans to investigate ARS. CT scans confirm opacification and mucosal thickening of the maxillary sinus as well as providing anatomical detail prior to endoscopic surgical intervention (Figure 51.48). Acute frontoethmoidal sinusitis can also occur and presents with mucopurulent discharge, facial pain (including frontal headache), nasal congestion and hyposmia. Again, mucopus is seen on endoscopy in the middle meatus and is investigated with CT . - Treatment Penetration of antibiotics into chronically inflamed sinus mucosa is reduced and, therefore, treatment may need to be prolonged. Topical nasal decongestants, such as ephedrine nasal drops, will often encourage the sinus to drain and topi - cal corticosteroids are used to reduce inflammation. Saline douches can also be beneficial. Antral lavage under local or general anaesthesia was previ - ously used to confirm the diagnosis and provided the opportu - nity to obtain samples for bacteriology . Nowadays, pus in the middle meatus can simply be sampled endoscopically in clinic and antral lavage is rarely performed. Endoscopic sinus surgery allo ws a more functional approach to diseases of the paranasal sinuses and enables the drainage pathways of the paranasal

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Frontal sinus sinuses to be opened. Most cases of ARS can be treated con servatively with antibiotics and topical treatment. Surgery is used for those patients unresponsive to medical management or with complications. The majority of patients with ARS who require surger y are treated endoscopically . However, in some cases an open surgical approach may be necessary . Percivall Pott , 1714–1788, surgeon, London, UK. Acute rhinosinusitis /uni25CF /uni25CF /uni25CF /uni25CF Complications Complications of ARS include orbital and intracranial prob - lems. The spread of infection from the sinuses occurs either through diploic veins or directly through bone erosion. This can result in epidural, subdural or cerebral abscesses or in meningitis/encephalitis. Cavernous sinus thrombosis may also r esult and can present with bilateral ptosis, proptosis, retro-ocular pain, ophthalmoplegia, papilloedema and spiking fevers. Orbital complications of ARS are more common. Most often this is related to ethmoid sinus infection (Figure 51.49). An ophthalmology re view is essential because of the threat to vision and intravenous antibiotics covering aerobic and anaer - obic organisms are used. If there are any concerns regard - ing the

eye, including proptosis, chemosis, ophthalmoplegia or reduced visual acuity , then CT with contrast is required (Figure 51.50). If an abscess is identified, this should be drained (endoscopically or open). Summary box 51.13 Complications of acute rhinosinusitis /uni25CF /uni25CF /uni25CF /uni25CF Summary box 51.14 Chandler classification of orbital complications of sinusitis /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF - Osteomyelitis of the frontal bones can also occur as a com - plication of ARS. If the anterior table of the frontal sinus is involved and becomes dehiscent, it can present with significant swelling of the skin of the forehead and a mass – Pott’s pu ff y tumour (Figure 51.51).

Infraorbital rim Infraorbital foramen Air/ /f_l uid level Figure 51.47 Plain radiograph showing the /f_l uid level in the left maxil lary antrum and total opacity of the right antrum. Figure 51.48 Coronal computed tomography scan showing left-sided maxillary sinus opaci /f_i cation due to maxillary sinusitis. The most common causative organisms are S. pneumoniae, H. /uni00A0 in /f_l

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Orbital – cellulitis, abscess Orbital infections may threaten sight Intracranial spread may cause meningitis, cerebral abscess or cavernous sinus thrombosis Osteomyelitis of the bones, particularly frontal, may occur I – preseptal cellulitis II – orbital cellulitis III – subperiosteal abscess IV – orbital abscess V – cavernous sinus thrombosis

Figure 51.49 Left periorbital cellulitis complicating acute left ethmoiditis. Figure 51.50 Axial computed tomography scan showing a subperi osteal abscess in the left orbit.

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Balance disorders

Balance disorders

V ertigo is the hallucination of movement. Benign paroxysmal positional vertigo Benign paroxysmal positional vertigo (BPPV) is the most common form of vertigo. It is caused by otoliths (calcium carbonate crystals) most commonly within the posterior semi - circular canal abnormally triggering the ampullary hair cells. Typically , the vertigo is triggered by turning, only lasts for a few seconds and is not associated with other otological symptoms. A positive Hallpike test confirms the diagnosis. The condition is usually self-limiting but reco very may be expediated by an Epley manoeuvre. Vestibular neuronitis Infection or inflammation of the superior vestibular nerve results in persistent vertigo lasting a few days. If the hearing is also a ff ected, this is known as labyrinthitis. Treatment is supportive with vestibular sedatives, such as prochlorperazine, in the first few days, early mobilisation and consideration of systemic steroids. Menière's disease It has been said that clinicians not only disagree on the cause of Menière's disease, but they also disagree on the spelling. There is certainly evidence of endolymphatic hydrops (longstanding high-pressure changes within the inner ear) in pathological specimens of patients who have had the condition. The condition is characterised by a triad of symptoms: intermittent attacks of vertigo, a unilateral fluctuating sensorineural hearing loss and tinnitus. The patient often has a sensation of pressure in the a ff ected ear before an attack. The hearing loss typically a ff ects the lower frequencies. The vertigo characteristically lasts between 30 minutes and 6 hours and is often accompa nied by nausea and vomiting. The investigations include pure tone audiometry and an MRI scan (to exclude an acoustic neuroma). The only evidence-based medical treatment is intratympanic injections of dexamethasone or gentamicin into the middle ear. Vestibular migraine Often confused with Menière's disease, this condition is five times more prevalent, presenting with similar symptoms but without the hearing loss or tinnitus. The migrainous process Prosper Menière , 1799–1862, physician, The Institute of Deaf Mutes, Paris, France, described this condition in 1861. Sir Charles Bell , 1774–1842, surgeon, The Middlesex Hospital, London UK, and from 1835 until his death, Professor of Surgery , The University of Edinburgh, Edinburgh, UK. a ff ects the labyrinth in up to 40% of migraineurs. Treatment includes addressing the risk factors, such as lifestyle and dietary triggers, with prophylactic medication such as propranolol, tricyclic antidepressants and antiepile ptic medication for those with ongoing symptoms. Facial paralysis Seventy-five per cent of all facial palsies are due to Bell's palsy . This probably results from a herpes simplex viral infection of the facial nerve. The nerve swells and is compressed within the temporal bone. Early treatment with high-dose steroids and eye protection is mandatory . Not all facial nerve palsies are due to viral infection and a thorough otoneurological examination is required. The facial nerve can be damaged at the cerebel - lopontine angle, within the internal auditory meatus, within the middle ear, at the skull base and within the parotid gland. It is essential to consider these potential sites of facial nerve damage in any pa tient with CN VII paralysis and perform an MRI scan if appropriate. - Summary box 51.7 Facial paralysis /uni25CF /uni25CF /uni25CF /uni25CF

–20 –10 0 10 20 30 40 50 60 70 Hearing level (dB ISO) 80 90 100 110 120 125 250 500 1000 2000 4000 6000 Frequency (Hz) Figure 51.30 A typical audiogram of noise damage: (a) right ear; –20 –10 0 X 10 X X 20 30 X 40 X 50 60 70 Hearing level (dB ISO) 80 X 90 100 110 120 125 250 500 1000 2000 4000 6000 Frequency (Hz) (b) left ear. The facial nerve passes through the middle ear and mastoid When considering a paralysis, think ‘complete’ or ‘partial’ Protect the eye: carry out a full otoneurological examination to /f_i nd the cause If acute, consider steroids

Ramsay Hunt syndrome This is caused by herpes zoster virus and is characterised by facial paralysis, pain and the appearance of vesicles on the tympanic membrane, ear canal, pinna or inside of the cheek (Figure 51.31). It may be accompanied by vertigo and senso rineural hearing loss (CN VIII). Treatment with aciclovir is e ff ective if given early . Neoplasms These are uncommon but can present with sensorineural hearing loss, tinnitus and vertigo. Acoustic neuromas, which are actually schwannomas of the vestibular division of CN VIII, are the most common, followed by meningiomas. Acous tic neuromas grow slowly and somewhat unpredictably and as they expand can cause CN palsies, brainstem compression Summary box 51.8 Conditions of the inner ear /uni25CF /uni25CF /uni25CF /uni25CF James Ramsay Hunt , 1874–1937, Professor of Neurology , Columbia College of Physicians and Surgeons, New Y ork, USA. and raised intracranial pressure. The early symptoms are a unilateral sensorineural hearing loss or unilateral tinnitus, or both. Therefore, it is essential to perform MRI on all patients with persistent unilateral sensorineural hearing loss or tinnitus. Relatively asymptomatic acoustic neuromas that are less than - 2 /uni00A0 cm in diameter and growing less than 2 /uni00A0 mm/year (70%) are generally treated with a ‘watch, wait and rescan’ policy or occasionally stereotactic radiotherapy . Tumour volumes greater than 2 /uni00A0 cm in diameter are often best treated by skull base surgery in the form of a translabyrinthine, retrolabyrin - thine or middle fossa approach. -

Figure 51.31 Herpes zoster infection of right cranial nerve (CN) VII Presbycusis is the bilateral high-frequency loss associated with ageing Unilateral tinnitus or sensorineural hearing loss needs to be investigated to exclude acoustic neuroma Sudden sensorineural hearing loss needs immediate treatment with steroids and routine MRI to exclude acoustic neuroma Menière’s disease presents with the triad of sensorineural hearing loss, tinnitus and vertigo (a) and right CN VIII (b) with vesicles on the pinna.

