NEET - SS

ENT GROUP

LMR | Notes

(LAST MINUTE REVISION NOTES)



NEET-SS ENT GROUP · LMR NOTES

Index

5 sections · 205 pages · 233 topics · 1028 subtopics

Otology and Audiology	Rhinology	Laryngology, Head and Neck	Pediatric ENT	Miscellaneous
1–47	48–91	92–156	157–190	191–205
48 topics	41 topics	97 topics	35 topics	12 topics

Otology and Audiology pp. 1–47

Embryology and anatomy I 1
- External ear embryology 1
- Middle-ear embryology 1
- Middle-ear compartments 1

Embryology and anatomy II 1
- Mastoid/temporal-bone landmarks 2
- Facial recess 2
- Prussak space 2
- Major surgical danger relationships 2
- Inner-ear anatomy 2

Physiology I 3
- Sound terminology 3
- Middle ear as impedance transformer 3
- Cochlear travelling wave 3
- Hair-cell transduction 3

Physiology II 4
- Hair cells 4
- Semicircular canals 4
- Otolith organs 4
- VOR 4
- Vestibulospinal function 4

Clinical examination of ear and hearing 5
- Diagnostic algorithm 5
- History: high-yield discriminators 5
- Otoscopy 5
- Retraction-pocket assessment 5
- Tuning forks 5
- Bedside vestibular/CN examination 6

External ear disease I 6
- Furunculosis 6
- Otomycosis 6
- Chronic otitis externa 6
- Perichondritis 7

External ear disease II 7
- Exostosis vs osteoma 7
- Keratosis obturans vs EAC cholesteatoma 7
- EAC soft-tissue lesions 7
- Necrotizing otitis externa 7

OTOLOGY AND AUDIOLOGY (CONTINUED)

Congenital ear abnormalities 8
- External ear 8
- Preauricular anomalies 8
- Congenital EAC atresia 8
- Inner-ear malformations 8
- Named membranous malformations 9

Eustachian tube disorders 9
- Core physiology 9
- Obstructive ETD 9
- Patulous ET 9
- Medical management of obstructive ETD 10

Acute otitis media and otitis media with effusion 10
- AOM 10
- Recurrent AOM 11
- OME 11

Chronic otitis media 11
- Modern classification 11
- TM perforation: hearing-loss determinants 11
- Tympanosclerosis 11
- Pars-flaccida retraction progression 12

Cholesteatoma 12
- Pathogenesis 12
- Cholesteatoma vs retraction 12

Complications of otitis media 13
- First recognition rule 13
- Extracranial/intratemporal 13
- Labyrinthine fistula 13
- Gradenigo syndrome 13
- Intracranial complications 13

Middle-ear surgery I 14
- Surgical decision 14
- Canal-wall-down mastoidectomy 14
- Canal-wall-up mastoidectomy 14
- Limited attic disease 14
- Key surgical landmarks 14

Middle-ear surgery II 15
- Revision mastoidectomy 15
- Tympanoplasty 15
- Wullstein classification 15
- Ossiculoplasty 15

Detailed Index

MIDDLE-EAR SURGERY II (CONTINUED)

- Surgery complications 15

Otosclerosis I 15

- Pathology and classic site 15
- Named clinical signs: Schwartze sign, paracusis Willisii, Carhart notch 16
- Speech discrimination, tympanometry and acoustic reflex 16

Otosclerosis II 16

- Fenestral vs cochlear otosclerosis 16
- HRCT temporal bone 16
- Treatment algorithm 16
- Stapedotomy 16

Hearing loss 17

- Three major types 17
- Audiogram pattern recognition 17
- Presbyacusis 17
- Noise-induced hearing loss 17
- Ototoxic hearing loss 17
- Asymmetric SNHL 18
- Speech discrimination trap 18

Sudden sensorineural hearing loss 18

- Emergency algorithm 18
- Causes to consider 18
- Investigation 18
- Treatment: first-line and intratympanic steroids 19

Other hearing disorders 19

- Autoimmune inner-ear disease 19
- Cogan syndrome 19
- Behçet disease 19
- Signature mismatch 19
- Recruitment 20
- Cochlear synaptopathy / hidden hearing dysfunction 20
- Non-organic hearing loss 20
- Retrocochlear localization pattern 20
- Master pattern table 20

Audiology: terminology and tests 20

- Decibel essentials 21
- Pure-tone audiometry 21
- Audiogram interpretation 21
- Masking algorithm 21
- Speech audiometry 21
- Recruitment vs hyperacusis 21
- MCL/UCL 21
- Audiogram traps 21

Objective and electrophysiological hearing assessment 21

- Tympanometry 21
- Acoustic reflex 22
- OAE 22
- ABR 22
- OAE vs ABR 22
- ASSR 22
- ECochG 22
- Auditory neuropathy spectrum disorder 22
- ENoG vs EMG 22

OTOLOGY AND AUDIOLOGY (CONTINUED)

Hearing aids 22

- Core signal chain 22
- Components 22
- Core electroacoustic terms 23
- Why compression matters 23
- Hearing-aid styles 23
- Open-fit principle 23
- Venting 23
- Directional microphones 23
- Binaural fitting 23
- Verification 23

Bone-conduction and middle-ear implants 23

- Bone-conduction principle 23
- Conventional vs bone-anchored 24
- CROS/BiCROS 24
- Middle-ear implants 24

Cochlear implantation I: selection and principles 24

- Candidate algorithm 24
- Paediatric vs adult 24
- Critical-period concept 24
- Pre-op imaging 25
- Absolute anatomical trap 25
- Contraindication framework 25
- CI vs HA 25

Cochlear implantation II: surgical technique 25

- Standard route 25
- Critical structures 25
- Facial recess 25
- Electrode 25
- Round-window vs cochleostomy 25
- Hearing preservation 25
- Intra-operative testing 25
- Programming 25
- Major complications 26

Difficult cochlear implantation 26

- Inner-ear malformations 26
- CSF gusher 26
- Cochlear ossification 26
- Aberrant facial nerve 26
- Electrode problems 26
- Facial stimulation 26
- Revision CI 26
- High-yield comparison 26

Auditory brainstem implants 27

- Why ABI outcomes are poorer 27
- Paediatric trap 27
- Core distinction 27
- ABI pathway 27
- Major indications 27
- Surgical concept 27

Ototoxicity 27

- Core mechanism 27
- Drug pattern table 27

Detailed Index

OTOTOXICITY (CONTINUED)

- Cisplatin pattern 27
- Synergistic combination 27
- Monitoring 27
- Vestibulotoxicity trap 28

Tinnitus and hyperacusis 28

- Tinnitus classification 28
- Pulsatile tinnitus: think vascular until explained 28
- Work-up algorithm 28
- Tinnitus + asymmetric SNHL 28
- Subjective tinnitus treatment 28
- Habituation chain 28
- Hyperacusis: Reduced tolerance of ordinary environmental sounds. 28
- Hyperacusis vs recruitment 28
- Somatosensory/somatic tinnitus clues 28

Vestibular disorders 28

- Ménière disease 28
- Vestibular migraine 29
- BPPV 30
- Bilateral vestibular failure 30

Vestibular examination and investigation 30

- Head impulse test 30
- Dix–Hallpike 30
- VOR 30
- Caloric testing 31
- VHIT 31
- Rotary chair 31
- VEMP 31
- Subjective visual vertical 31
- Posturography 31

Vestibular rehabilitation 31

- Three fundamental strategies 31
- VOR adaptation 31
- VOR x1 31
- VOR x2 31
- Progression 31
- Balance retraining 32
- Unilateral vs bilateral loss 32
- Vestibular suppressants 32

Vestibular schwannoma: clinical and diagnosis 32

- Clinical progression 32
- Clinical clues 32
- Hitzelberger sign 32
- MRI sequences 32
- ABR 32

Vestibular schwannoma: management 32

- Management algorithm 32
- Main strategies 32
- Surgical approaches 33
- Radiosurgery 33
- NF2 33
- Hearing-preservation trap 33

OTOLOGY AND AUDIOLOGY (CONTINUED)

Cerebellopontine angle tumours 33

- CPA differential 33
- Epidermoid 33
- Arachnoid cyst 33
- Meningioma 33
- Facial nerve schwannoma 33
- Lower cranial nerve schwannoma 33
- Important vascular differential 33
- Imaging trap 33

Facial nerve I: anatomy and localization 34

- Branches 34
- Functional localization 34
- UMN vs LMN 34
- Intratemporal course 34
- IAC fundus arrangement 34
- Key surgical landmarks 34
- Major LMN causes 34

Facial nerve II: disorders and sequelae 34

- Bell palsy 34
- Late sequelae 35
- Master rapid-recall table 35

Imaging I: temporal-bone CT 36

- HRCT technique 36
- What CT is best for 36
- Cholesteatoma CT checklist 36
- Otosclerosis 36
- SSCD 36
- Facial canal 37
- Congenital CT checklist 37

Imaging II: MRI, IAC and skull base 37

- MRI sequence recognition 37
- Signal traps 37
- Cholesteatoma 37
- Epidermoid vs arachnoid cyst 37
- Vestibular schwannoma 37
- Meningioma 37
- Paraganglioma 37
- Petrous-apex cholesterol granuloma 37
- Vascular imaging 37

Temporal-bone trauma 38

- Clinical findings 38
- Facial palsy 38
- Trauma algorithm 38
- Longitudinal vs transverse 38
- CSF leak 38
- Ossicular injury 38
- Perilymphatic fistula 38
- Delayed complications 38
- Carotid injury 38

Tumours of ear and temporal bone 38

- External auditory canal SCC 38
- Pinna tumours 39
- Adenoid cystic carcinoma 39
- Other ceruminous tumours 39

Detailed Index

OTOLOGY AND AUDIOLOGY (CONTINUED)

Skull base and petrous apex lesions 39
- Master differential 40

Otalgia 40
- Primary vs referred 40
- Sensory map 40

Congenital and skull-base surgical integration 41
- The operative principle 41
- Atresia + ossicular abnormality 41
- Inner-ear malformation classification 41
- Imaging-to-surgery algorithm 42
- Cochlear nerve deficiency 42
- Why CT + MRI both matter 42
- Facial nerve anomaly 42
- Difficult CI anatomy 42
- High-risk surgical associations 42

Advanced implantation and hearing-rehabilitation integration 42
- Device-selection algorithm 42

Advanced skull-base and neuro-otology integration 43
- Case 1 — Unilateral SNHL and tinnitus 43
- Case 2 — Bilateral VS 43
- Case 3 — CPA lesion mimicking VS 43
- Case 4 — Pulsatile tinnitus 44
- Case 5 — Petrous apex lesion 44
- Case 6 — Multiple cranial neuropathies 44
- Case 7 — Lower-CN syndrome 44
- Case 8 — Facial palsy localization 44
- Case 9 — Trauma 44
- Case 10 — Otalgia 44

Master otology and audiology rapid recall 45
- Objective test signatures 45
- Audiogram signatures 45
- Imaging signatures 45
- Vestibular pattern matrix 45
- Vestibular test traps 45
- Tumour triggers 46
- Facial-nerve localization 46
- Otalgia localization 46
- Implant selection 46
- Surgical approach triggers 46
- Emergency first steps 47

Rhinology pp. 48–91

Sinonasal anatomy: embryology, framework and surgical relationships 48
- Embryology and development 48
- External nose: structural framework 48
- Septal blood supply and innervation 48
- Lateral nasal wall: exact drainage map 48
- High-risk variants on CT 49
- Imaging checklist before endoscopic sinus surgery 49

RHINOLOGY (CONTINUED)

Nasal physiology, mucociliary clearance and objective airway assessment 49
- Functions of the nose 49
- Nasal cycle 49
- Mucociliary clearance 49
- Nasal airflow resistance 49
- Objective nasal-airway tests 50
- Outpatient assessment algorithm 50
- Choose imaging according to the question 50

Sinus development, drainage pathways and functional anatomy 50
- Paranasal sinus development 50
- Drainage map — test the exact route 50
- Why the natural maxillary ostium matters 51
- Functional anatomy → clinical pattern 51

Sinonasal CT mapping and surgical danger zones 51
- Danger zones and consequences 51
- Preoperative CT: systematic review 51
- Imaging selection: quick decision table 51
- Image-recognition triggers 51

Outpatient assessment and objective nasal airway measurement 52
- Examination sequence 52
- Objective airway tests 52
- History: classify the problem before ordering tests 52
- Nasal patency: two concepts 52
- Choose the next step 52

Allergic rhinitis: mechanism, classification and diagnosis 52
- Pathophysiology chain 52
- ARIA classification 52
- Diagnostic algorithm 52
- Differentials that commonly overlap 53
- Associated disease 53
- Image / examination pearls 53