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Benign tumours

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- Simple papillomas or viral warts can grow inside the nasal vestibule. They can be confused with carcinomas and are best excised for histological diagnosis. Osteomas of the nasal skeleton are not uncommon and are often detected on radiology as an incidental finding (Figure 51.55). In symptomatic individuals the osteoma can be removed endoscopically or via an open procedure. Inverted (transitional cell) papillomas can occur in both the nasal cavity and the nasal sinuses. T hey are inverted papillomas because histologically the hyperplastic epithelium inverts into the under lying stroma. The papillomas are covered with transitional epithelium. Calcification within the tumour may be seen on CT along with sclerosis of the bone at the margins of the growth (Figure 51.56). Inverted papillomas can undergo malignant change. Full surgical resection is required, and this can usually be performed endoscopically .

Figure 51.55 Coronal computed tomography (CT) scan showing an osteoma in the left anterior ethmoid sinus adjacent to the orbit The second coronal CT scan is following endoscopic excision of the osteoma (b) . Figure 51.56 Coronal computed tomography scan showing an extensive inverted papilloma involving the left maxillary sinus; arrow indicates calci /f_i cation within the tumour. Figure 51.57 Squamous cell carcinoma of the nasal septum.

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CONDITIONS OF THE EXTERNAL EAR Congenital anomalie

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CONDITIONS OF THE INNER EAR Congenital sensorineur

CONDITIONS OF THE INNER EAR Congenital sensorineural hearing loss

Half of congenital sensorineural hearing loss is genetic and half is acquired. Of the genetic hearing loss 75% is non-syndromic, of which the most common is a connexin 26 gene mutation. Syndromic causes include Usher, Pendred, Jervell and Lange-Nielsen, Waardenburg, Treacher Collins, Alport, Stickler, neurofibromatosis type 2 and branchio-oto renal syndromes. Acquired causes are intrauterine infections, including rubella, toxoplasmosis and cytomegalovirus infection; perinatal hypoxia, jaundice and prematurity; and postnatal meningitis. All newborn babies in the UK are now screened at birth for deafness b y measuring otoacoustic emissions in response to ‘clicks’ in the ear. Children failing this are referred for auditory brainstem response to establish hearing thresholds (Figure 51.26). If some hearing is present, the early fitment of hearing aids can maximise the neural plasticity that is pres ent in the developing brain. If a child has a profound hearing loss, early intervention with a cochlear implant is essential for the development of the auditory cortex (Figure 51.27). Most cases of prof ound sensorineural hearing loss are due to loss of cochlear hair cells, so an implant inserted through the round window can selectively stimulate the cochlear neurones, which usually remain intact.

Figure 51.26 Evoked-response audiometry. A simple non-invasive objective test of hearing thresholds. (Reproduced with permission from O’Donoghue GM, Bates GJ, Narula A. Clinical ENT: an illustrated textbook . Oxford: Oxford University Press, 1991.)

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Chronic otitis media

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Chronic otitis media (COM) is a persisting (at least 2 weeks to 3 months) abnormality of the tympanic membrane from previ ous recurrent AOM and/or OME. It is classified as active (i.e. inflammation and pus present), inactive (potential to become active) or healed (no potential to become active). Active and passive are then further subclassified as mucosal or squamous (Figure 51.22). Active mucosal COM implies a perforation with otor rhoea (ear dischar ge) due to inflamed middle ear mucosa with or without granulation tissue. Inactive mucosal COM implies a dry perforation without inflammation. Surgery in the form of tympanoplasty (repair of the perforation) is indicated in patients with recurrent infection (to reduce symptoms of otor rhoea and prevent further deterioration of the hearing due to the ototoxic e ff ects of infection) and where there is a likelihood that it will restore hearing in the operated ear to 30 /uni00A0 dB or better or to within 15 /uni00A0 dB of the contralateral ear (this is known as the Belfast rule of thumb). Active squamous COM is otherwise known as acquired cholesteatoma. This represents a quarter of all active COM with an incidence of 1/10 /uni00A0 000. It usually presents with per - sistent otorrhoea and hearing loss as a result of keratinising - squamous epithelium within the middle ear. The cholesteatoma - matrix destroys the structures in its path through the release of lytic enzymes , inflammatory mediators and pressure necrosis. If left, there is a risk of all the complications attributable to AOM. The lifetime risk of intracerebral abscess is 1/200 in a 30-year-old patient. The recommended treatment is mastoid surgery using a drill under microscopic or endoscopic guidance to access and remove the cholesteatoma. Often the ossicles are involved or eroded so an ossiculoplasty (to restore hearing by reconstructing the ossicular chain) may be performed at the same time.

media Active Inactive Mucosal: Squamous: Mucosal: Squamous: dry retraction/ discharging cholesteatoma perforation atelectasis perforation Figure 51.22 Classi /f_i cation of chronic otitis media.

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Chronic rhinosinusitis

Chronic rhinosinusitis

CRS is common, a ff ecting around 11% of the population. The aetiology is multifactorial and a number of factors have been linked to CRS, including ciliary dyskinesia, allergy , asthma, bacteria (Staphylococcus aureus), fungi and a number of host factors, including anatomical variations (deviated septum, concha bullosa of the middle turbinates). CRS has tradition ally been divided into CRS with nasal polyps (CRSwNPs) and without (CRSsNPs). More recently CRS has been classified into primary or secondary , and local or di ff use disease. Pathology Nasal polyps are benign swellings of the sinus mucosa of unknown origin. Histologically , the polyps contain an oedema - tous stroma infiltrated with inflammatory cells and eosinophils. Inflammatory polyps tend to be bilateral and extend into the middle meatus. A single large polyp arising from the maxillary). antrum is referred to as an antrochoanal polyp (Figure 51.52 This usually fills the nose and eventually prolapses posteriorly down into the nasopharynx. Clinical features Patients with CRSwNPs present with nasal obstruction, watery rhinorrhoea, postnasal drip and often hyposmia/anosmia. Pain does not tend to be a significant feature. Polyps are easily identifiable within the nose as pale semitransparent grey masses, which are mobile and insensitive when palpated with a fine probe (Figure 51.53). This allows them to be distinguished from hypertrophied turbinates. In CRSsNPs the middle meatus is often congested, with mucopus present. Malignancy should be considered in adults with unilateral nasal polyps whereas in children such polyps must be - distinguished from a meningocele or encephalocele by high- the anterior cranial fossa. Nasal resolution CT scanning of polyps are unusual in children; however, they do occur in conjunction with cystic fibrosis in 10% of cases.

(b) Figure 51.51 Sagittal (a) and axial (b) computed tomography scans showing complete opaci /f_i cation of the frontal sinus (marked with an asterisk) due to frontal sinusitis. The anterior wall of the

frontal sinus is absent owing to infection.

Management Medical treatment of CRSwNPs with systemic steroids will often reduce the size of the nasal polyps and give short-term relief of nasal blockage. Unfortunately , the polyps tend to recur when the treatment stops. Topical corticosteroid drops and sprays are also used along with saline douching. Biological treatments using monoclonal antibodies are a potential new therapy for CRSwNPs. In CRSsNPs, in addition to topical treatments, a long course of low-dose antibiotics (macrolides) can be used in those patients with a normal level of immu noglobulin E. Surgical treatment is indicated in patients who do not respond to medical treatment. Endoscopic nasal polypectomy and functional endoscopic sinus surgery (FESS) is performed follo wing a CT scan that confirms the extent of disease and shows the important bony anatomy preoperatively . Serious complications following FESS include CSF leak and orbital problems, including orbital haematoma, and so it is important to review the level and symmetry of the anterior skull base and the integrity of the lamina papyracea on the CT scan prior to surgery . Endoscopic polypectomy is performed using a powered nasal microdebrider (Figure 51.54). Image guidance can be used in endoscopic sinus surgery and extended endoscopic procedures such as pituitary and anterior skull base surgery to provide real-time feedback of instrument position in the nose based on preoperative CT or MRI scans. Summary box 51.15 Nasal polyps /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF

Figure 51.52 Antrochoanal polyp. Figure 51.53 Nasal polyp in the right nasal vestibule. Figure 51.54 Powered nasal microdebrider. Polyps are insensitive to touch and are mobile In /f_l ammatory polyps are usually bilateral Unilateral nasal polyps should be removed for histology Bleeding polyps may indicate malignancy Meningocele and encephalocele must be excluded in children with polyps Polyps are removed using a powered microdebrider

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EPISTAXIS

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The causes of epistaxis are listed in Table 51.2 . The most common site of bleeding is from Kiesselbach’s plexus in Little’s area of the anterior portion of the septum (Figure 51.37). Anterior bleeding is common in children and young adults owing to nose blowing or picking. In the elderly , anticoagulants and hypertension are the underlying causes of arterial bleeding from the posterior part of the nose. Hereditary haemorrhagic telangiectasia (HHT) (Osler’s dis ease) gives rise to recurrent multifocal bleeding from thin-walled vessels deficient in muscle and elastic tissue (Figure 51.44 Juvenile angiofibroma is an uncommon condition that a ff ects adolescent boys and may lead to massive life-threatening episodes of b leeding. Diagnosis is made with contrast CT or MRI. Anterior bowing or indentation of the posterior antral wall (Holman–Miller or antral sign) is the classical finding but may be seen in other expansive lesions in this ar ea. It is a very vascular tumour, which should not be biopsied because of the risk of uncontrollable haemorrhage. Excision is best carried out by an experienced surgeon and is usually performed endoscopically , often using image guidance (Figure 51.45 Preoperative embolisation of the feeding blood vessels may help to reduce blood loss during surgery . /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF

Figure 51.43 Silastic prosthesis for septal perforation. TABLE 51.2 Causes of epistaxis. Local Nose picking Nasal trauma Nasal foreign bodies Tumours Infection Granulomatous disorders Juvenile angio /f_i broma Systemic Hypertension Warfarin therapy New anticoagulants (rivaroxaban) Aspirin, clopidogrel therapy Haemophilia von Willebrand’s disease Leukaemia Hereditary haemorrhagic telangiectasia (Osler’s disease)

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EXAMINATION OF THE EAR

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The instruments required for examination are shown in Figure 51.5 . Examination of the ear is part of the general ear, nose and throat (ENT) examination. Rinne and Weber tuning fork tests are used to distinguish between a conductive and a sensorineural hearing loss. The correct way to hold an otoscope is shown in Figure 51.6 . The CNs and especially the function of the facial nerve should be examined. Although conversational testing can give a useful guide to the level of hearing, pure tone audiometry the best way of establishing the in a soundproof booth is air and bone hearing levels (Figure 51.7). Other common audiological tests include speech audiometry , tympanometry , stapedial reflexes, electric response audiometry , otoacoustic emissions, caloric testing and electronystagmography (see Further reading).

Figure 51.5 Tools of the trade: a /f_i breoptic otoscope, with pneumatic attachment and a selection of specula. Also a 512-Hz tuning fork. Figure 51.6 The correct method of holding the otoscope. Note the pinna is retracted to straighten the ear canal. Hold the barrel of the otoscope so that the examiner’s little /f_i nger is balanced on the patient’s cheek; this prevents the speculum impinging on the tym panic membrane in case of sudden movement.

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IMAGING OF PARANASAL SINUSES

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Plain radiographs are of limited value in the assessment of sinus disease. CT is far superior in demonstrating sinus pathol ogy and for assessing bony anatomy to plan any surgical inter vention. CT scans are acquired and reconstructed to produce images in axial, coronal and sagittal planes. The three planes allow the drainage of the frontal sinus to be identified and important surgical landmarks can be reviewed preoperatively , including the cribrif orm plate, anterior skull base, lamina papyracea and location of the anterior ethmoid artery . MRI is useful in sinus pathology to assess any intracranial or orbital extension of disease. IMAGING OF PARANASAL SINUSES

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Introduction

INTRODUCTION

Disorders a ff ecting the ear, nose and sinus are common reasons for primary care attendance; however, few surgeons will encounter such diseases in day-to-day practice. Nonethe less, traumatic, infective and neoplastic processes can impact on these organs and their anatomical proximity to critical anatomical structures demands a basic understanding in order to e ffi ciently diagnose, refer and treat conditions. A full and detailed review of the management of ear and nose conditions is beyond the scope of this text. Instead, the aim of this chapter is to familiarise the reader with the basic anatomy and pathol ogy relevant to patients who present with conditions a ff ecting the ear, nose and sinuses.

Inflammation and infection

Inflammation and infection

Otitis externa is very common and consists of generalised inflammation of the skin of the external auditory meatus. The cause is often cotton bud use, a moist environment, immunocompromise, allergies or skin disorders, such as psori asis and eczema. Common pathogens are Pseudomonas Staphylococcus bacteria, Candida and Aspergillus . Once the skin of the ear canal becomes oedematous, skin migration stops and debris collects in the ear canal. This acts as a substrate for the pathogens. Movement of the pinna elicits pain, which distinguishes it from otitis media. The initial treatment is with a topical antibiotic and ste roid ear drops together with analgesia. If this fails, meticulous removal of the debris with the aid of an operating microscope is required. Fungal infection can be recognised by the presence Friedrich Theodor Schwann , 1810–1882, Professor of Anatomy and Physiology , successively at Louvain (1839–1848) and Liège (1848–1880), Belgium, described the neurilemma in 1839. of hyphae within the canal (Figure 51.12). Fungal infection causes irritation and itch. The treatment is meticulous removal - of the fungus and any debris, as well as stopping any con - and current antibiotics. Systemic antibiotics are rarely required for otitis externa but should be used if cellulitis of the pinna occurs (Figure 51.13). Necrotising otitis externa is a rare but important condition because, if left untrea ted, it has a high mortality . It presents as a severe, persistent, unilateral otitis externa possibly with facial - weakness in an immunocompr omised individual (e.g. elderly Pseu - patient with diabetes). Usually the infecting organism is domonas aeruginosa . Osteomyelitis of the skull base may result in lower CN palsy (VII–XII). A multidisciplinary approach

Figure 51.10 Haematoma of the pinna. Figure 51.11 Removal of a foreign body from the ear canal can be a challenge (courtesy of Dr Christian Deguine).

involving microbiology and radiology is required with long term systemic antibiotic treatment.

Figure 51.12 Fungal otitis externa. Note the spores. Figure 51.13 Cellulitis of the pinna.

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Learning objectives

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To be familiar with: The anatomy of the ear • The conditions of the outer, middle and inner ear • The examination of the ear, including hearing tests • The basic anatomy of the nose and paranasal sinuses • The principles of managing post-traumatic nasal and • septal deformity The causes and management of epistaxis • To understand: The outer layer of the tympanic membrane migrates • outwards Learning objectives

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Malignant tumours

Malignant tumours

The most common malignant tumours to occur within the nasal cavity and paranasal sinuses are squamous cell carcinomas (Figure 51.57), adenoid cystic carcinomas and adenocarcinomas. Adenocarcinoma has been linked to exposure to hard wood dust in the furniture industry . Adenoid cystic carcinomas arise from minor salivary glands, which can be found in the nose. Suspicious signs of invasion of neighbouring tissues include diplopia, proptosis, loosening of the teeth (Figure 51.58), trismus, CN palsies and regional lymphadenopathy . Figure 51.59 shows invasion of a left maxillar y antral carcinoma into adjacent structures, including the orbit, on an MRI scan. Patients with sinus or intranasal malignancy are best managed in a combined clinic where the expertise of ENT surgeons, maxillofacial surgeons and oncologists can be employed.

(a) . Figure 51.58 Maxillary antral carcinoma presenting through an oro

antral /f_i stula. Figure 51.59 Coronal magnetic resonance imaging scan of the paranasal sinuses showing extensive left maxillary antral carcinoma invading adjacent structures.

Tumours of the nose and sinuses /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF Fokkens WJ, Lund VJ, Hopkins C et al . European position paper on rhinosinusitis and nasal polyps. Rhinology 2020; 58 (Suppl S29): 1–464. Watkinson JC, Clarke RW (eds). Scott-Brown’s otorhinolaryngology and head and neck surgery , 8th edn. Boca Raton, FL: CRC Press, 2018.