Rhinitis treatment, non-allergic rhinitis and occupational disease 53
- Drug and treatment table 53
- Treatment algorithm 53
- Non-allergic rhinitis: recognize the subtype 54
- Occupational rhinitis 54
- Rhinitis medicamentosa 54
- Pathophysiology and treatment matching 54

Acute rhinosinusitis and chronic rhinosinusitis: diagnostic patterns 54
- Definitions and time course 54
- Acute rhinosinusitis: decision algorithm 55
- Viral versus bacterial: high-yield comparison 55
- CRS: diagnosis requires symptoms and objective evidence 55
- CRS with versus without nasal polyps 55
- Red flags that override routine outpatient management 55

Detailed Index

RHINOLOGY (CONTINUED)

Septal deviation, septal haematoma and septal abscess 55

- Septal anatomy: clinical relevance 55
- Septal deviation: presentation and evaluation 55
- Septal haematoma versus septal abscess 55
- Emergency algorithm after nasal trauma 56
- Septoplasty: indications and traps 56

Nasal valve dysfunction, turbinate hypertrophy and septal perforation 56

- Nasal valve: fixed narrowing versus dynamic collapse 56
- Turbinate hypertrophy 56
- Turbinate reduction: principles 56
- Septal perforation 56
- Septal perforation: cause-directed approach 56
- Red flags for destructive septal disease 57
- Rapid pattern recognition 57

Granulomatosis with polyangiitis and ENT vasculitis 57

- GPA: high-yield clinical pattern 57
- Pathophysiology chain 57
- Diagnostic approach 57
- Differential diagnosis of destructive nasal disease 57
- Management principles 57

EGPA, other granulomatous disorders and destructive nasal lesions 58

- GPA versus EGPA 58
- EGPA: pattern recognition 58
- Other granulomatous and destructive conditions 58
- Suspected invasive fungal disease: emergency pathway 58
- Biopsy and investigation traps 59
- Pattern-recognition mini-table 59
- Thirty-second clinical recall card 59

EPOS classification, diagnostic criteria and CRS phenotypes 59

- Definition and diagnostic criteria 59
- Acute rhinosinusitis classification 59
- CRS phenotype classification 59
- Inflammatory endotypes 59
- Systemic and local causes of difficult CRS 60
- Diagnostic traps 60

Nasal polyps: classification, clinical features and differentials 60

- Localized versus diffuse polyposis 60
- Typical inflammatory nasal polyps 60
- Unilateral nasal mass: differential diagnosis 60
- Nasal polyp grading 60
- Histopathology pearls 61
- Associated conditions to screen for 61
- High-yield recall 61

Medical management of CRS and nasal polyposis 61

- Treatment objectives 61
- First-line topical management 61
- Correct intranasal spray technique 61

MEDICAL MANAGEMENT OF CRS AND NASAL POLYPOSIS (CONTINUED)

- Saline irrigation: examination points 61
- Oral corticosteroids 61
- Antibiotics and culture 62
- Other medical options: selected indications 62
- Treatment-failure checklist 62

Endoscopic sinus surgery, recurrence and postoperative care in CRS 62

- Indications for endoscopic sinus surgery (ESS) 62
- Preoperative assessment 62
- Surgical principles 62
- Why polyps recur 62
- Postoperative care 62
- Complications of ESS 62
- Postoperative emergency traps 63

Biologics, difficult CRSwNP and newer treatment strategies 63

- When to consider biologic therapy 63
- Main biologics 63
- Biological mechanism → clinical effect 63
- Choosing biologic therapy versus surgery 63
- Monitoring response to biologics 63
- NSAID-exacerbated respiratory disease (N-ERD/AERD) 63
- Difficult CRS: final decision algorithm 64

Secondary CRS, odontogenic sinusitis and unified airway disease 64

- Odontogenic maxillary sinusitis (ODS) 64
- CRS secondary to mucociliary dysfunction 65
- Immunodeficiency-associated CRS 65
- Unified upper and lower airway disease 65

Orbital complications of acute sinusitis 65

- Spread of infection 65
- Chandler classification 65
- Clinical differentiation 66
- Emergency diagnostic pathway 66
- Management principles 66
- Important differentials 66

Intracranial, osseous and other complications of sinusitis 66

- Intracranial complications 66
- Red-flag clinical features 66
- Intracranial complication: investigation and management 67
- Pott's puffy tumour 67
- Cavernous sinus thrombosis 67
- Other complications 67
- Emergency priority list 67

Fungal rhinosinusitis: classification and non-invasive disease 67

- Allergic fungal rhinosinusitis (AFRS) 68
- AFRS versus fungal ball 68
- AFRS management 68
- Fungal ball management 68
- Fungal disease: image-recognition checklist 68

Detailed Index

Invasive fungal rhinosinusitis and fungal disease MCQs 68

- Acute invasive fungal rhinosinusitis (AIFR) 68
- Diagnostic pathway 69
- Histopathology and organisms 69
- Management of acute invasive fungal disease 69
- Chronic invasive fungal rhinosinusitis 69
- AFRS, fungal ball and invasive disease: final comparison 69
- Fungal rhinosinusitis: high-yield traps 69

Functional endoscopic sinus surgery (FESS/ESS): anatomy and operative principles 70

- Ostiomeatal complex: operative sequence 70
- Preoperative planning 70
- Essential anatomical landmarks 70
- Principles that reduce complications 70
- Common operative errors 70

Frontal recess, frontal sinus disease and surgical approaches 70

- Frontal recess anatomy 70
- Frontal sinusitis: clinical pattern 71
- Frontal sinus mucocele 71
- Frontal sinus surgical approaches 71
- Frontal surgery: complications 71
- Frontal disease red flags 71

ESS complications: recognition and immediate management 71

- Orbital injury and orbital haematoma 71
- Skull-base injury and CSF leak 72
- Major haemorrhage 72
- Other complications 72
- Immediate postoperative red flags 72

Primary ciliary dyskinesia, cystic fibrosis and mucociliary disorders 72

- PCD: diagnostic investigations 72
- Primary ciliary dyskinesia (PCD) 72
- PCD versus cystic fibrosis 73
- Cystic fibrosis 73
- Other causes of impaired mucociliary clearance 73
- Management principles for PCD-related CRS 73
- High-yield pattern recognition 73

Epistaxis 73

- Vascular anatomy 73
- Anterior versus posterior epistaxis 73
- Immediate management algorithm 74
- Unstable patient: do not delay resuscitation 74
- Cautery and packing 74
- Definitive treatment for recurrent or refractory epistaxis 74
- Recurrent epistaxis: investigate the cause 74
- Hereditary haemorrhagic telangiectasia (HHT) 74
- High-yield epistaxis traps 75

Headache and facial pain: sinonasal versus non-sinonasal causes 75

- Headache red flags — emergency assessment 75
- History and clinical assessment 75

- Common causes of facial pain 75
- Sinonasal headache: diagnostic approach 76
- Facial pain pattern-recognition 76

Migraine, cluster headache, neuralgia and “sinus headache” 76

- Migraine 76
- Cluster headache 76
- Trigeminal neuralgia 76
- Tension-type headache 77
- “Sinus headache”: key traps 77
- Quick comparison 77

Contact-point pain, sphenoid sinus disease and dangerous mimics 77

- Sphenoid sinus disease 77
- Contact-point headache 77
- Sphenoid mucocele 77
- Non-sinonasal mimics that must not be missed 78
- Investigation pathway 78

Sinonasal tumours: presentation, imaging and histology 78

- Malignancy warning features 78
- Important tumour types 78
- Benign lesions that mimic malignancy 78
- Histology and radiology clues 79
- Management principles 79

Juvenile nasopharyngeal angiofibroma (JNA) and sinonasal cancer 79

- JNA: classic presentation 79
- Imaging signs 79
- Biopsy warning 79
- JNA management 79
- JNA versus other nasal masses 79
- Tumour-specific exam pearls 79
- Thirty-second clinical recall 79

Snoring and obstructive sleep apnoea (OSA) 80

- Screening and assessment 80
- Sleep testing 80
- Definitions and clinical features 80
- Anatomical sites of obstruction 80
- ENT exam traps 80

Olfactory anatomy, physiology and clinical testing 80

- Olfactory pathway 80
- Causes of olfactory dysfunction 81
- Clinical assessment 81
- Conductive versus sensorineural loss 81

Gustation, taste disorders and smell–taste confusion 81

- Basic taste pathways 81
- Five basic taste qualities 81
- Definitions and causes 81
- Smell versus taste: rapid recall 82

Detailed Index

RHINOLOGY (CONTINUED)

Nasolacrimal disorders and dacryocystitis 82

- Anatomy of tear drainage 82
- Clinical presentation 82
- Acute versus chronic dacryocystitis 82
- Diagnostic approach 82
- Management principles 82

CSF rhinorrhoea: diagnosis, localization and management 83

- CSF leak versus ordinary rhinorrhoea 83
- Confirmation and localization 83
- Emergency red flags 83

Rhinoplasty, nasal cosmesis and functional reconstruction 83

- Preoperative assessment 83
- Structural anatomy relevant to rhinoplasty 83
- Rhinoplasty approaches 84
- Functional grafts and structural support 84
- Common deformities and corrective principles 84

Nasal and facial fractures: assessment and management 84

- Initial trauma priorities 84
- Nasal fracture 84
- Septal haematoma after trauma 85
- Facial fracture patterns 85
- Orbital fracture: emergency signs 85
- Facial swelling: differential diagnosis 85

Facial flaps, scars and keloids 85

- Principles of facial reconstruction 85
- Common local flaps 86
- Nasal reconstruction: defect-based planning 86
- Wound healing and scar maturation 86
- Hypertrophic scar versus keloid 86
- Scar management 86
- Final integrated emergency recall 86

Image bank: rhinology figures and spotters 87

Laryngology, Head and Neck Oncology pp. 92–156

Laryngeal framework and surgical anatomy 92

- Framework 92
- Important spaces 93
- Laryngeal joints and membranes 93

Muscles, innervation, blood supply and lymphatics 93

- Intrinsic muscles 93
- Blood supply 93
- Lymphatic drainage 94

Vocal-fold microanatomy and physiology 94

- Layers of the true vocal fold 94
- Phonation 94
- Airway protection during swallowing 94
- Respiratory role 94

VOCAL-FOLD MICROANATOMY AND PHYSIOLOGY (CONTINUED)

- Voice assessment: what each test answers 94

Dysphonia, reflux-associated symptoms and acute laryngitis 94

- Laryngopharyngeal reflux (LPR) 95
- Acute laryngitis 95

Chronic laryngitis and benign phonotraumatic lesions 95

- Chronic laryngitis 95
- Comparison table 96
- Management pathway for a benign-appearing lesion 97
- Two fundamentally different approaches 97
- Common manoeuvres 97
- Rehabilitation pathway 97

Other non-neoplastic laryngeal disorders 97

- Contact granuloma / vocal-process granuloma 97
- Muscle tension dysphonia (MTD) 97
- Spasmodic dysphonia / laryngeal dystonia 97
- Laryngomalacia 98
- Rapid differentiation 98

Benign laryngeal tumours 98

- Laryngeal haemangioma 99

Malignant laryngeal tumours: presentation and spread 99

- Subsite-based presentation 99
- Histopathology 99
- Routes of local spread 99
- Common mimics 100

Laryngeal cancer: investigations and staging 100

- What each investigation contributes 100
- TNM staging: exam framework 100
- Pretreatment functional assessment 100

Laryngeal cancer: treatment selection and rapid recall 101

- Treatment comparison 101
- Rapid treatment table 101
- Ten high-yield recall pearls 101
- Common exam traps 102
- Thirty-second clinical recall card 102

Laryngeal cancer: treatment decisions and salvage 102

- Early versus advanced disease 102
- Organ-preservation principles 102
- Total laryngectomy — key points 102
- Salvage surgery 102

Vocal-fold palsy: causes and diagnosis 103

- Causes of unilateral palsy 103
- Core classification 103
- Diagnostic algorithm 103
- Investigation traps 103

Detailed Index

Vocal-fold palsy: treatment and framework surgery 103

- Management according to severity 103
- Injection laryngoplasty versus thyroplasty 103
- Isshiki thyroplasty classification 104
- Arytenoid adduction 104
- Bilateral vocal-fold paralysis 104

Non-neoplastic salivary gland disease: anatomy and stones 104

- Major salivary glands 104
- Why submandibular stones are common 104
- Sialolithiasis — diagnosis 104
- Management algorithm 104
- Stone treatment selection 105

Sialadenitis, autoimmune disease and sialoendoscopy 105

- Chronic/recurrent sialadenitis 105
- Acute suppurative sialadenitis 105
- Sjögren syndrome 105
- Sialoendoscopy 105
- Salivary gland differentials 105