Unilateral nasal blockage, discharge and bleeding are often presenting symptoms in nasal or sinus tumours Osteomas are often asymptomatic Inverted papilloma is a benign tumour, which presents as a unilateral polyp that can undergo malignant change Squamous cell carcinoma is the most common malignant tumour Almost 50% of sinonasal cancers arise on the lateral nasal wall and 33% in the maxillary antrum Multidisciplinary management of malignant sinonasal tumours requires input from ENT surgeons, maxillofacial surgeons and oncologists

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Management of epistaxis

Management of epistaxis

Anterior bleeding from Kiesselbach’s plexus may be controlled by silver nitrate cautery under local anaesthesia. Even in more posterior epistaxis, the bleeding point can often be identified using rigid nasendoscopy and controlled with the use of a topical vasoconstrictor, and then dealt with directly using electrocautery . However, posterior bleeding, as seen in the Sir William Osler , 1849–1919, Professor of Medicine, successively at McGill University , Montreal, Canada, University of Pennsylvania, Philadelphia, PA, and Johns Hopkins University , Baltimore, MD, USA, finally becoming Regius Professor of Medicine at Oxford University , Oxfor Colin B Holman , 1917–2008, American radiologist, Mayo Clinic. W Eugene Miller , American radiologist, Mayo Clinic, with Colin Holman described the eponymous sign on plain radiographs in 1965. Erik Adolf von Willebrand , 1870–1949, physician, Diakonissanstaltens Hospital, Helsinki, (Helsingfors), Finland, described hereditary pseudohaemophilia in 1926. impregnated ribbon gauze or a non-absorbable sponge. There are also many haemostatic, absorbable materials that can be used to pack the nose to help control bleeding . An alternative to anterior packing is the use of an inflatable epistaxis balloon catheter (Figure 51.46). The catheter is passed into the nose and the distal balloon is inflated in the nasopharynx to secure it. The proximal balloon, which is sausage shaped, is then inflated within the nasal fossa to compress the bleeding point. Although usually e ff ective, they can be uncomfortable. Postnasal packing may be required in refractory cases whereby a gauze pack is positioned in the nasopharynx under general anaesthesia. Endoscopic sphenopalatine artery clip - - ping is an e ff ective treatment for significant epistaxis not responding to direct cautery or nasal packing .). For uncontrolled life-threatening epistaxis in which the above methods have prov ed ine ff ective, haemostasis is secured by vascular ligation. Depending on the origin of bleeding it ma y be necessary to ligate the internal maxillary artery in the pterygopalatine fossa (which can be accessed endoscopically) and the anterior and posterior ethmoidal arteries. An alter - native measure is external carotid artery ligation above the origin of the lingual artery . Another option is to involve the interventional radiologist for possible embolisation. It is also important to recognise, and treat, any factors contributing to). the e pistaxis, such as clotting or platelet abnormalities. d, UK, in 1904.

Figure 51.44 Hereditary haemorrhagic telangiectasia (Osler’s disease) showing multiple telangiectasia.

In HHT , anterior nasal packing is best avoided if at all possible because it is most likely to lead to further mucosal trauma and bleeding. High-dose oestrogen induces squamous metaplasia of the nasal mucosa and has been used e ff ectively in treating this condition. Medications that block vessel growth, such as bevacizumab, and those that slow the disintegration of clots, such as tranexamic acid, help reduce the bleeding asso ciated with HHT . There are also surgical options, including cautery/ablation of the telangiectasia, septodermoplasty or surgical closure of the nostril (Y oung’s procedure).

Figure 51.45 Endoscopic resection of juvenile angio /f_i broma using image guidance (merged computed tomography and magnetic res onance imaging scans). Figure 51.46 Epistaxis balloon catheter.

Management of epistaxis

Anterior bleeding from Kiesselbach’s plexus may be controlled by silver nitrate cautery under local anaesthesia. Even in more posterior epistaxis, the bleeding point can often be identified using rigid nasendoscopy and controlled with the use of a topical vasoconstrictor, and then dealt with directly using electrocautery . However, posterior bleeding, as seen in the Sir William Osler , 1849–1919, Professor of Medicine, successively at McGill University , Montreal, Canada, University of Pennsylvania, Philadelphia, PA, and Johns Hopkins University , Baltimore, MD, USA, finally becoming Regius Professor of Medicine at Oxford University , Oxfor Colin B Holman , 1917–2008, American radiologist, Mayo Clinic. W Eugene Miller , American radiologist, Mayo Clinic, with Colin Holman described the eponymous sign on plain radiographs in 1965. Erik Adolf von Willebrand , 1870–1949, physician, Diakonissanstaltens Hospital, Helsinki, (Helsingfors), Finland, described hereditary pseudohaemophilia in 1926. impregnated ribbon gauze or a non-absorbable sponge. There are also many haemostatic, absorbable materials that can be used to pack the nose to help control bleeding . An alternative to anterior packing is the use of an inflatable epistaxis balloon catheter (Figure 51.46). The catheter is passed into the nose and the distal balloon is inflated in the nasopharynx to secure it. The proximal balloon, which is sausage shaped, is then inflated within the nasal fossa to compress the bleeding point. Although usually e ff ective, they can be uncomfortable. Postnasal packing may be required in refractory cases whereby a gauze pack is positioned in the nasopharynx under general anaesthesia. Endoscopic sphenopalatine artery clip - - ping is an e ff ective treatment for significant epistaxis not responding to direct cautery or nasal packing .). For uncontrolled life-threatening epistaxis in which the above methods have prov ed ine ff ective, haemostasis is secured by vascular ligation. Depending on the origin of bleeding it ma y be necessary to ligate the internal maxillary artery in the pterygopalatine fossa (which can be accessed endoscopically) and the anterior and posterior ethmoidal arteries. An alter - native measure is external carotid artery ligation above the origin of the lingual artery . Another option is to involve the interventional radiologist for possible embolisation. It is also important to recognise, and treat, any factors contributing to). the e pistaxis, such as clotting or platelet abnormalities. d, UK, in 1904.

Figure 51.44 Hereditary haemorrhagic telangiectasia (Osler’s disease) showing multiple telangiectasia.

In HHT , anterior nasal packing is best avoided if at all possible because it is most likely to lead to further mucosal trauma and bleeding. High-dose oestrogen induces squamous metaplasia of the nasal mucosa and has been used e ff ectively in treating this condition. Medications that block vessel growth, such as bevacizumab, and those that slow the disintegration of clots, such as tranexamic acid, help reduce the bleeding asso ciated with HHT . There are also surgical options, including cautery/ablation of the telangiectasia, septodermoplasty or surgical closure of the nostril (Y oung’s procedure).

Figure 51.45 Endoscopic resection of juvenile angio /f_i broma using image guidance (merged computed tomography and magnetic res onance imaging scans). Figure 51.46 Epistaxis balloon catheter.

Neoplasms

Neoplasms

Exostosis is an area of hyperostosis rather than a neoplasm that arises from the bone of the ear canal in individuals who swim in cold water (synonym ‘surfer’s ear’) (Figure 51.14 treatment is required unless the exostosis obstructs the canal. Osteomas are true neoplasms, often singular and more lateral than exostosis. Other benign tumours include papillomas and adenomas. Malignant primary tumours of the external ear are either basal cell or squamous cell carcinomas (Figure 51.15 Summary box 51.3 - Types of otitis externa /uni25CF /uni25CF /uni25CF). No /uni25CF /uni25CF).

Figure 51.14 Exostoses grow from the bony part of the ear canal in response to cold and so are found in swimmers, surfers and divers. Treatment is only required if the exostoses occlude the ear canal. Figure 51.15 Squamous cell carcinomas of the external ear usually originate from the pinna. In this case the tumour is growing from the canal (courtesy of Mr P Beasley). Acute bacterial otitis externa is very common and painful; treat with topical steroid and antibiotic drops Systemic antibiotics should be reserved for cellulitis of the pinna Chronic otitis externa needs the underlying dermatitis to be treated Fungal otitis externa itches and can be diagnosed by the presence of hyphae and spores; treat with meticulous cleaning and stop antibiotics Necrotising otitis externa is a progressive skull base infection that occurs in immunocompromised individuals and can be life-threatening; intensive long-term antibiotic treatment is required

Both may present as ulcerating or crusting lesions that grow slowly and may be ignored by elderly patients. Squamous cell carcinomas metastasise to the parotid and/or neck nodes. The ear canal may be invaded by tumours from the parotid gland and postnasal space carcinomas, which ‘creep’ up the Eusta chian tube. All resectable malignant tumours of the ear are treated primarily with surgery , with or without the addition of radiation therapy .

(b) Figure 51.16 (a) Traumatically perforated tympanic membrane. /uni00A0 (b) The same tympanic membrane 2 days later (courtesy of Dr Christian Deguine). (Reproduced with permission from O’Donoghue GM, Bates GJ, Narula A. Clinical ENT : an illustrated textbook. Oxford: Oxford University Press, 1991.)

Neoplasms

Middle ear tumours are rare, with the most common being a glomus tumour (Figure 51.25). Glomus tumours are para - - gangliomas arising from non-chroma ffi n paraganglionic tissue (the carotid body tumour arising in the neck is an example of this type of tumour). In the temporal bone, two types of glomus tumour are recognised and classification depends on the location: glomus tympanicum (arising in the middle ear) - and glom us jugulare (arising next to the jugular bulb). Symptoms include pulse synchronous tinnitus and conduc - tive and sensorineural hearing loss. Palsies of CNs VII, IX, X, XI and/or XII may occur. The classic sign is a cherry-red mass lying

behind the tympanic membrane. The treatment of choice is preoperative embolisation followed by surgical exci - sion. Radiotherapy is also e ff ective. Squamous cell carcinoma may also occur within the mid - dle ear. It usually presents with deep-seated pain and a blood - stained discharge. Facial paralysis often occurs. Squamous carcinomas usually arise in a chronically discharging ear and can arise in a chronically infected mastoid cavity . Radical sur - gical excision with or without radiotherapy provides the only chance of cure. Summary box 51.6 Neoplasms of the middle ear /uni25CF /uni25CF

(b) Figure 51.23 Section of normal stapes (a) and section of stapes affected by otosclerosis (b) . Figure 51.24 The stapedotomy operation showing the piston linking the incus to the vein graft, left ear. (b) Figure 51.25 (a, b) Glomus tumour in the middle ear, left ear (courtesy of Professor Peter Rea, Leicester). Highly vascular glomus tumours are rare and may present with pulsatile tinnitus Squamous cell cancer usually presents with pain and facial paralysis

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Otitis media with effusion (glue ear)

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Otitis media with e ff usion (OME) is a middle ear e ff usion with no evidence of infection. It has a bimodal incidence a ff ecting 40% of 2-year-olds (age of starting nursery) and 20% of 5-year-olds (age of starting school). It arises mainly in the winter months, suggesting an infective aetiology . Infection and inflammation of the immature Eustachian tube results in poor middle ear ventilation, negative pressure and the transudation of fluid. The following symptoms may be associated with glue ear: /uni25CF hearing impairment, which often fluctuates; /uni25CF delayed speech; /uni25CF behavioural problems; /uni25CF recurrent ear infections (the exudate is an ideal culture me dium for microorganisms); /uni25CF reading and learning di ffi culties at school. Antonio Maria Valsalva , 1666–1723, Italian physician and anatomist. Otoscopic findings with glue ear The otoscopic findings of exudative glue ear are of a dull drum that is immobile on pneumatic otoscopy . The tympanic membrane is retracted and radial blood vessels may be present (Figure 51.19). In children first presenting with bilateral glue ear, 50% will be better within 12 weeks, therefore a ‘wait and watch’ policy is appropriate. If a bilateral conductive hearing loss persists, reduced IQ and behaviour changes. there is some evidence of However, speech delays are reversed by age 8. Medical treatment is of limited value. Valsalva manoeu - ® device (Figure 51.20) are worth trying vres and the Otovent for patients old enough to comply in an attempt to improve Eustachian tube function. Surgical insertion of ventilation tubes (grommets) (Figure 51.21) and adenoidectomy are e ff ective and should be discussed if there is no resolution after - a period of watchful waiting. A middle ear e ff usion in adults is often associated with an upper respiratory tract infection.

Figure 51.17 Acute otitis media of the left ear. Note the bulging tym panic membrane. Figure 51.18 Child with acute mastoiditis whose tympanic membrane is shown in

Figure 51.16 . Figure 51.19 The initial serous transudate of glue ear, left ear (courtesy of Dr Christian Deguine). (Reproduced with permission from O’Dono

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ghue GM, Bates GJ, Narula A. Clinical ENT: an illustrated textbook . Oxford: Oxford University Press, 1991.) ® Figure 51.20 Otovent device.

A persistent unilateral e ff usion in an adult requires examina tion of the postnasal space to exclude obstructive nasopharyn geal carcinoma, which is the most common carcinoma in men in southern China. Summary box 51.5 AOM and OME /uni25CF /uni25CF /uni25CF /uni25CF

Figure 51.21 Ventilation tube in the tympanic membrane, left ear (courtesy of Dr Christian Deguine). AOM is very common but rarely associated with severe complications such as mastoiditis OME is very common in children and usually resolves without treatment Persistent OME and/or recurrent AOM are best treated with grommets and/or adenoidectomy A persistent middle ear effusion in an adult may be caused by a nasopharyngeal carcinoma; this is commonest in people from southern China

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Figure 51.21 Ventilation tube in the tympanic membrane, left ear (courtesy of Dr Christian Deguine). AOM is very common but rarely associated with severe complications such as mastoiditis

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Otosclerosis

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This is an autosomal dominant condition of variable pene - trance in which excess bone is laid down around the footplate of the stapes, impeding mobility of the stapes and resulting in a conductive hearing loss (Figure 51.23). A diagnosis should be suspected in any patient with a conductive hearing loss and a normal tympanic membrane. The trea tment options are simple reassurance, a conven - tional hearing aid, a stapedotomy operation or bone conduc - - tion hearing aid (Figure 51.24). Otosclerosis

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PARANASAL SINUSES

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Internal inspection of the nasal fossae can be achieved to a limited extent with the use of a Thudichum speculum. The anterior nasal septum, nasal vestibule and anterior inferior turbinate can be assessed. A more detailed examination of the nose is possible with the use of either rigid or flexible endoscopes and camera and the image is displayed on a monitor.

Figure 51.38 Rigid nasendoscope.

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Presbycusis

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Presbycusis is characterised by a gradual loss of hearing in both ears, with or without tinnitus. The hearing loss usually a ff ects the higher frequencies and a classical audiogram is shown in Figure 51.28 . The consonants of speech lie within the high-frequency range, which makes speech discrimination di ffi cult. Many patients with presbycusis are concerned that they may lose their hearing completely and need reassurance. Hear ing aid technology has improved dramatically over recent years and most patients can derive benefit (Figure 51.29). Charles Howard Usher , 1865–1942, ophthalmologist, Aberdeen Royal Infirmary , Aberdeen, UK. Vaughan Pendred , 1869–1946, general practitioner, Durham, UK. Anton Jervell , 1901–1987, physician, University of Oslo, Oslo, Norway . Fred Lange-Nielsen , 1919–1989, physician and jazz musician, Oslo, Norway . Petrus Johannes Waardenburg , 1886–1979, ophthalmologist, Utrecht, The Netherlands. Arthur Cecil Alport , 1880–1959, Professor of Medicine, King Fuad I Hospital, University of Cairo, Egypt. Gunnar B Stickler , 1925–2010, pediatrician, Mayo Clinic, USA.

Figure 51.27 Multichannel cochlear implant (Cochlear Corporation).

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RHINOSINUSITIS

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Rhinosinusitis is inflammation of the sinonasal mucosa and is defined as the presence of nasal congestion or nasal discharge and at least one of facial pain or hyposmia with endoscopic and/or CT changes to confirm the diagnosis. It can be divided into acute rhinosinusitis (ARS) and chronic rhinosinusitis (CRS) depending on the timing of symptoms. Symptoms are present for less than 12 weeks in ARS and more than 12 weeks in CRS. Austen Y oung , 1914–2005, ENT surgeon, She ffi eld, UK, first described surgical closure of the nostril for the management of atrophic rhinitis. Epistaxis /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF

The most common causes are nose picking, hypertension and anticoagulant therapy Young people bleed from the anterior septum – Kiesselbach’s plexus Elderly people bleed from the posterior part of the nose Epistaxis is ideally treated with direct cautery to the bleeding point under endoscopic guidance Silver nitrate cautery can be used to control anterior bleeding Moderate bleeding may require anterior nasal packing Severe bleeding may require anterior and posterior nasal packing Persistent bleeding may require endoscopic sphenopalatine artery ligation

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Radiological investigation

Radiological investigation

Computed tomography (CT) scanning of the temporal bones is commonly performed before mastoid surgery to show detailed individual anatomy , as well as alerting the surgeon to anatomical variants. Pus, bone and air are shown well on high-resolution CT (Figure 51.8). Magnetic resonance imaging (MRI) is better than CT at imaging soft tissue (e.g. facial and auditory nerve) and is the best method for imaging tumours of the acoustic nerves (Figure 51.9). Di ff usion-weighted MRI is also commonly used to detect recurrent cholesteatoma.

Figure 51.7 Audiometry. The patient sits in a soundproof room and the audiologist presents sounds at different thresholds and records the responses. Figure 51.8 Computed tomography scan showing a normal left ear. The air-/f_i lled middle ear and the incus and stapes, the semicircular canals and internal acoustic meatus can be seen. In the right ear the entire

middle ear and mastoid are opaque and /f_i lled with soft tissue. This is the typical appearance of a cholesteatoma.