Salivary gland tumours: assessment and surgical anatomy 106

- Diagnostic work-up 106
- General clinical principles 106
- Milan System — salivary gland cytopathology 106
- Parotid surgical anatomy 106
- Salivary tumour work-up algorithm 106

Benign salivary gland tumours 106

- Pleomorphic adenoma 106
- Warthin tumour 107
- Other benign tumours 107
- Benign versus malignant clinical clues 107
- Pleomorphic adenoma versus Warthin tumour 107

Malignant salivary tumours and pathology 107

- Important malignant entities 107
- Mucoepidermoid carcinoma 107
- Adenoid cystic carcinoma 107
- Acinic cell and secretory carcinoma 108
- Malignancy work-up and management 108

Parotidectomy, facial nerve and complications 108

- Choosing the operation 108
- Facial nerve assessment 108
- Neck management 108
- Postoperative complications 109
- Frey syndrome — exam pearl 109
- Postoperative red flags 109

Salivary gland rapid recall 109

- One-line clinical patterns 109
- Tumour comparison table 109
- Investigations: what to choose first 110

LARYNGOLOGY, HEAD AND NECK ONCOLOGY (CONTINUED)

Oral cavity anatomy and lymphatic drainage 110

- Oral cavity subsites 110
- Lymphatic drainage 110
- Anatomical features that change management 110
- Examination checklist for a suspicious oral lesion 110

Oral cavity squamous cell carcinoma (SCC) 111

- Biopsy and imaging 111
- Risk factors and presentation 111
- Histopathology: what to request/report 111
- Oral cavity versus oropharyngeal SCC 111

Oral cancer staging, DOI and neck management 111

- Depth of invasion versus tumour thickness 111
- Clinically N0 neck 112
- Clinically positive neck 112
- Neck dissection levels: rapid recall 112

Oral cancer surgery, reconstruction and adjuvant therapy 112

- Surgical principles 112
- Reconstruction by defect 112
- Adjuvant treatment 112
- Margin and pathology traps 112
- Functional outcomes to anticipate 113

Oral potentially malignant disorders (OPMDs) 113

- Definition and recognition 113
- Risk assessment 113
- Dysplasia 113
- Biopsy strategy 113

Leukoplakia, erythroplakia and oral lichen planus 113

- Oral leukoplakia 113
- Erythroplakia 114
- Proliferative verrucous leukoplakia (PVL) 114
- Oral lichen planus versus lichenoid lesions 114
- Actinic cheilitis 114

OPMD: biopsy, treatment and surveillance 114

- Treatment principles 114
- Surveillance traps 114
- Direct-recall facts 114

Oral submucous fibrosis (OSMF): pathogenesis and clinical grading 115

- Aetiopathogenesis 115
- Examination and functional assessment 115
- Clinical grading 115
- Histopathology 115

OSMF: diagnosis and treatment 115

- Diagnostic work-up 115
- Differential diagnosis of trismus 115
- Medical treatment: evidence and traps 116
- Surgical management 116
- OSMF and oral cancer 116

Detailed Index

Oral cancer, OPMD and OSMF: integrated rapid recall 116

- Pattern-recognition table 116
- Pathology and staging — must distinguish 116

Thyroid anatomy and benign thyroid disease 117

- Important surgical relations 117
- Surgical anatomy 117
- Thyroid function tests 117
- Benign thyroid disease: clinical patterns 117

Thyroid nodule: evaluation and ultrasound risk 117

- Ultrasound features suspicious for malignancy 117
- Initial algorithm 117
- ACR TI-RADS — high-yield scoring 117
- Features that override reassurance 118

FNA cytology, Bethesda system and molecular tests 118

- Follicular-patterned lesions — classic trap 118
- Bethesda thyroid cytology 118
- Molecular testing 118
- When FNA is not the whole answer 118

Differentiated thyroid carcinoma: pathology and staging 118

- Main differentiated carcinomas 118
- PTC: characteristic histology 118
- FTC: key distinction 119
- Staging and prognosis 119

Papillary versus follicular thyroid carcinoma 119

- Papillary carcinoma variants 119
- Follicular carcinoma 119
- Treatment principles 119

Medullary, anaplastic and other thyroid malignancies 119

- Medullary thyroid carcinoma (MTC) 119
- Anaplastic thyroid carcinoma 120
- Quick comparison 120

Thyroid surgery: extent, complications and prevention 120

- Choosing the operation 120
- Major complications 120
- Post-thyroidectomy red flags 120
- Radioiodine and TSH suppression 120

Parathyroid disease and hyperparathyroidism 121

- Primary hyperparathyroidism 121
- Calcium/PTH patterns 121
- Localisation studies 121
- Surgical indications 121
- Postoperative hypocalcaemia 121

Multiple endocrine neoplasia (MEN) 121

- MEN comparison 121
- MEN1 — “3 Ps” 121
- MEN2A versus MEN2B 121

- Critical surgical sequence in hereditary MTC 121

Thyroid and parathyroid rapid recall 122

- Essential exam traps 122
- Pattern-recognition list 122

Tracheostomy: indications and anatomy 122

- Tracheostomy versus cricothyrotomy 122
- Indications for tracheostomy 122
- Surgical anatomy 122
- Pre-procedure assessment 122

Tracheostomy: procedure, tubes and immediate complications 123

- Tracheostomy tube types 123
- Open surgical tracheostomy: key steps 123
- Cuff principles 123
- Immediate complications 123

Tracheostomy emergencies 123

- Tube displacement 123
- Bleeding 123
- Tracheo-innominate fistula (TIF) 124

Tracheostomy care, decannulation and late complications 124

- Routine care 124
- First tube change 124
- Decannulation: readiness checklist 124
- Late complications 124
- Speaking valve: essential conditions 124

Laryngeal and tracheal trauma 124

- Mechanisms and red flags 124
- Investigation choices 125
- Laryngeal trauma: treatment principles 125
- Tracheal injury 125

Airway stenosis: causes, assessment and classification 125

- Diagnostic work-up 125
- Myer–Cotton grading of paediatric subglottic stenosis 126

Subglottic and tracheal stenosis: treatment 126

- Treatment selection 126
- Endoscopic treatment 126
- Open surgery 126
- Postoperative principles 126
- GPA-associated subglottic stenosis 126

Deep neck spaces and pathways of spread 126

- Important spaces 126
- Ludwig angina 127
- Spread pathways 127
- Key clinical clues 127

Deep neck space infection: diagnosis and management 127

- Microbiology and antibiotics 127
- When drainage is important 127
- Dangerous complications 127

Detailed Index

LARYNGOLOGY, HEAD AND NECK ONCOLOGY (CONTINUED)

Emergency airway and deep neck infection: pattern recognition 128

Benign neck masses I: congenital and developmental lesions 128

- Thyroglossal duct cyst (TGDC) 128
- Branchial cleft cyst 128
- Dermoid and epidermoid cysts 128
- Cystic hygroma / lymphatic malformation 128
- Rapid differentiation 128

Benign neck masses II: vascular lesions and the adult neck lump 128

- Carotid body tumour (paraganglioma) 128
- Other important benign neck lesions 129
- Adult neck mass: safe diagnostic sequence 129
- Red flags for malignancy 129

Pharyngeal tumours: site-based pattern recognition 129

- Localise before naming the tumour 129

Neurological disease of the pharynx 129

- Localise the neurological deficit 129

Nasopharyngeal carcinoma (NPC) 130

- High-yield associations 130
- Clinical presentation 130
- Diagnostic pathway 130
- Treatment principles 130
- NPC versus other pharyngeal SCC 130

Oropharyngeal tumours: HPV, p16 and treatment selection 130

- Common subsites 130
- HPV-associated oropharyngeal SCC 130
- Diagnostic sequence 131
- Treatment principles 131
- Important pathology distinctions 131

Hypopharyngeal carcinoma 131

- Anatomical subsites 131
- Pathophysiology chain 131
- Work-up 131
- Management principles 131
- Prognostic cues 131

Parapharyngeal space: anatomy and localisation 131

- Compartment anatomy 131
- Clinical presentation 131
- Imaging-based localisation 132
- Management principles 132

Parapharyngeal tumours: differentials 132

- Common tumour patterns 132
- Paraganglioma distinctions 132
- Work-up 132
- Treatment traps 132

LARYNGOLOGY, HEAD AND NECK ONCOLOGY (CONTINUED)

Metastatic neck disease and unknown primary 132

- Nodal level pattern recognition 132
- Adult neck node: investigation algorithm 132
- SCC of unknown primary 133
- Neck dissection: principles 133

Chemotherapy and radiotherapy in head and neck cancer 133

- Treatment selection: quick framework 133
- Key systemic agents 133
- Regimen-level principles 134
- Radiotherapy toxicities 135
- Before, during and after treatment 135
- Must-remember postoperative risk features 135
- Chemotherapy drugs: mechanisms and toxicities 134

Transoral laser microsurgery (TLM) 135

- Indications and selection 135
- Procedure sequence 135
- CO_2 laser: key principles 135

Transoral robotic surgery (TORS) 136

- Patient selection 136
- Complications to anticipate 136
- Platform and components 136
- Indications listed in the source 136
- Advantages versus disadvantages 136

TLM: European Laryngological Society cordectomy classification 136

- ELS classification of endoscopic cordectomy 136
- Extended cordectomy 137
- Operative principles 137
- Voice outcome pattern 137

Rhinoplasty following nasal trauma 137

- Injury pattern → deformity → reconstruction 137
- Acute injury assessment 137

Post-traumatic rhinoplasty: timing and reconstruction 137

- Timing of treatment 137
- Reconstruction by missing structure 138

Post-traumatic deformities: saddle nose, crooked nose and valve failure 138

- Crooked nose 138
- Saddle-nose deformity 138
- Nasal valve injury 138

Pre-operative assessment for rhinoplasty 138

- History and patient selection 138

Facial and nasal analysis 139

- Facial analysis 139
- External nasal examination 139
- Nasal proportions: use carefully 140
- Skin and soft-tissue envelope 140

Detailed Index

Functional assessment and standard photography 140
- Functional assessment 140
- Standard photographic views 140

External rhinoplasty — approach and incisions 140
- Open versus endonasal approach 140
- Incision schematic 141

External rhinoplasty: structural anatomy and tip support 141
- Structural manoeuvres 141
- Lower lateral cartilage anatomy 141

External rhinoplasty: osteotomies and dorsal reconstruction 141
- Osteotomy principles 141
- Middle-vault reconstruction 141

Revision rhinoplasty — diagnosis before reoperation 141
- Complication versus technical error 141
- Common residual deformities 142

Revision rhinoplasty: grafts, donor sites and complications 142
- Graft selection in revision surgery 142
- Donor-site consent 142
- Revision complications 142

Aesthetic dorsal reduction rhinoplasty 142
- Fundamental operative objective 142
- Preoperative analysis 143
- Contraindications to isolated dorsal reduction 143

Dorsal reduction: operative techniques and avoiding complications 143
- Classic versus incremental reduction 143
- Composite dorsal reduction: sequence 143
- Spreader graft versus autospreader flap 143
- Complications and mechanisms 144

Dorsal reduction: special patterns and final checks 144
- True dorsal hump 144
- Pseudohump 144
- Thick-skinned nose 144
- Dorsal reduction checklist 144

Nasal reconstruction: history, anatomy and aesthetic subunits 144
- Historical names 144
- Three structural thirds 145
- Aesthetic subunits 145

Nasal reconstruction: the three-layer principle 145
- Every defect must be analysed in three dimensions 145
- External cover and structural framework 145
- Internal lining 145
- Important principles 145

- Why each layer matters 145
- Defect assessment 145

Local flaps for nasal skin defects 145
- Selection by defect 145
- Bilobed flap 146

Dorsal nasal flap, note flap and nasolabial flap 146
- Dorsal nasal flap 146
- Note flap 146
- Nasolabial / melolabial flap 146

Nasal lining flaps and composite grafts 146
- Septal mucosal flaps 146
- Auricular composite graft 146
- Composite graft harvest schematic 147

Midline and paramedian forehead flaps 147
- Midline forehead flap 147
- Paramedian forehead flap (PMFF) 147

Nasal reconstruction: free flaps, cartilage framework and total reconstruction 148
- Cartilage and bone grafts 148
- Free flaps used in major nasal reconstruction 148
- Total or near-total nasal reconstruction 148

Nasal reconstruction: flap selection, complications and examination traps 148
- Choose the reconstruction by defect 148
- Complications by mechanism 149
- High-yield flap principles 149
- Named historical contributors 149

Image spotters: facial plastics 150

Benign skin lesions: recognition and management 151
- Common lesions 151
- Epidermoid cyst: operative principles 151

Cutaneous malignancy: basal cell and squamous cell carcinoma 151
- Basal cell carcinoma (BCC) 151
- Cutaneous squamous cell carcinoma (cSCC) 151

Melanoma, margin control and facial skin reconstruction 152
- Malignant melanoma: essentials 152
- Mohs micrographic surgery 152
- Reconstruction after facial skin-cancer excision 152

Facial reanimation: anatomy and assessment 152
- Facial nerve anatomy 152
- Facial palsy assessment 153
- Acute versus established paralysis 153

Facial reanimation: nerve repair and nerve transfer 153
- Choose according to nerve integrity and denervation duration 153

Detailed Index

FACIAL REANIMATION: NERVE REPAIR AND NERVE TRANS (CONTINUED)

- Nerve transfers 153

Facial reanimation: muscle transfer and static procedures 154

- Static facial suspension 154
- Free functional muscle transfer 154
- Eye protection procedures 154

Partial and total ear reconstruction: anatomy and planning 154

- Auricular framework 154
- Causes of auricular defects 154
- Reconstruction options 154
- Preoperative assessment 155

Ear reconstruction: named techniques and frameworks 155

- Antia–Buch helical advancement flap 155
- Staged postauricular reconstruction 155
- Autologous rib cartilage framework for microtia 155
- Porous polyethylene framework 155

Temporoparietal fascial flap and prosthetic ear reconstruction 155

- Temporoparietal fascial flap (TPFF) 155

Combined surgical and prosthetic reconstruction 156

- Osseointegrated craniofacial implants 156
- When to combine surgery and prosthesis 156
- Rehabilitation pathway 156

Pediatric ENT pp. 157–190

Congenital inner-ear malformations 157

- Bony labyrinthine malformations 157
- Classification by anatomical site 157
- Vestibular malformations 158
- Membranous labyrinthine malformations 158

Congenital middle-ear abnormalities 158

- Congenital ossicular anomalies 158
- Management principles 159

Microtia, anotia and accessory auricular abnormalities 159

- Microtia and anotia 159
- Preauricular tags and accessory auricles 159
- Preauricular pits and sinuses 159
- Prominent ears 160

Congenital aural atresia and external auditory canal stenosis 160

- Definitions and hearing phenotype 160
- Jahrsdoerfer CT score — 10 points 160
- Management algorithm 160
- Atresiaplasty versus bone-conduction technology 160

PEDIATRIC ENT (CONTINUED)

Syndromic ear malformations and integrated assessment 161

- Syndromic associations 161
- Time-critical syndromic clue 161
- Targeted assessment when a syndrome is suspected 162
- Pattern-based differential 162
- Hearing tests in congenital ear abnormalities 162

Classification, causes and genetic nonsyndromic hearing loss 162

- Classification: define the phenotype first 162
- Genetic hearing loss 162
- GJB2 — connexin 26 163

Syndromic, intrauterine and perinatal causes 163

- Syndromic hearing loss: rapid associations 163
- Intrauterine infections 163
- Other prenatal/perinatal risk factors 163

Disorders causing hearing loss during childhood 164

- Fibrous dysplasia of temporal bone 164
- Acquired and delayed-onset causes 164
- OME: who is predisposed? 164

OAE versus ABR, imaging and rehabilitation 164

- Imaging and targeted systemic work-up in severe/profound SNHL 165
- Rehabilitation: do not wait for the aetiological label 165

Paediatric hearing assessment 165

- Developmental auditory and speech milestones 165
- Newborn hearing screening 165
- Behavioural audiometry by developmental ability 165
- Integrated diagnostic sequence 166

Congenital neck lesions: epidemiology, embryology and site-based diagnosis 166

- Congenital neck mass: rapid differential 166
- Embryological associations 166
- Thyroglossal duct cyst (TGDC) 166
- Dermoid versus epidermoid 166
- Lingual thyroid 167

Branchial cleft anomalies and unusual congenital neck lesions 167

- First branchial cleft anomaly 167
- Second branchial cleft anomaly 167
- Third/fourth pouch sinus 167
- Other congenital sinuses 167
- Syndromic craniosynostosis and surgical patterns 168
- Genetic testing clues 168
- Craniofacial clefts and associated conditions 169

Laryngomalacia: epidemiology, natural history and treatment 169

- Incidence and pathophysiology 169
- Natural history — numerical details 169
- Clinical severity 170
- Diagnostic approach 170
- Named treatments and indications 170

Detailed Index

PEDIATRIC ENT (CONTINUED)

Congenital laryngeal lesions: atresia, webs, cysts and clefts 170

- Laryngeal atresia 170
- Congenital laryngeal web 170
- Vallecular and other laryngeal cysts 171
- Laryngeal cleft 171

Vocal-fold paralysis, subglottic stenosis, RRP and subglottic haemangioma 171

- Paediatric vocal-fold paralysis/immobility 171
- Subglottic stenosis (SGS) 172
- Juvenile-onset recurrent respiratory papillomatosis (RRP) 172
- Congenital subglottic haemangioma 172
- Final rapid-recall table 172

Oesophageal atresia and tracheo-oesophageal fistula (EA/TOF) 173

- Epidemiology and embryology 173
- Classification — Gross/Vogt patterns 173
- Diagnostic algorithm 174
- Clinical presentation and radiographic pattern 173
- Immediate management 173
- H-type TOF — the delayed diagnosis 173
- Pathophysiology chain 174

EA/TOF management and VACTERL association 174

- Preoperative assessment 174
- Waterston risk classification 174
- Operative principles 174

Tracheomalacia, aspiration and GORD 175

- Tracheomalacia 175
- Tracheomalacia versus bronchomalacia 175
- Aspiration in children 175
- GORD and airway symptoms 176

Caustic ingestion and oesophageal injury 176

- Mechanism and agents 176

Airway foreign body; croup versus epiglottitis 176

- Airway foreign-body aspiration 176
- Examination clues 176
- Investigations and imaging pearls 176
- Management algorithm 176
- Delayed diagnosis: complications 177
- Integrated airway anatomy — clinically relevant points 177
- Rapid differentiation: croup versus epiglottitis/supraglottitis 177

Paediatric neck masses 178

- Classic congenital lesions 178

Neck masses: lymph nodes, deep lesions and vascular malformations 178

- Differential by clinical pattern 178
- Vascular malformations — what the exam expects 178

Paediatric airway assessment and management 179

- First decision: is the child unstable? 179
- Stridor by timing and sound 179

PAEDIATRIC AIRWAY ASSESSMENT AND MANAGEMENT (CONTINUED)

- Key physiology and airway traps 179

Paediatric tracheostomy 180

- Operative facts 180
- Paediatric anatomy → surgical implications 180
- Operative technique — named details 180
- Tube selection and care 180
- Decannulation 181

Development and the drooling child 181

- Physiology and developmental facts 181
- Aetiology and associations 181
- Assessment: numbers worth memorising 181
- Treatment facts 181

Tonsils and adenoids 182

- Anatomy and function 182
- Tonsil size: Brodsky grading 182
- Acute tonsillitis: pattern recognition 182
- Complications to remember 182

Tonsillectomy, adenoidectomy and paediatric OSA 182

- Indications for surgery 182
- Adenotonsillectomy pathway for suspected OSA 183
- Surgical complications 183
- Technique recognition 183
- OSA: complications 183

Infantile haemangioma 183

- Biology and natural history 183
- Image recognition 183
- Which lesions need treatment? 184
- Diagnosis and management 184

Vascular malformations: lymphatic, venous, capillary and AV 184

- Lymphatic malformations (LM) 184

Cervicofacial infections 185

- Cervical lymphadenitis 185
- Deep neck-space infections 186
- Lemierre syndrome 186
- Specific infections 186
- Non-infective mimics 187

Choanal atresia and congenital nasal masses 187

- Choanal atresia 187
- Congenital nasal masses — the critical distinction 187

Other congenital lesions and nasal stenosis 188

- Cysts and less common congenital lesions 188
- Pyriform aperture stenosis 188
- Midnasal stenosis 188
- Nasal agenesis / arrhinia 188
- Septal deformity and fibrous dysplasia 188

Nasal masses, neonatal rhinitis and rapid recall 188

- Vascular lesions and tumours 188
- Neonatal rhinitis and acquired obstruction 189

Detailed Index

PEDIATRIC ENT (CONTINUED)

Assessment and peripheral vestibular disorders 189
- Differential diagnosis by hearing status 189
- Common vestibular disorders 189

Hearing-associated disorders, ataxia and management 189
- Vestibular disorders associated with hearing loss 189
- Persistent imbalance and ataxia 190

Miscellaneous pp. 191–205

Electrophysiology and intraoperative monitoring 191
- Facial nerve monitoring 191
- Photodynamic therapy (PDT) 191

Optical coherence tomography (OCT) 191
- Polarization-sensitive OCT (PS-OCT) 192
- Applications and limitations 192

PET–CT: principles and squamous cell carcinoma 192
- Main head and neck cancer uses 193
- Post-chemoradiotherapy assessment 193

PET–CT: recurrence, limitations and emerging applications 193
- Limitations and pitfalls 193
- Other tracers and emerging applications 193

Image-guided surgery and 3D planning 193
- Registration and tracking 194
- ENT applications 194

Interventional neuroradiology and embolization 194
- Embolic agents 194

Laser principles in ENT 195
- Fundamental mechanism 195
- Laser–tissue interaction 196
- ENT applications 196

Contact endoscopy of the upper aerodigestive tract 196
- Principle 196
- What it evaluates 196
- Normal versus pathological patterns 196
- Contact endoscopy versus conventional endoscopy 196
- Combination with narrow-band imaging (NBI) 196

Pharmacology and anaesthetics 197
- Local Anaesthetics 197
- Neuromuscular Blocking Drugs 197
- Autonomic Nervous System 197
- Bronchodilators 198
- Cromoglycate 198
- Antihistamines 198
- General Anaesthetics 198

MISCELLANEOUS (CONTINUED)

Medical statistics 198
- Data Display and Summary 198
- Summary Statistics for Quantitative and Binary Data 198
- Populations and Samples 199
- Statements of Probability and Confidence Intervals 199
- Differences Between Means: Type I and Type II Errors and Power 199
- Confidence Intervals for Summary Statistics of Binary Data 199
- The t Tests 199
- The Chi-Squared Test 199
- Exact Probability Test 200
- Rank Score Tests 200
- Correlation and Regression 200
- Survival Analysis 200
- Statistical Tests in Study Design 200

Clinical governance and audit 200
- Clinical Governance (CG) 200
- Audit 201

Other topics: dimensions, incisions, fMRI, Gamma Knife, gene therapy, HBOT 201
- Dimensions in ENT 201
- Incisions in ENT 202
- Functional MRI (fMRI) 203
- Gamma Knife Stereotactic Radiosurgery 203
- Gene Therapy 204
- Hyperbaric Oxygen Therapy (HBOT) 204

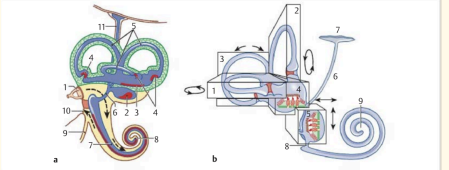


Fig. 1.2a, b
a The inner ear. **1**, Oval window with stapes; **2**, saccule; **3**, utricle; **4**, ampulla of the semicircular canals, with cupula; **5**, membranous semicircular canals (horizontal, superior, and posterior); **6**, ductus reuniens; **7**, cochlear duct; **8**, helicotrema; **9**, the perilymphatic duct, which passes through the cochlear aqueduct; **10**, round window; **11**, endolymphatic sac on the posterior surface of the pyramid.
b The vestibular apparatus. **1**, Lateral semicircular canal; **2**, vertical semicircular canal; **3**, posterior semicircular canal; **4**, utricle; **5**, saccule; **6**, endolymphatic duct; **7**, endolymphatic sac; **8**, ductus reuniens; **9**, cochlea. Arrows mark the direction of movement.

PHYSIOLOGY I

Hearing: Sound → Cochlea → Neural Signal

Core concept

The middle ear solves the **air-to-fluid impedance mismatch**; the cochlea converts mechanical energy into a frequency-specific neural code.

Middle ear as impedance transformer

Air → TM → ossicles → stapes → cochlear fluid

Without middle-ear transformation:
→ major energy loss at air–fluid interface.

Three major mechanisms

1. **TM effective area advantage**
2. **Ossicular lever advantage**
3. **TM/stapes-footplate area ratio**

Source framework:

- Incudomalleolar lever ratio ≈ **1:1.3**
- TM : stapes footplate area ratio ≈ **17:1**
- Overall pressure multiplication ≈ **22-fold**, approximately **25 dB energy gain**.