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Septal deformity

Septal deformity

Deviation of the nasal septum may occur naturally or arise as a result of nasal trauma and is readily apparent on anterior rhinoscopy (Figure 51.40). Surgical correction can be achieved by a submucous resection (SMR) of the septum where the deformed septal cartilage is excised while preserving a caudal and dorsal strut for support (Figure 51.41). The alternative is a septoplasty procedure during which the septal cartilage is preserved but the anatomical abnormalities giving rise to its deformity , such as a twisted maxillary crest or inclination of the bony septum posteriorly , are corrected. Complications of septal surgery include septal perforation. If too much cartilage is excised in the SMR procedure, loss of support to the dorsum of the nose may result in a supra-tip depression or drooping of the tip of the nose. Septal deformity

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Septal injury

Septal injury

A blunt injury of moderate force may lead to lateral displace - ment or deformity of the septal cartilage, restricting the nasal airway . Unlike the nasal bones the nasal septum cannot be manipulated back into position and requires a formal septo - plasty procedure to restore the anatomy and the patency of the nasal airwa ys. - Bleeding under the mucoperichondrium of the septum will cause a septal haematoma and nasal obstruction. Untreated, a septal haematoma will progress to abscess forma tion and ulti - mately result in necrosis of the septal cartilage, septal perfora - tion and nasal collapse. A septal haematoma should be treated - by incision and drainage of the blood clot, insertion of a small s and silicone drain and packing of the nasal fossa. A broad-spectrum - prophylactic antibiotic should be prescribed. Summary box 51.9 Nasal trauma /uni25CF /uni25CF /uni25CF /uni25CF

Do not overlook a septal haematoma Displaced nasal bone fractures should be reduced within 3 /uni00A0 weeks of injury Severe persistent epistaxis after trauma suggests lacrimal bone fracture and injury to the anterior ethmoid artery CSF rhinorrhoea indicates a fracture involving the anterior skull base with a dural tear

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Septal perforation

Septal perforation

A hole in the nasal septum causes turbulent airflow through the nose and a resulting sensation of nasal blockage, extensive nasal crusting, bleeding and whistling. The causes of septal perforation are listed in Summary box 51.10 . Septal perforations seldom heal spontaneously . A great variety of operations have been described to close septal per forations but none has met with universal success . These have included closing the perforation using cartilage or synthetic material and covering with local flaps. Alternatively , the perf ration may be occluded by inserting a Silastic biflanged pros thesis or ‘septal button’ (Figures 51.42 and 51.43). In some cases, particularly those patients with significant whistling and bleeding from the posterior edge, the perforation can be enlarged and mucosa folded ar ound the posterior edge to sta bilise it. Granulomatosis with polyangiitis is a systemic idiopathic autoimmune disease a ff ecting the nose, lungs and kidneys. Summary box 51.10 Causes of septal perforations /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF James Laurence Little , 1836–1885, Professor of Surgery , The University of V ermont, Montpelier, VT , USA. - o - - - Mucosal granulations on the nasal septum destroy cartilage, producing a septal perforation with saddle deformity of the nose. Laboratory findings include a high erythrocyte sedimen - tation rate, impaired creatinine clearance and antineutrophil cytoplasmic antibodies (c-ANCA) in most cases.

Trauma Iatrogenic following septal surgery Nose picking Following a septal haematoma from nasal injury Infection Syphilis Tuberculosis Vasculitis Granulomatosis with polyangiitis Tumours Toxins Chrome salts Cocaine Idiopathic Narrow airway Contralateral inferior turbinat e Deviated septum hypertrophy Figure 51.40 Coronal section through the anterior nasal fossae with deviated nasal septum to the right side. Frontal sinus Nasal bone Perpendicular plate ethmoid Dorsal strut Septal cartilage Vomer that can be excised in SMR Palatine bone Caudal strut Figure 51.41 Area of cartilage that can be removed in submucous resection (SMR) leaving dorsal and caudal strut for support. Septal perforation Septal prosthesis Figure 51.42 Anterior and lateral views of septal perforation occluded with a prosthesis.

Septal perforation

A hole in the nasal septum causes turbulent airflow through the nose and a resulting sensation of nasal blockage, extensive nasal crusting, bleeding and whistling. The causes of septal perforation are listed in Summary box 51.10 . Septal perforations seldom heal spontaneously . A great variety of operations have been described to close septal per forations but none has met with universal success . These have included closing the perforation using cartilage or synthetic material and covering with local flaps. Alternatively , the perf ration may be occluded by inserting a Silastic biflanged pros thesis or ‘septal button’ (Figures 51.42 and 51.43). In some cases, particularly those patients with significant whistling and bleeding from the posterior edge, the perforation can

be enlarged and mucosa folded ar ound the posterior edge to sta bilise it. Granulomatosis with polyangiitis is a systemic idiopathic autoimmune disease a ff ecting the nose, lungs and kidneys. Summary box 51.10 Causes of septal perforations /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF James Laurence Little , 1836–1885, Professor of Surgery , The University of V ermont, Montpelier, VT , USA. - o - - - Mucosal granulations on the nasal septum destroy cartilage, producing a septal perforation with saddle deformity of the nose. Laboratory findings include a high erythrocyte sedimen - tation rate, impaired creatinine clearance and antineutrophil cytoplasmic antibodies (c-ANCA) in most cases.

Trauma Iatrogenic following septal surgery Nose picking Following a septal haematoma from nasal injury Infection Syphilis Tuberculosis Vasculitis Granulomatosis with polyangiitis Tumours Toxins Chrome salts Cocaine Idiopathic Narrow airway Contralateral inferior turbinat e Deviated septum hypertrophy Figure 51.40 Coronal section through the anterior nasal fossae with deviated nasal septum to the right side. Frontal sinus Nasal bone Perpendicular plate ethmoid Dorsal strut Septal cartilage Vomer that can be excised in SMR Palatine bone Caudal strut Figure 51.41 Area of cartilage that can be removed in submucous resection (SMR) leaving dorsal and caudal strut for support. Septal perforation Septal prosthesis Figure 51.42 Anterior and lateral views of septal perforation occluded with a prosthesis.

Sudden sensorineural hearing loss

Sudden sensorineural hearing loss

Defined as >30 /uni00A0 dB sensorineural hearing loss at three frequen - - cies within 3 days. History and examination should focus on a cause, which may be infective, neoplastic, traumatic, ototoxic, neurological or autoimmune. Investigations such as MRI are important (1% of acoustic neuromas present as sudden sensorineural hearing loss) but screening blood tests are of low yield where there is nothing in the history to suggest a cause. The majority are idiopathic and the recommended treatment is oral steroids with/without intratympanic steroids with salvage intratympanic steroids for those who do not recover after 2 /uni00A0 weeks. Sudden sensorineural hearing loss

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THE EAR

THE EAR

The mammalian ear is an evolutionary masterpiece. Its highly complex ‘three-dimensional anatomy’ is best learnt by dissect ing cadaver temporal bones. THE EAR

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THE NOSE AND SINUSES BASIC ANATOMY OF THE NOSE AND PARANASAL SINUSES

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The supporting structures of the nose are shown in Figure 51.32 . The septum consists of the anterior quadri - lateral cartilage, the perpendicular plate of the ethmoid and the vomer (Figure 51.33). The lateral wall of the nasal cavity contains the superior, middle and inferior turbinates, which warm and moisten nasal airflow (Figure 51.34). There are paired frontal, sphenoid, maxillary and anterior and posterior ethmoid sinuses. The anterior nasal sinuses (frontal, maxillary and anterior ethmoid) drain into the middle meatus (between the middle turbinate and lateral wall of the nose). The posterior ethmoid and sphenoid sinuses drain into the superior meatus and sphenoethmoidal recess (between the superior turbinate and nasal septum), respectively (Figures 51.35 and 51.36 The nasal fossae and sinuses receive their blood supply via the external and internal carotid arteries. The external carotid artery supplies the interior of the nose via the maxillary and sphenopalatine arteries. The grea ter palatine artery supplies the anteroinferior septum via the incisive canal. The con tribution from the internal carotid artery is via the anterior and posterior ethmoidal arteries, which are branches of the ophthalmic artery (Figure 51.37). All these arteries anasto mose to form a plexus of vessels (Kiesselbach’ s plexus) on the anterior part of the nasal septum. V enous drainage is via the ophthalmic and facial veins and the pterygoid and pharyngeal plexuses. Intracranial drainage into the cavernous sinus via the ophthalmic vein is of particular clinical importance because of the potential for intracranial spread of nasal sepsis. Wilhelm Kiesselbach , 1839–1902, Professor of Otology , Erlangen, Germany . Johann Ludwig Wilhelm Thudichum , 1829–1901, biochemist and general practitioner, London, UK.). -