Eustachian tube contribution

Gas absorption from middle ear
→ pressure falls
→ ET intermittently opens
→ gas replenishment
→ pressure equalization.

ET failure:
→ negative pressure
→ TM retraction
→ middle-ear disease.

Hair-cell transduction

Basilar-membrane movement
→ stereocilia deflection
→ mechanotransduction channels open
→ **K^+ enters from endolymph**
→ depolarization
→ voltage-gated Ca^{2+} entry
→ neurotransmitter release
→ auditory afferent firing.

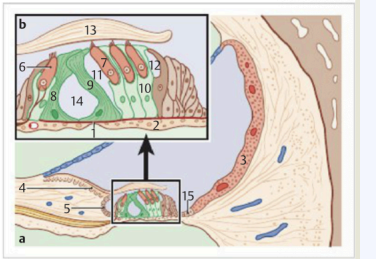


Fig. 1.4a, b The cochlear duct (**a**) and spiral organ of Corti (**b**). The organ of Corti (**b**) rests on the basilar membrane (**1**, **2**) in the cochlear duct. Limbus of the spiral lamina (**4**), internal spiral sulcus (**5**), stria vascularis (**3**). Inner hair cells (**6**) and outer hair cells (**7**) supported by pillar cells (**8**, **9**), perilymph (**11**, **14**), external phalangeal cells of Deiters (**10**), outer tunnel (**12**), external spiral sulcus (**15**), tectorial membrane (**13**), a gelatinous mass extending from the limbus of the spiral lamina (**4**).

Sound terminology

Term	Meaning
Frequency	Cycles/second; Hz
Intensity	Acoustic power/amplitude
dB	Logarithmic ratio
dB SPL	Sound-pressure level
dB HL	Hearing-level scale referenced to normal thresholds
Pitch	Perceptual correlate of frequency
Loudness	Perceptual correlate of intensity

Cochlear travelling wave

Stapes movement at oval window
→ pressure wave in scala vestibuli
→ basilar-membrane travelling wave
→ maximal displacement at frequency-specific cochlear site.

Tonotopy

Base = stiff/narrow → high frequencies

Apex = wider/more compliant → low frequencies

Organ of Corti

Structure	Main function
IHC	Principal sensory receptor
OHC	Cochlear amplifier
Basilar membrane	Frequency-dependent vibration
Tectorial membrane	Mechanical interaction with hair bundles
Stria vascularis	Endolymph + endocochlear potential
Supporting cells	Structural/metabolic support

Treatment (cont.)

Complicated AOM

AOM + postauricular swelling/facial palsy/vertigo/neurological signs
→ complication work-up + appropriate antimicrobial therapy ± surgery.

OME

Definition

Middle-ear effusion without acute infection.

Mechanism

ET dysfunction
→ negative pressure
→ transudation/secretory changes
→ persistent fluid
→ conductive hearing loss.

Clinical

- Often asymptomatic.
- Hearing fluctuation.
- Delayed speech/language development in children.
- Aural fullness.

Tympanometry

Type B + normal canal volume → OME

Management

- Observation when spontaneous resolution likely.
- Address contributing ET/adenoid pathology.
- Ventilation tube when persistent effusion causes clinically important hearing/symptom burden.

Grommet complications

- Otorrhoea.
- Tympanosclerosis.
- Persistent TM perforation.
- Retraction.
- Recurrent effusion after extrusion.

Adult unilateral OME

Unexplained unilateral effusion
→ nasopharyngeal examination
→ exclude obstructive lesion.

TM perforation: hearing-loss determinants

- Size.
- Site.
- Umbo involvement.
- Ossicular status.
- Incudostapedial injury.
- Middle-ear mucosal disease.
- Loss of normal TM/round-window acoustic baffling.
- Altered TM/stapes-footplate area relationship.

Umbo-involving perforation → greater hearing loss than some peripheral perforations.

Recurrent AOM

Source definition:

- Approximately **3 episodes/6 months or ~4/year**.

Options:

- Observation.
- Selected prophylactic strategies.
- Tympanostomy/grommet in appropriate recurrent disease.

OME (cont.)

30-second recall

AOM = bulging TM + acute inflammation. OME = effusion without acute infection + Type B. Adult unilateral OME = nasopharynx.

Exam importance: Must-know

CHRONIC OTITIS MEDIA

Modern classification

Type	Otoscopic finding	Significance
Healed COM	Healed perforation/tympanosclerosis/thinning	Structural sequela
Inactive mucosal	Dry pars-tensa perforation, non-inflamed mucosa	Stable disease
Inactive squamous	Retraction without debris/inflammation	Cholesteatoma precursor
Active mucosal	Perforation + inflamed mucosa + mucopus	Active infection
Active squamous	Retraction + retained squamous debris	Cholesteatoma

Avoid simplistic "safe/unsafe ear" terminology: either type can produce complications.

Pathophysiology

Persistent inflammation
→ inflammatory-cell infiltration
→ increased vascularity/goblet cells
→ granulation
→ osteitis/bone destruction
→ TM/ossicular damage.

Tympanosclerosis

Inflammation/trauma
→ collagen injury
→ fibroblast response
→ excessive collagen
→ hyalinization
→ calcification
→ stiffness.

Terminology

Myringosclerosis
= tympanosclerotic change confined to TM.

Tympanosclerosis
= TM ± middle-ear involvement.

Asymmetric SNHL

Source revision framework:

- approximately **>15 dB difference at 4 kHz**, or
- difference at two consecutive frequencies, especially with unilateral tinnitus/poor speech discrimination → investigate.

Possible causes:

- Vestibular schwannoma.
- Ménière.
- Labyrinthitis.
- Previous meningitis.
- Viral disease.
- Trauma.
- Noise.
- Ototoxicity.
- MS.
- Congenital disease.

Retrocochlear pattern

Unilateral SNHL

- **unilateral tinnitus**
- **speech discrimination disproportionately poor**

→ MRI IAC/CPA.

Emergency algorithm

Sudden hearing complaint
→ otoscopy
→ tuning fork
→ **urgent audiogram**

If SNHL confirmed:
→ initiate appropriate treatment promptly
→ assess red flags
→ arrange IAC/CPA imaging when indicated.

Exclude conductive mimics

- Wax.
- Otitis media.
- TM pathology.
- Ossicular disease.

Investigation

Immediate

Pure-tone audiogram

MRI/IAC particularly important when

- Persistent asymmetry.
- Unilateral tinnitus.
- Neurological signs.
- Retrocochlear concern.
- Recurrent/unexplained episodes.

Targeted tests

Only when history/phenotype suggests:

- ESR.
- Autoimmune testing.
- Syphilis serology.
- Lyme testing where appropriate.
- Other directed investigations.

Speech discrimination trap

PTA severity ≠ functional speech ability.

If speech understanding is much worse than predicted:
→ consider retrocochlear/neural synchrony pathology.

30-second recall

4-kHz notch = noise. High-frequency bilateral = presbyacusis/noise/ototoxicity. Low-frequency = Ménière. Cookie-bite = genetic. Asymmetric + poor speech = retrocochlear.

Exam importance: INI-SS favorite

SUDDEN SENSORINEURAL HEARING LOSS

Definition

Source revision framework:
≥ 30 dB loss across ≥ 3 contiguous frequencies developing within approximately 72 hours.

Clinical pattern

- Usually unilateral.
- Sudden hearing loss.
- Tinnitus.
- Aural fullness.
- Vertigo in some.
- Often idiopathic.

Causes to consider

Category	Examples
Idiopathic	Commonest practical category
Trauma	Temporal-bone injury
Barotrauma	Diving/blast
Infection	Mumps, measles, VZV, syphilis, labyrinthitis/meningitis
Tumour	VS
Vascular	Labyrinthine/central vascular event
Neurological	MS
Iatrogenic	Ear surgery
Drugs	Ototoxic agents

Prognosis

Better	Worse
Mild initial loss	Severe/profound
No vertigo	Severe vertigo
Younger	>60 years
Early treatment	Delayed treatment
Lower inflammatory markers	Raised ESR

Source revision material states approximately **60% of idiopathic cases may recover spontaneously**.

Core electroacoustic terms

Term	Meaning
Gain	Output − input
Frequency response	Gain/output across frequency
SSPL	Maximum/saturation output capability
OSPL90	Output measured with 90 dB SPL input
Compression	Reduces amplification of louder inputs
Compression ratio	Input-level change relative to output-level change
Compression threshold	Input level at which compression begins
WDRC	Wide dynamic-range compression
Feedback	Output leaks back into microphone → oscillation/whistle

Scott Brown describes **linear** amplification as relatively constant gain for a given frequency, whereas **non-linear** systems vary gain with input level; SSPL is commonly characterized using OSPL90.

Directional microphones

- Improve signal-to-noise performance when speech is preferentially from the front.
- Not equivalent to noise cancellation.

Binaural fitting

Benefits:

- improved speech identification;
- localization;
- reduced head-shadow effect;
- more balanced auditory input;
- potentially lower gain requirement.

Verification

Prescription → physical/electroacoustic verification → real-ear measurement → patient outcome → fine tuning

Exam traps

- Hearing aid **amplifies sound; CI electrically stimulates cochlear neural elements**.
- Compression does not simply "reduce all volume"; it alters gain according to input level.
- Open fitting is not synonymous with mild hearing loss.
- CROS routes information from the deaf side; it does not restore binaural cochlear input.

30-second card

High-frequency SNHL + preserved lows → open fit.
Recruitment → compression.
Feedback → leakage/output returning to microphone.
CROS → unilateral deaf ear + normal better ear.
BiCROS → deaf ear + hearing-impaired better ear.

Why compression matters

Cochlear loss → recruitment → reduced dynamic range
→ high gain needed for soft sounds
→ excessive loudness for loud sounds
→ **compression becomes essential**

Hearing-aid styles

Device	Typical niche
BTE	Broadest range; severe/profound loss
Open-fit BTE	High-frequency SNHL + good low frequencies
RITE/RIC	Receiver positioned in ear canal
ITE	Concha
ITC	Canal
CIC	Deep canal/cosmetic
Bone-conduction	Conductive/mixed/atresia/selected SSD
CROS	Unilateral deafness + normal better ear
BiCROS	Deaf/very poor ear + impaired better ear

Open-fit principle

Best when:

- low frequencies relatively preserved;
- high-frequency SNHL dominates;
- occlusion is undesirable.

Trap: open fit can increase feedback and is unsuitable for very high gain requirements.

Venting

Vent → less occlusion + more natural low-frequency sound
but
vent → acoustic leakage → reduced low-frequency gain + greater feedback risk.

BONE-CONDUCTION + MIDDLE-EAR IMPLANTS

Bone-conduction principle

Skull vibration → cochlear stimulation → bypass EAC + middle ear

Bone conduction stimulates **both cochleae**.

Indications

Situation	Why BC helps
Aural atresia	No usable EAC
Chronic discharging ear	Avoids canal occlusion
Large conductive loss	Bypasses external/middle-ear component
Mixed loss	Useful if cochlear reserve adequate
SSD	Routes sound to functioning cochlea
Poor mould tolerance	Alternative coupling

Step 3 — Confirm sensitization if it will change management: skin-prick testing (SPT) or serum allergen-specific IgE.

Step 4 — Interpret in context: a positive allergy test indicates sensitization; it does not prove that the allergen explains the patient's symptoms.

6. Image / examination pearls

- Pale or edematous turbinates may occur in allergic rhinitis but are not diagnostic by themselves.
- Nasal endoscopy is useful for identifying coexisting polyps, structural obstruction or chronic sinonasal disease.
- Skin-prick test: wheal response must be interpreted against positive and negative controls and the clinical history.

Rapid recall: Symptoms suggest the diagnosis; relevant sensitization testing supports the allergic mechanism; neither should override an atypical clinical presentation.

RHINITIS TREATMENT, NON-ALLERGIC RHINITIS AND OCCUPATIONAL DISEASE

1. Drug and treatment table

Intervention	Main role / mechanism	Exam trap
Intranasal corticosteroid	Suppresses mucosal inflammation; effective for congestion and broader nasal symptoms	Correct technique and regular use matter
Second-generation H1 antihistamine	Blocks histamine-mediated itching, sneezing and rhinorrhoea	Usually less effective for marked obstruction than intranasal corticosteroids
Intranasal antihistamine	Local antihistaminic effect; can help allergic and some non-allergic symptoms	Not interchangeable with every systemic antihistamine
Intranasal ipratropium	Anticholinergic; reduces glandular watery secretion	Helps rhinorrhoea much more than congestion or itching
Intranasal cromone	Mast-cell stabilizing effect	Generally less effective than intranasal corticosteroids; repeated dosing may be required
Leukotriene receptor antagonist	Blocks leukotriene-mediated effects	Usually less effective for rhinitis than intranasal corticosteroids; consider overall asthma context
Topical decongestant	Vasoconstriction rapidly reduces congestion	Prolonged use risks rhinitis medicamentosa; restrict to a short course

4. Differentials that commonly overlap

Feature	Allergic rhinitis	Non-allergic rhinitis	Rhinosinusitis
Itching / paroxysmal sneezing	Common	Variable	Not usually dominant
Watery rhinorrhoea	Common	Common	May occur, but purulent discharge may predominate in acute bacterial disease
Trigger	Specific allergens	Irritants, temperature, humidity, other non-allergic triggers	Infection or chronic sinonasal inflammation
Allergy testing	May demonstrate relevant sensitization	No relevant sensitization expected in pure non-allergic disease	Not diagnostic
Reduced smell	Possible	Possible	Important symptom, especially in chronic disease or polyposis

5. Associated disease

- Allergic rhinitis and asthma frequently coexist: assess the lower airway when there is wheeze, cough or breathlessness.
- Consider conjunctivitis when ocular itching and watering accompany nasal symptoms.
- Persistent unilateral symptoms, a visible mass, significant bleeding or progressive deformity are atypical for uncomplicated allergic rhinitis.

Intervention	Main role / mechanism	Exam trap
Allergen-specific immunotherapy	Modifies allergen-specific immune response	Requires appropriate allergen selection and safety assessment; systemic reactions can occur
Saline irrigation	Mechanical clearance and symptom support	Adjunctive, not a substitute for indicated anti-inflammatory therapy

2. Treatment algorithm

Confirmed or strongly suspected allergic rhinitis

Mild/intermittent symptoms: allergen/irritant measures where useful + appropriate antihistamine or intranasal therapy.

Persistent or moderate/severe symptoms: intranasal corticosteroid; assess technique, adherence and ongoing exposure.

Inadequate response: verify diagnosis, spray technique, regular use and coexisting structural disease; consider combination intranasal therapy or specialist review.

Selected persistent allergen-driven disease: consider allergen-specific immunotherapy after appropriate assessment.

Safety trap: Systemic corticosteroids should not become routine long-term rhinitis therapy. Topical decongestants are not maintenance treatment.

Complication	Clinical concern
Persistent smell loss	May reflect disease severity, olfactory mucosal damage or postoperative factors

7. Postoperative emergency traps

- Reduced vision, proptosis or tense orbit after ESS: treat as an orbital emergency; urgent assessment and decompression may be required.
- Persistent clear unilateral rhinorrhoea after surgery: assess for CSF leak.
- New severe headache, neurological deficit or meningism: urgent evaluation for intracranial complication.
- Do not delay emergency treatment while waiting for routine outpatient review.

2. Main biologics

Drug	Target	Clinical role / exam point
Dupilumab	IL-4 receptor alpha; inhibits IL-4 and IL-13 signalling	Improves polyp burden, obstruction and smell in selected severe CRSwNP; may also benefit coexisting type 2 asthma
Omalizumab	IgE	Useful in selected severe CRSwNP, especially where IgE-mediated disease/asthma also influences treatment choice
Mepolizumab	IL-5	Reduces eosinophilic inflammation; may benefit severe CRSwNP and associated eosinophilic asthma

- Eligibility, age limits, dosing and funding/approval criteria depend on the applicable jurisdiction and current product guidance.
- Other biologic pathways may be under study or used for related indications; do not assume every asthma biologic is established for CRSwNP.

3. Biological mechanism → clinical effect

- IL-4/IL-13 blockade → reduced type 2 inflammatory signalling → reduced polyp burden and improved obstruction/smell.
- IgE blockade → reduced free IgE and downstream allergic inflammatory activation.
- IL-5 blockade → reduced eosinophil maturation/survival → reduced eosinophilic inflammation.

6. NSAID-exacerbated respiratory disease (N-ERD/AERD)

Classic triad

- Asthma.
- Chronic rhinosinusitis with nasal polyps.
- Respiratory reactions to aspirin or other COX-1-inhibiting NSAIDs.

Mechanism

- COX-1 inhibition → reduced protective prostaglandin E_2 signalling and altered arachidonic-acid metabolism → increased cysteinyl leukotriene activity → bronchoconstriction and sinonasal inflammation.

Clinical features

BIOLOGICS, DIFFICULT CRSWNP AND NEWER TREATMENT STRATEGIES

1. When to consider biologic therapy

Biologics are considered for selected patients with severe, uncontrolled CRSwNP, particularly when type 2 inflammation is prominent and disease remains burdensome despite appropriate standard treatment.

Common features supporting specialist assessment:

- Bilateral polyps with persistent symptoms.
- Previous ESS with recurrence, or surgery unsuitable/not desired after informed discussion.
- Need for repeated systemic corticosteroid courses or corticosteroid contraindication.
- Significant smell loss and impaired quality of life.
- Coexisting asthma or other type 2 inflammatory disease.
- Objective evidence of ongoing disease on endoscopy/CT.

4. Choosing biologic therapy versus surgery

Clinical situation	Consideration
Significant obstructive disease with accessible surgical targets and no prior adequate ESS	ESS may be appropriate after assessment of medical therapy and patient goals
Recurrent severe polyps after ESS despite appropriate topical treatment	Assess inflammatory endotype and biologic eligibility
Severe CRSwNP plus difficult asthma/type 2 disease	A biologic that addresses both disease domains may be advantageous
Predominantly anatomical obstruction without diffuse inflammatory disease	Structural treatment may be more relevant than a biologic
Unilateral mass or atypical lesion	Establish diagnosis; do not treat as routine bilateral inflammatory polyposis
Active invasive infection or suspected malignancy	Investigate and manage the specific condition rather than reflexively escalating biologic therapy

5. Monitoring response to biologics

Document before treatment and during follow-up:

- Endoscopic polyp score.
- Nasal obstruction and discharge.
- Smell function.
- SNOT-22 or another validated quality-of-life score.
- Need for systemic corticosteroids.
- Need for surgery.
- Asthma control and other relevant comorbid outcomes.
- Adverse effects and treatment adherence.

Continue, switch or discontinue treatment according to objective response, adverse effects, patient goals and current eligibility guidance.

FUNCTIONAL ENDOSCOPIC SINUS SURGERY (FESS/ESS): ANATOMY AND OPERATIVE PRINCIPLES

1. Preoperative planning

- Review thin-section CT in axial, coronal and sagittal planes.
- Identify:
  - Uncinate process and natural maxillary ostium.
  - Ethmoid bulla and basal lamella.
  - Frontal recess and frontal cells.
  - Skull-base height and asymmetry.
  - Lamina papyracea and orbital defects.
  - Anterior ethmoidal artery.
  - Onodi cell and optic nerve relationship.
  - Sphenoid septum attachment to carotid prominence.
- Record previous surgery, altered anatomy, skull-base defects and disease extent.
- MRI is added when a mass, orbital/intracranial extension, perineural spread or selected soft-tissue lesion requires characterization.
- Image guidance is particularly useful in revision surgery, extensive polyposis, distorted anatomy, frontal/sphenoid disease and selected skull-base procedures.

5. Common operative errors

- Mistaking an accessory maxillary ostium for the natural ostium.
- Incomplete frontal recess clearance with postoperative stenosis.
- Excessive mucosal trauma leading to adhesions.
- Injury to the skull base during superior ethmoid dissection.
- Orbital entry through a dehiscent/thin lamina papyracea.
- Carotid injury from sphenoid instrumentation or septal manipulation.
- Operating on a presumed polyp without excluding a vascular lesion or encephalocele when features are atypical.

FRONTAL RECESS, FRONTAL SINUS DISEASE AND SURGICAL APPROACHES

1. Frontal recess anatomy

- The frontal sinus drains through the frontal sinus ostium into the frontal recess pathway.
- The pathway is influenced by the uncinate process, agger nasi cell, ethmoid bulla and frontal cells.
- Agger nasi cell: anterior ethmoid cell frequently relevant to frontal recess access.
- Frontal cells can narrow or redirect the drainage corridor; their relationship to the frontal ostium is more important than the name alone.
- The frontal recess has substantial anatomical variability; review sagittal and coronal CT before surgery.

2. Ostiomeatal complex: operative sequence

- Endoscopic orientation: identify inferior turbinate, middle turbinate and middle meatus.
- Uncinectomy: expose the ethmoidal infundibulum and natural maxillary ostium.
- Maxillary antrostomy: identify and enlarge the natural ostium, joining an accessory ostium when present to avoid mucus recirculation.
- Anterior ethmoidectomy: identify and remove obstructive ethmoid partitions while maintaining orientation to the lamina papyracea and skull base.
- Basal lamella: separates anterior and posterior ethmoid compartments; crossing it enters the posterior ethmoid region.
- Posterior ethmoidectomy: proceed only with clear anatomical orientation and knowledge of the optic nerve/Onodi cell relationship.
- Sphenoidotomy: identify the natural sphenoid ostium and nearby optic nerve, carotid artery and skull-base structures.
- Frontal sinus surgery: requires specific frontal recess mapping; do not assume the frontal drainage pathway is a single straight channel.

3. Essential anatomical landmarks

Landmark	Surgical significance
Uncinate process	Key structure for access to the infundibulum and maxillary drainage pathway
Ethmoid bulla	Prominent anterior ethmoid cell; landmark in middle-meatal surgery
Basal lamella of middle turbinate	Boundary between anterior and posterior ethmoid compartments
Lamina papyracea	Thin medial orbital wall; injury may cause orbital fat prolapse or muscle injury
Ethmoid roof / lateral lamella	Skull-base injury can cause CSF leak or intracranial complications

Landmark	Surgical significance
Anterior ethmoidal artery	Injury may cause major bleeding and orbital haematoma
Onodi cell	May closely surround the optic nerve; high-risk posterior ethmoid variant
Sphenoid septum	May attach to the internal carotid artery prominence

4. Principles that reduce complications

- Keep the endoscope and instruments oriented to known landmarks.
- Avoid blind instrumentation or forceful advancement into an unidentified cavity.
- Maintain awareness of the direction of the skull base and lamina papyracea.
- Preserve healthy mucosa wherever feasible.
- Use navigation as an adjunct—not as a substitute for anatomical knowledge.
- Intraoperative bleeding that obscures landmarks should prompt restoration of visualization before proceeding.

Term	Meaning
Presbyosmia	Age-associated decline in olfactory function

Term	Meaning
Specific anosmia	Selective inability to perceive particular odorants

4. Clinical assessment

- Establish onset, duration, fluctuation and trigger.
- Ask about recent viral illness, head injury, nasal surgery, polyps, neurological symptoms, medication and toxic exposure.
- Assess nasal obstruction, discharge and smell-specific complaints.
- Perform nasal endoscopy to inspect the olfactory cleft, polyps, oedema and masses.
- Use validated psychophysical tests where available:
  - UPSIT: University of Pennsylvania Smell Identification Test.
  - Sniffin' Sticks: threshold, discrimination and identification testing.
- CT PNS is useful when inflammatory sinonasal disease or a structural obstruction is suspected.
- MRI is indicated when a central lesion, olfactory groove tumour, congenital abnormality or selected neurological cause is suspected.

5. Conductive versus sensorineural loss

Feature	Conductive	Sensorineural/neural
Typical cause	Polyps, oedema, obstruction	Viral injury, trauma, neural disease
Endoscopy	May reveal obstructive disease	May be normal
Response to treatment	May improve when inflammation/obstruction resolves	Variable; depends on cause and duration
Imaging	CT for sinonasal disease	MRI for selected neural/intracranial causes

3. Definitions and causes

Term	Meaning
Ageusia	Complete loss of taste
Hypogeusia	Reduced taste
Dysgeusia	Distorted or unpleasant taste perception
Phantom taste	Taste perception without a corresponding stimulus

Causes

- Post-viral illness.
- Medication effects.
- Zinc deficiency in selected cases.
- Oral candidiasis, xerostomia and oral/dental disease.
- Head injury or facial nerve injury.
- Radiation or surgery involving taste pathways.
- Neurological disease.
- Smoking and chemical exposure.

3. Causes of olfactory dysfunction

Conductive

- Allergic rhinitis or marked mucosal oedema.
- CRSwNP.
- Nasal obstruction or a mass preventing odour access to the olfactory cleft.

Sensorineural/neural

- Post-viral olfactory dysfunction, including after SARS-CoV-2 infection.
- Head trauma with olfactory nerve injury.
- Neurodegenerative disease, including Parkinson's disease and Alzheimer's disease.
- Congenital olfactory dysfunction.
- Selected medication, toxic or occupational exposures.
- Intracranial or olfactory groove lesions in selected presentations.