Orbit Nasal bone Frontal process Upper lateral of maxilla cartilage Lower lateral cartilage Fibroareolar tissue Figure 51.32 The nasal skeleton. Frontal sinus Sphenoid sinus Perpendicular plate of ethmoid Septal cartilage Vomer Anterior nasal spine Palatine bone Figure 51.33 The left side of the nasal septum. Middle turbinate Superior turbinate Inferior turbinate Figure 51.34 The right lateral nasal wall. Frontal sinus Sphenoid A ostium B Openings of SM posterior Frontal recess ethmoid cells MM and ostium C Maxillary ostium IM Openings of anterior Nasolacrimal ethmoid cells duct opening Figure 51.35 The right lateral nasal wall with turbinates removed to show the sinus ostia. A, insertion of superior turbinate; B, insertion of middle turbinate; C, insertion of inferior

turbinate; IM, inferior meatus; MM, middle meatus; SM, superior meatus. Ethmoid sinus Orbit Superior turbinate Superior meatus Middle turbinate Maxillary antrum Middle meatus Inferior turbinate Maxillary ostium Inferior meatus Figure 51.36 Coronal section through the left maxillary and ethmoid sinuses. Anterior Posterior ethmoidal ethmoidal artery artery Kiesselbach’s plexus Sphenopalatine artery Greater palatine Facial artery artery Figure 51.37 Arterial blood supply to the left side of the nasal septum.

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TRAUMA TO THE NOSE AND PARANASAL SINUSES

Fracture of the nasal bones

TRAUMA TO THE NOSE AND PARANASAL SINUSES Fracture of the nasal bones

Blunt injury to the nose may fracture the nasal bones (Figure 51.39). The fracture line can extend into the lacrimal bone and tear the anterior ethmoidal artery , producing cata strophic haemorrhage. This may be delayed, occurring only as the soft-tissue swelling subsides, reducing the tamponade e ff ect on the torn vessel. Violent trauma to the frontal area of the nose can result in a fracture of the frontal and ethmoid sinuses with poten tial extension into the anterior cranial fossa. Dural tear brain injuries, either open or closed, are then at risk from sino nasal ascending infection, which may prog ress to meningitis or brain abscess. CSF rhinorrhoea is a certain sign of a dural tear. CSF rhinorrhoea can be confirmed b y collecting a sample of the fluid and sending for β - transferrin assay . A bony defect 2 in the anterior skull base following trauma can be identified on high-resolution CT . The CSF leak will often settle with conservative management but, if persistent, it can be repaired endoscopically . Management of fractured nasal bones Fractured nasal bones are normally accompanied by extensive overlying soft-tissue swelling and bruising, which may hinder - the assessment of any underlying bony deformity . Reviewing - after 4–5 days when the soft-tissue swelling has diminished will allow a better assessment of any deformity . If there is a significant degree of nasal deformity , this can be corrected by manipulation of the nasal bones under local or general anaesthesia. This should be carried out within 3 weeks of the injury while the bony fragments are still mobile. After this period, if there is significant cosmetic or functional issues, a septorhinoplasty can be performed at least 6 months following the injury .

Figure 51.39 Fracture of the nasal bones with displacement of the bony nasal complex to the right side.

TRAUMA TO THE NOSE AND PARANASAL SINUSES

Fracture

TRAUMA TO THE NOSE AND PARANASAL SINUSES Fracture of the nasal bones

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TUMOURS OF THE NOSE AND SINUSES

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Tumours arising in the nose or paranasal sinuses may present with unilateral nasal obstruction, persistent unilateral anterior rhinorrhoea, postnasal drip, epistaxis, unilateral bloodstained rhinorrhoea, facial swelling or proptosis. TUMOURS OF THE NOSE AND SINUSES

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The external ear

The external ear

The external and middle ear develop from the first two branchial arches. The external ear canal is 3 /uni00A0 cm in length; the outer two-thirds is cartilage and the inner third is bony . The skin on the lateral surface of the tympanic membrane is highly specialised and migrates outwards along the ear canal. As a result of this migration most people’s ears are self-cleaning. The external canal is richly innervated and the skin is tightly Ernst Reissner , 1824–1878, Professor of Anatomy at Dorpat and later at Breslau, Germany (now Wroc ł aw , Poland), described the vestibular membrane of the cochlea in 1851. bound down to the perichondrium so that swelling in this region results in severe pain. The lymphatics of the external ear drain to the retroauric - ular, parotid, retrophar yngeal and deep upper cervical lymph - nodes.

The facial nerve can be damaged by trauma and ear • disease Chronic ear disease can lead to intracranial sepsis • There are two types of hearing loss: conductive and • sensorineural The clinical features of sinus infection, its treatment and • potential complications The diagnosis and management of chronic rhinosinusitis • with and without nasal polyposis The common sinonasal tumours, their presentation, • investigation and principles of treatment

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The inner ear

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The inner ear comprises the cochlea and vestibular labyrinth (saccule, utricle and semicircular canals). These structures are - embedded in dense bone called the otic capsule. The cochlea is a coiled shell of two and three-quarter turns. Within the cochlea is a spiral structure called the cochlear duct (Figure 51.3), which contains endolymph that is partitioned by Reissner's membrane from the perilymph of the scala ves - tib uli and the basilar membrane from the perilymph of the scala tympani. The perilymph sits uninterrupted from the oval window and stapes footplate at the start of the scala vestibuli in continuity with the round window membrane at the end of the scala tympani (Figur e 51.4). The endolymph has a high concentration of potassium, similar to intracellular fluid, and (b) the perilymph has a high sodium concentration and commu nicates with the cerebrospinal fluid (CSF). Maintenance of the ionic gradients is an active process and is essential for neuronal activity . Bartolomeu Eustachio (Eustachius) , 1513–1574, Professor of Anatomy , appointed physician to the Pope in 1547. Alfonso Giacomo Gaspare Corti , 1822–1876, Italian anatomist. There are approximately 15 /uni00A0 500 hair cells in the human cochlea. They are arranged in rows of 3500 inner and 12 /uni00A0 000 outer hair cells. Movement of the stapes footplate causes a pres - sure wave through the perilymph, resulting in vibration of the basilar membrane and a shearing motion between the tops of the hair cells and the tectorial membrane. The inner hair cells act as mechanicoelectric transducers, converting the acoustic signal into an electric impulse. The outer hair cells contain contractile proteins and serve to tune the basilar membrane on which they are positioned. Each inner hair cell responds - to a particular frequency of vibration. When stimula ted, it depolarises and passes an impulse to the cochlear nuclei in the brainstem.

Short process of malleus The majority of the ossicles lie out of Long sight in the attic process of incus Pars /f_l accida Oval Handle of malleus window Pars tensa Stapes Anterior bulge of ear canal Round window Light re /f_l ex niche Figure 51.1 (a) Right tympanic membrane and (b) diagram to illustrate the anatomy of the tympanic membrane and ossicles (courtesy of Dr Christian Deguine). Scala vestibuli Scala media Inner hair cell nerves Cochlear nerve /f_i bres Figure 51.3 The cochlear duct. Posterior Antrum cranial fossa Attic Mastoid M ET TM Hypotympanum Stylomastoid Medial foramen VII Anterior Posterior Lateral Figure 51.2 Diagram of the right ear to show the relationships of the middle ear. ET, Eustachian tube; M, malleus; TM, tympanic membrane; VII, facial nerve (courtesy of Dr Christian Deguine). Reissner's membrane Stria vascularis Tectorial membrane Organ of Corti Tunnel Outer hair cell nerve Tunnel Basilar /f_i bres membrane Scala tympani

The vestibular labyrinth consists of the semicircular canals, utricle and saccule and their central connections. The three semicircular canals are arranged in the three planes of space at right angles to each other. Like the auditory system, hair cells are present. In the lateral canals, the hair cells are embedded in a gelatinous cupula. Shearing forces, caused by angular move ments of the head, produce hair cell movements and generate action potentials. In the utricle and saccule the

hair cells are embedded in an otoconial membrane, which contains parti cles of calcium carbonate. These respond to changes in linear acceleration and the pull of gravity . Impulses are carried centrally by the vestibular nerve and connections are made to the spinal cord, cerebellum and exter nal ocular muscles. Its function is to r ecord the position and movements of the head.