6. Management

- Treat underlying CRS, polyps or rhinitis when present.
- Consider topical corticosteroids for relevant inflammatory disease; delivery technique and sinus access matter.
- Olfactory training: repeated, structured exposure to several distinct odours over weeks to months; useful in many patients with post-viral dysfunction.
- Counsel on smoke/gas alarms, food spoilage and other safety consequences of anosmia.
- Persistent unexplained dysfunction, unilateral symptoms, neurological signs or a mass warrants further investigation.
- Avoid promising recovery: prognosis depends on cause, severity, age, duration and residual olfactory function.

GUSTATION, TASTE DISORDERS AND SMELL–TASTE CONFUSION

1. Basic taste pathways

- Anterior two-thirds of tongue: taste via chorda tympani, a branch of the facial nerve (VII).
- Posterior one-third: taste via glossopharyngeal nerve (IX).
- Epiglottis/lower pharyngeal region: taste via vagal pathways (X).
- General sensation from the anterior two-thirds of the tongue is primarily carried by the lingual nerve, a branch of V3.
- Flavour perception combines taste, retronasal olfaction, texture, temperature and trigeminal chemesthesis.

2. Five basic taste qualities

- Sweet.
- Salty.
- Sour.
- Bitter.
- Umami.

Exam trap: Most patients who report "loss of taste" have reduced flavour perception caused by olfactory dysfunction rather than complete loss of gustation.

LARYNGEAL FRAMEWORK AND SURGICAL ANATOMY

1. Framework

- Unpaired cartilages: thyroid, cricoid, epiglottis.
- Paired cartilages: arytenoid, corniculate, cuneiform.
- Only complete cartilaginous ring of the airway: cricoid.
- Thyroid cartilage: two laminae meet anteriorly; prominence is the laryngeal prominence.
- Epiglottis: elastic cartilage; directs the bolus away from the laryngeal inlet during swallowing.
- Arytenoids: sit on the posterior cricoid lamina; vocal processes anchor the vocal ligaments; muscular processes provide leverage for intrinsic muscles.

Tracheobronchial anatomy

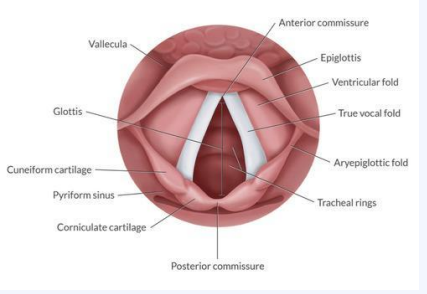
- Trachea extends from the cricoid region to the carina, approximately at T4–T5 depending on respiration and posture.
- The right main bronchus is more vertical than the left; aspirated foreign bodies therefore enter the right bronchial tree more often.
- The tracheal wall has C-shaped cartilage rings with a posterior membranous wall.
- The trachealis muscle alters airway calibre and participates in cough mechanics.
- Mucociliary clearance moves secretions towards the pharynx.

Three major functions Larynx

1. Airway protection: laryngeal closure during swallowing and reflex responses to penetration/aspiration.
2. Respiration: vocal-fold abduction permits airflow; laryngeal muscles coordinate with breathing.
3. Phonation: controlled vocal-fold vibration converts airflow into sound.

Supraglottis

- Epiglottis, aryepiglottic folds, arytenoids, false vocal folds and laryngeal ventricles.
- Rich lymphatic drainage → comparatively early cervical nodal spread.
- Symptoms may include dysphagia, odynophagia, referred otalgia or a neck node.



1. Framework (cont.)

Factors affecting pitch

- Greater vocal-fold tension and effective longitudinal stiffness → generally higher fundamental frequency.
- Greater vibrating mass → generally lower fundamental frequency.
- Vocal-fold length, tissue elasticity and subglottic pressure also influence pitch.

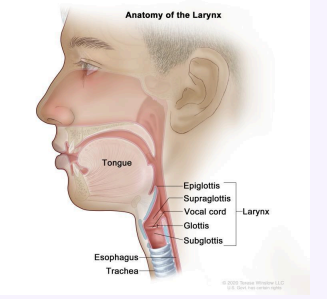
Factors affecting intensity

- Increased subglottic pressure generally increases vocal intensity.
- Vocal-fold adduction and glottic configuration influence the efficiency of converting airflow into acoustic energy.
- Excessive laryngeal constriction may produce strain rather than efficient projection.

High-yield distinctions

Parameter	Meaning
Fundamental frequency (F0)	Perceived pitch correlate
Intensity	Acoustic energy, commonly expressed in decibels
Jitter	Cycle-to-cycle variation in frequency
Shimmer	Cycle-to-cycle variation in amplitude
Harmonics-to-noise ratio	Relative periodic harmonic energy versus noise

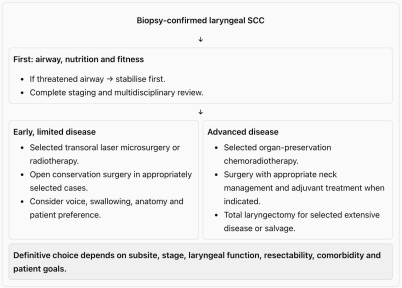
2. Laryngeal subsites



Glottis

- True vocal folds and anterior/posterior commissures.
- Relatively sparse lymphatics → early glottic lesions have a low nodal-metastasis risk.
- Early hallmark: persistent hoarseness.

LARYNGEAL CANCER: TREATMENT SELECTION AND RAPID RECALL



2. Treatment comparison (cont.)

Treatment-selection traps

- Anatomical preservation is not the same as functional preservation.
- Chemoradiotherapy is not automatically preferable to surgery for every advanced tumour.
- Total laryngectomy separates the respiratory and digestive tracts; a permanent tracheostoma is required.
- After total laryngectomy, speech rehabilitation may use tracheoesophageal voice, an electrolarynx or oesophageal speech.
- Post-treatment surveillance must assess recurrence, airway, swallowing, nutrition and functional outcomes.

4. Ten high-yield recall pearls

1. Only intrinsic vocal-fold abductor: posterior cricoarytenoid.
2. Cricothyroid motor supply: external branch of superior laryngeal nerve.
3. Sensory supply above the folds: internal branch of superior laryngeal nerve.
4. Motor supply to other intrinsic muscles: recurrent laryngeal nerve.
5. Reinke space: superficial lamina propria.
6. Early glottic cancer: hoarseness often prompts early detection; nodal risk is relatively low.
7. Supraglottic cancer: richer lymphatic drainage and greater nodal risk.
8. Most common benign laryngeal tumour in the source material: recurrent respiratory papillomatosis.
9. RRP association: HPV types 6 and 11.
10. Persistent suspicious laryngeal lesion: visualise and obtain tissue diagnosis as appropriate; do not rely on symptoms or imaging alone.

2. Treatment comparison

Treatment	Typical role	Major consideration
Transoral laser microsurgery (TLM)	Selected early lesions; selected cases in experienced centres	Margin assessment, exposure, voice outcome and expertise
Radiotherapy	Selected early glottic/supraglottic disease; selected organ-preservation pathways	Late oedema, swallowing/voice effects and salvage options
Concurrent chemoradiotherapy	Selected locally advanced disease when organ preservation is appropriate	Toxicity and the need for a functional larynx
Partial/conservation laryngectomy	Selected tumours with adequate residual function and resectability	Aspiration, rehabilitation and selection criteria
Total laryngectomy	Extensive disease, nonfunctional larynx, selected salvage cases	Permanent neck stoma and voice rehabilitation
Salvage surgery	Persistent/recurrent disease after nonsurgical treatment in suitable patients	Higher complication risk, including fistula

3. Rapid treatment table

Intervention	Mechanism / principle	Key use	Exam trap
Transoral laser microsurgery	Precise endoscopic tumour excision	Selected resectable lesions	Requires adequate exposure and careful margin assessment
Radiotherapy	**DNA damage in tumour cells**	Early disease or selected organ-preservation treatment	Late tissue effects can impair function
Cisplatin-based concurrent chemoradiotherapy	Radiosensitisation plus systemic cytotoxic effect	Selected locally advanced cancers	Renal, auditory, neurological and other toxicities may limit use
Total laryngectomy	Complete removal of larynx	Selected extensive or salvage disease	Permanent neck stoma; requires voice rehabilitation
Voice therapy	Behavioural modification and improved phonatory efficiency	Functional dysphonia and rehabilitation	Does not replace biopsy of suspicious lesions

Regimens and eligibility must be individualised; the table is not a prescribing protocol.

3. Adenoid cystic carcinoma (cont.)

Trap: absence of cervical nodal metastasis does not exclude clinically significant adenoid cystic carcinoma; perineural and distant spread are important.

5. Malignancy work-up and management

- Obtain histological/cytological diagnosis and stage the primary and neck.
- MRI is valuable when perineural spread or deep soft-tissue extent is suspected.
- CT helps assess bone and selected complex anatomy.
- Surgery is the main treatment for many resectable salivary malignancies.
- Neck dissection is guided by clinical nodal disease, tumour site, grade, stage and occult nodal risk.
- Postoperative radiotherapy is considered for adverse features such as high-grade histology, advanced stage, positive/close margins where relevant, perineural invasion, lymphovascular invasion or nodal disease.
- Systemic treatment is selected for specific advanced/recurrent settings; targeted therapy depends on actionable alterations and clinical context.

2. Facial nerve assessment

Document preoperatively:

- Forehead wrinkling and eyebrow elevation.
- Eye closure.
- Smile and upper-lip movement.
- Lower facial movement.
- Any pre-existing weakness, asymmetry or synkinesis.

Intraoperative nerve monitoring may help identify and assess nerve function, but does not replace anatomical knowledge and careful dissection.

3. Neck management

- Clinically involved nodes → therapeutic neck management based on site and extent.
- Clinically N0 neck → consider occult nodal risk according to primary site, grade and stage.
- High-grade and advanced salivary malignancies may justify elective neck treatment.
- Radiotherapy decisions depend on pathological risk factors and multidisciplinary review.

4. Acinic cell and secretory carcinoma

- Acinic cell carcinoma shows serous **acinar differentiation**; it often arises in the parotid.
- Secretory carcinoma can resemble other low-grade salivary neoplasms morphologically.
- Molecular testing may distinguish secretory carcinoma, especially when diagnosis is uncertain.
- Do not equate a low-grade appearance with zero recurrence or metastatic risk.

PAROTIDECTOMY, FACIAL NERVE AND COMPLICATIONS

1. Choosing the operation

Procedure	Typical role
Extracapsular dissection	Selected small, superficial, well-circumscribed benign tumours in appropriately selected patients and experienced hands
Partial superficial parotidectomy	Selected superficial lesions with limited gland resection
Superficial parotidectomy	Tumours in the superficial surgical lobe requiring formal nerve identification and gland removal
Total conservative parotidectomy	Selected deep-lobe, multifocal or extensive disease when the facial nerve can be preserved
Total parotidectomy with nerve sacrifice	Selected malignancy with direct nerve involvement when required for oncological clearance
Radical parotidectomy	Extensive malignancy requiring removal of the facial nerve and/or adjacent structures as part of the oncological resection

Principle: preserve the facial nerve when oncologically appropriate. Preoperative weakness, fixation or imaging suggesting invasion requires careful assessment; sacrifice is not automatic solely because a tumour is malignant.

2. Anaplastic thyroid carcinoma

- Highly aggressive, undifferentiated malignancy.
- Often presents with rapidly enlarging neck mass, dysphagia, dyspnoea, hoarseness or invasion of adjacent structures.
- Requires urgent airway assessment and rapid tissue diagnosis.
- Contrast CT/MRI assesses invasion and resectability; core biopsy may be needed.
- Treatment depends on resectability, molecular findings, performance status and goals of care.
- Test for actionable alterations, particularly BRAF V600E, because targeted treatment may be relevant.
- Surgery is appropriate only when meaningful oncological resection is feasible; otherwise multimodal or palliative approaches may be needed.

THYROID SURGERY: EXTENT, COMPLICATIONS AND PREVENTION

1. Choosing the operation

Procedure	Typical indication
Hemithyroidectomy/lobectomy	Selected unilateral indeterminate nodules, benign symptomatic unilateral disease, and selected low-risk cancers
Total thyroidectomy	Selected bilateral disease, higher-risk malignancy or when required for planned adjuvant management
Therapeutic central compartment dissection	Clinically involved or otherwise appropriately selected central nodal disease
Therapeutic lateral neck dissection	Confirmed/suspicious lateral nodal disease according to staging and surgical plan
Completion thyroidectomy	Selected patients after lobectomy when final pathology indicates benefit from further surgery

3. Post-thyroidectomy red flags

- Rapidly expanding neck swelling.
- Stridor, dyspnoea or difficulty handling secretions.
- New voice change or weak cough.
- Perioral tingling, paraesthesia, cramps or tetany.
- Wound drainage suggesting chyle or significant bleeding.