Oval window Stirrup (stapes) Anvil (incus) Hammer (malleus) Eardrum (tympanum) Air Air Ear canal Endolymph Round window Perilymph Eustachian tube Inner ear Middle ear

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The sensory nerve supply

The sensory nerve supply

The external ear is supplied by the auriculotemporal branch of the trigeminal nerve (cranial nerve [CN] V) and the greater auricular nerve (C2/3), together with branches of the lesser occipital nerve (C2). CNs VII, IX and X also supply small sensory branches to the external ear. The middle ear is supplied by the glossopharyngeal nerve (CN IX). This complicated and rich sensory innervation means that referred otalgia is common and may originate from the nor mal area of distribution of any of the above nerves. A classic e xample is the referred otalgia caused by cancer of the larynx or hypopharynx. Taking a thorough history is the most important part of the assessment; the symptoms that need to be enquir ed after are listed in Table 51.1 . Friedrich Heinrich Adolf Rinne , 1819–1868, otologist, Göttingen, Germany , described this test in 1855. Friedrich Eugen Weber-Liel , 1832–1891, otologist, University of Berlin and Jena, Germany , described the operation of tenotomy of the tensor tympani used for certain forms of partial deafness. Summary box 51.1 A p p l i e d a n a t o m y /uni25CF - /uni25CF /uni25CF - /uni25CF /uni25CF - /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF -

nerves Organ of Corti Tectorial membrane Figure 51.4 Perilymph pressure wave (adapted from lumenlearning.com; originally taken from Urone PP , Hinrichs R. College Physics , OpenStax, 2012 under https://creativecommons.org/licenses/ by/4.0/) . The skin on the outer surface of the eardrum migrates outwards so that the ear canal is ‘self-cleaning’ Infection of the middle ear and mastoid can easily spread to the cranial cavity The facial nerve pursues a tortuous course through the middle ear The ear has a rich sensory innervation so that ‘referred otalgia’ is common Cancer of the larynx or lower pharynx can present with otalgia TABLE 51.1 History taking. Ask about: Earache, pain and itch Hearing loss Discharge: type, quantity and smell Tinnitus Vertigo Facial weakness Speech and development (in children) Past history: head injury, baro- or noise trauma, ototoxics, family history and previous ear surgery

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The tympanic membrane and middle ear

The tympanic membrane and middle ear

The anatomy of the tympanic membrane and ossicles is shown in Figure 51.1 . The relations of the middle ear are important - (Figure 51.2). The tympanic membrane and ossicles act as a transformer of vibrations in the air to vibrations within the fluid-filled inner ear. The tympanic membrane and middle ear

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Tinnitus

Tinnitus

Tinnitus is the perception of sound when no external sound source is present. It may have an extrinsic cause; for example, the pulsatile tinnitus of a glomus tumour. Usually , however, the tinnitus is generated within the internal auditory pathway . - Thirty per cent of people will experience tinnitus at some time in their lives. Tinnitus frequently accompanies presbycusis, as well as any other condition that a ff ects hearing. Most individu - als habituate to the presence of tinnitus but in some patients it proves intrusive. Treatment is with reassurance, masking and hearing aids (for patients with hearing loss). Tinnitus

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Trauma

Trauma

A haematoma of the pinna occurs when blood collects under the perichondrium. The cartilage receives its blood supply from the perichondrial layer and will die if the haematoma is not evacuated, resulting in a so-called cauliflower ear. A generous incision under anaesthetic, with a pressure dressing or compressive sutures and antibiotic cover, is recommended (Figure 51.10). Foreign bodies in the ear canal are most easily removed at the first attempt by an experienced practitioner with the aid of a microscope. General anaesthesia may be required in children and those with learning di ffi culties. Batteries need to be remo ved within the hour (Figure 51.11). Summary box 51.2 Trauma of the external ear /uni25CF /uni25CF /uni25CF

Figure 51.9 Computed tomography scan showing a vestibular schwannoma occluding the left internal acoustic meatus (arrow). A haematoma of the pinna requires thorough drainage, antibiotics and a compressive dressing or sutures Foreign bodies in the ear canal are most easily removed at the /f_i rst attempt with the aid of a microscope Batteries need to be removed urgently

Trauma

Trauma to the middle ear can result in a perforated tympanic membrane (Figure 51.16a); 90% of such perforations heal spontaneously within 6 weeks (Figure 51.16b). Trauma can also result in ossicular discontinuity and it is usually the incus that is displaced. A damaged ossicular chain and tympanic membrane are repaired by ossiculoplasty or tympanoplasty , respectively . Summary box 51.4 Congenital anomalies and trauma of the middle ear /uni25CF /uni25CF /uni25CF /uni25CF

Congenital anomalies may be isolated or associated with general congenital deformities Traumatic perforations of the tympanic membrane usually heal spontaneously but explosive and welding injuries do not A myringoplasty is an operation that repairs the tympanic membrane With severe head trauma the incus can be displaced, which leads to a conductive hearing loss

Trauma

Noise exposure Hair cells within the cochlea are damaged by sudden acoustic trauma (blast injury or gunfire) or prolonged exposure to exces - sive noise. The sensorineural hearing loss that results is greatest - between 3 and 6 /uni00A0 kHz and is often accompanied by tinnitus (Figure 51.30). The law in the UK requires that workers are protected from noise . Head injury The otic capsule is the hardest bone in the body but, if trauma to the head is severe, temporal bone fractures may occur. These are traditionally described as either longitudinal (80%) or transverse (20%); however, the majority have longitudinal and transverse components. Longitudinal fractures may lead to fracture of the external auditory canal, conductive hearing loss and CSF otorrhoea. Transverse fractures may involve Charles Skinner Hallpike , 1900–1979, aural surgeon, National Hospital for Neurology and Neurosurgery , London, UK. John W Epley , contemporary , Director, Portland

Otology Clinic, Portland, OR, USA, established his clinic in 1975; he developed the Epley manoeuvre for treating benign paroxysmal positional vertigo (BPPV). the facial nerve, leading to palsy , and labyrinth, leading to a sensorineural hearing loss that is permanent. Profound vertigo occurs initially , followed by gradual compensation. Drug ototoxicity Antibiotics such as aminoglycosides, vancomycin and erythro - mycin, loop diuretics such as frusemide, chemotherapy agents such as cisplatin and carboplatin, and salicylates such as aspirin and quinine are all ototoxic. Recognition of risk factors, such as poor renal function in patients being treated with amino - glycosides, is therefore important. Although many topical ear drops contain aminoglycosides, there is little evidence that short periods of topical treatment cause sensorineural hearing loss.

–20 –10 0 10 20 30 40 50 60 70 Hearing level (dB ISO) 80 90 100 110 120 125 250 500 1000 2000 4000 6000 Frequency (Hz) Figure 51.28 Typical audiogram of pr esbycusis: (a) right ear; Figure 51.29 Modern hearing aid. –20 –10 0 10 20 X X 30 X 40 50 X 60 70 Hearing level (dB ISO) 80 X 90 X 100 110 120 125 250 500 1000 2000 4000 6000 Frequency (Hz) (b) left ear.

Trauma

A haematoma of the pinna occurs when blood collects under the perichondrium. The cartilage receives its blood supply from the perichondrial layer and will die if the haematoma is not evacuated, resulting in a so-called cauliflower ear. A generous incision under anaesthetic, with a pressure dressing or compressive sutures and antibiotic cover, is recommended (Figure 51.10). Foreign bodies in the ear canal are most easily removed at the first attempt by an experienced practitioner with the aid of a microscope. General anaesthesia may be required in children and those with learning di ffi culties. Batteries need to be remo ved within the hour (Figure 51.11). Summary box 51.2 Trauma of the external ear /uni25CF /uni25CF /uni25CF

Figure 51.9 Computed tomography scan showing a vestibular schwannoma occluding the left internal acoustic meatus (arrow). A haematoma of the pinna requires thorough drainage, antibiotics and a compressive dressing or sutures Foreign bodies in the ear canal are most easily removed at the /f_i rst attempt with the aid of a microscope Batteries need to be removed urgently

Trauma

Trauma to the middle ear can result in a perforated tympanic membrane (Figure 51.16a); 90% of such perforations heal spontaneously within 6 weeks (Figure 51.16b). Trauma can also result in ossicular discontinuity and it is usually the incus that is displaced. A damaged ossicular chain and tympanic membrane are repaired by ossiculoplasty or tympanoplasty , respectively . Summary box 51.4 Congenital anomalies and trauma of the middle ear /uni25CF /uni25CF /uni25CF /uni25CF

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–20 –10 0 10 20 30 40 50 60 70 Hearing level (dB ISO) 80 90 100 110 120 125 250 500 1000 2000 4000 6000 Frequency (Hz) Figure 51.28 Typical audiogram of pr esbycusis: (a) right ear; Figure 51.29 Modern hearing aid. –20 –10 0 10 20 X X 30 X 40 50 X 60 70 Hearing level (dB ISO) 80 X 90 X 100 110 120 125 250 500 1000 2000 4000 6000 Frequency (Hz) (b) left ear.