Emergency trap: a rapidly expanding postoperative neck haematoma can obstruct the airway. Do not wait for imaging when airway compromise is developing.

3. Quick comparison

Feature	PTC/FTC	MTC	Anaplastic carcinoma
Cell of origin	Follicular cells	C cells	Undifferentiated malignant cells
Marker	Thyroglobulin often useful	Calcitonin, CEA	No single universal specific marker
Radioiodine	Selected cases	Not effective	Not effective
Hereditary clue	Selected familial syndromes	RET/MEN2	Some tumours arise in background of differentiated disease
Urgency	Risk-dependent	Specialist staging and RET evaluation	Urgent airway and oncological assessment

Trap: a normal calcitonin result does not replace appropriate clinical/pathological assessment when suspicion remains high.

2. Major complications

Complication	Mechanism / presentation	Prevention or response
Neck haematoma	Expanding swelling, pressure, stridor	Emergency recognition and airway/wound management; do not delay for imaging
RLN injury	Hoarseness, vocal-fold immobility	Identify and preserve nerve where possible; assess postoperative voice
Bilateral RLN injury	Critical airway obstruction	Urgent airway management
EBSLN injury	Loss of pitch/projection	Careful superior-pole dissection
Hypocalcaemia	Parathyroid injury/devascularisation	Preserve parathyroids and blood supply; monitor calcium/PTH as indicated
Hypothyroidism	Loss of functioning thyroid tissue	Thyroid hormone replacement as needed
Chyle leak	Thoracic duct/lymphatic injury, especially left lower neck	Identify and manage leak; nutritional and procedural measures as indicated

4. Radioiodine and TSH suppression

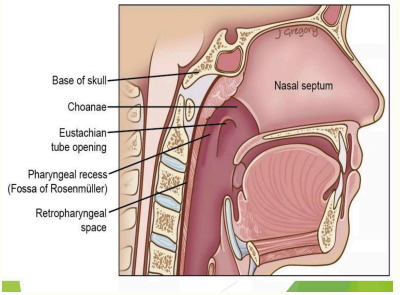
- Radioactive iodine is used selectively in differentiated thyroid carcinoma according to recurrence risk, residual disease and iodine avidity.
- TSH suppression with levothyroxine is individualised.
- Excessive suppression can cause atrial fibrillation and bone loss, particularly in older adults and postmenopausal patients.
- Follow-up may include thyroglobulin, anti-thyroglobulin antibodies and neck ultrasound, selected according to treatment and risk.

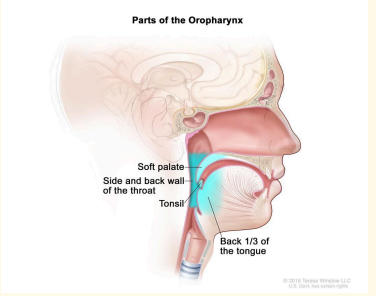
2. Other important benign neck lesions

- Lipoma: soft, mobile, slow-growing; imaging if deep or atypical.
- Cervical thymic cyst: often lateral neck along the thymopharyngeal tract.
- Laryngocele: air-filled laryngeal saccule dilatation; may enlarge with raised intralaryngeal pressure.
- Reactive lymphadenopathy: often tender and temporally related to infection; persistent or suspicious nodes need evaluation.

PHARYNGEAL TUMOURS: SITE-BASED PATTERN RECOGNITION

1. Localise before naming the tumour





Oropharynx

- Tonsil, base of tongue, soft palate, posterior pharyngeal wall.
- Dysphagia, sore throat, referred otalgia, neck node.
- HPV-associated SCC is particularly important at tonsil/base of tongue.

3. Adult neck mass: safe diagnostic sequence

Persistent adult neck mass → focused history and examination, including oral cavity, oropharynx, nasopharynx, larynx, thyroid, skin and scalp → contrast-enhanced CT or MRI when malignancy risk is significant → FNA of an appropriate mass/node → targeted endoscopy and further investigations based on cytology and likely primary site.

- Avoid repeated empirical antibiotics without evidence of bacterial infection.
- If FNA is nondiagnostic, reassess imaging and sampling strategy; core biopsy may be appropriate in selected cases.
- Avoid open biopsy before appropriate imaging and specialist assessment when metastatic head-and-neck malignancy is possible.

Red flags for malignancy

- Persistent, enlarging, firm or fixed mass
- Supraclavicular lymphadenopathy
- Unexplained weight loss or constitutional symptoms
- Dysphagia, odynophagia, hoarseness, referred otalgia
- Tobacco/alcohol exposure, prior skin cancer, or relevant viral risk
- Adult cystic lateral neck mass

NEUROLOGICAL DISEASE OF THE PHARYNX

Localise the neurological deficit

Structure / nerve	Main swallowing contribution	Deficit clue
CN V	Mastication, oral sensation and part of oral-phase control	Weak mastication, impaired oral bolus preparation
CN VII	Lip closure, buccal tone and facial movement	Food pocketing, anterior leakage
CN IX	Pharyngeal sensation and afferent limb of gag reflex	Impaired pharyngeal sensation; findings must be interpreted with other signs
CN X	Palatal elevation, pharyngeal contraction, laryngeal function	Nasal regurgitation, dysphonia, weak cough, aspiration
CN XII	Tongue movement and bolus propulsion	Tongue weakness, atrophy or fasciculation, oral residue

Nasopharynx

- Nasal obstruction, epistaxis, serous otitis media.
- Cranial neuropathies or neck nodes may indicate advanced disease.
- Common malignant tumour: nasopharyngeal carcinoma (NPC).

DISORDERS CAUSING HEARING LOSS DURING CHILDHOOD

1. Acquired and delayed-onset causes

Disorder	Mechanism / classic clue	Investigation or action
Bacterial meningitis	Cochlear injury → fibrosis/ossification; can compromise later electrode insertion	Formal audiology after stabilisation; urgent CI-centre referral for severe/profound SNHL
Congenital CMV	Progressive or delayed-onset cochlear injury	Serial audiology; neonatal diagnostic window is time-sensitive
OME	Middle-ear effusion → CHL	Otoscopy, tympanometry where feasible, age-appropriate hearing assessment
Congenital cholesteatoma	White mass behind an intact TM; usually no preceding perforation or recurrent discharge	Assess extent; temporal-bone imaging as indicated; surgical management
Temporal-bone fibrous dysplasia	Canal narrowing or ossicular involvement → progressive CHL	CT: characteristic ground-glass matrix

Disorder	Mechanism / classic clue	Investigation or action
Osteogenesis imperfecta	Ossicular/stapes abnormalities and/or cochlear involvement → CHL, mixed or SNHL	Fractures, blue sclera in some patients, dentinogenesis imperfecta, family history

Disorder	Mechanism / classic clue	Investigation or action
Down syndrome / cleft palate / primary ciliary dyskinesia / selected cystic fibrosis cases	Increased risk of OME and conductive loss	Longitudinal middle-ear and hearing surveillance
Trauma	CHL, SNHL or mixed loss depending on injury	Sudden loss, vertigo, haemotympanum or suspected temporal-bone injury → urgent assessment
Ototoxic medication	Cochlear or auditory-neural injury	Review exposure, baseline hearing, renal function and monitoring results

Fibrous dysplasia of temporal bone

- Fibro-osseous replacement of normal bone.
- EAC narrowing → CHL; ossicular involvement may contribute.
- CT clue: ground-glass appearance.
- Management depends on symptoms, progression and complications; not every asymptomatic lesion requires aggressive surgery.

OME: who is predisposed?

- Cleft palate / craniofacial abnormalities.
- Down syndrome.
- Primary ciliary dyskinesia, including Kartagener syndrome.
- Cystic fibrosis in relevant clinical contexts.
- Immunodeficiency and recurrent upper-respiratory infections.

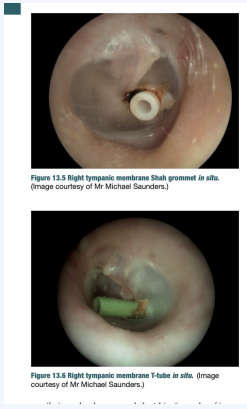


Figure 13.5 Right tympanic membrane Shah grommet *in situ*. (Image courtesy of Mr Michael Saunders.)

Figure 13.6 Right tympanic membrane T-tube *in situ*. (Image courtesy of Mr Michael Saunders.)

OAE VERSUS ABR —

Feature	OAE	ABR
Measures	Sound generated by functioning cochlear outer hair cells and transmitted back through middle ear	Synchronous neural response to acoustic stimulation along the auditory pathway
Main role	Rapid cochlear screening; selected cochlear-function monitoring	Objective threshold estimation and neural-pathway assessment
Middle-ear dysfunction	Can impair/abolish response	Conductive transmission also affects response and interpretation

Feature	OAE	ABR
Classic ANSD	May be preserved	Typically abnormal/absent in classic ANSD
Gives complete behavioural thresholds alone?	No	Can estimate thresholds; clinical interpretation remains necessary

ANSD pattern: preserved OAE with abnormal/absent ABR → suspect auditory neuropathy spectrum disorder. Interpret in clinical context; OAE may change over time.

2. Subglottic stenosis (SGS)

Aetiology and epidemiology

- **Congenital:** cricoid cartilage deformity or abnormal subglottic development.
- Acquired: most commonly associated with intubation injury in the source text; external injury, an excessively high tracheostomy and infection are additional causes.
- Approximately 90% of acquired cases to internal injury such as prolonged intubation.
- Risk factors include repeated intubation, local/systemic infection, reflux and systemic illness such as anaemia or dehydration.
- A correctly selected endotracheal tube should allow an appropriate leak at the specified pressure; avoid excessive pressure and prolonged traumatic intubation.

Myer–Cotton grading

Grade	Percentage obstruction
I	0–50%
II	51–70%
III	71–99%
IV	No detectable lumen

3. Juvenile-onset recurrent respiratory papillomatosis (RRP)

- Caused principally by **HPV-6 and HPV-11.**
- Presents with **progressive hoarseness, weak cry or chronic voice change**; **stridor** indicates significant airway involvement.
- Diagnosis: laryngoscopic visualisation, with histopathology when appropriate.
- Treatment: endoscopic removal/debulking to maintain airway and preserve usable voice.
- Repeated procedures may be required because recurrence is characteristic.
- Adjuvant treatment may be considered for severe or frequently recurrent disease by specialist teams.
- HPV vaccination is important for prevention at population level; it does not replace treatment of established symptomatic disease.

4. Congenital subglottic haemangioma

- May cause progressive airway obstruction in early infancy.
- A cutaneous beard-distribution haemangioma is a useful associated clue; its absence does not exclude airway involvement.
- Airway endoscopy defines the lesion and its extent.
- Propranolol is a major first-line systemic treatment in appropriately selected patients, under specialist supervision with monitoring.
- Further airway intervention depends on severity, anatomy and response to treatment.

2. Subglottic stenosis (SGS) (cont.)

Treatment options — lesion-specific

Intervention	Role / limitation
Conservative observation	Selected mild, stable lesions
Smallest appropriate endotracheal tube / airway rest	Selected acute or evolving injuries under specialist care
Endoscopic dilation	Selected short, mild stenoses; recurrence is possible
Endoscopic laser	Selected mild lesions; not appropriate for every stenosis

Intervention	Role / limitation
Intralesional steroid	Selected cases to limit inflammatory scar response
Mitomycin C	Used in selected endoscopic cases; evidence and indications are not universal
Anterior cricoid split ± stenting	Selected paediatric stenosis according to anatomy and airway plan
Laryngotracheal reconstruction (LTR) with costal cartilage graft	Complex or severe stenosis requiring expansion
Partial cricotracheal resection (PCTR)	Selected severe/refractory stenoses in specialist centres
Tracheostomy	Airway security for critical or complex obstruction

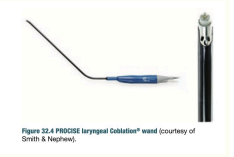


Figure 32.4 PRECISE laryngeal Coblation® wand (courtesy of Smith & Nephew).

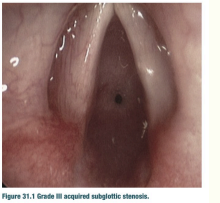


Figure 31.1 Grade III acquired subglottic stenosis.

Complications: recurrent airway obstruction, vocal-fold scarring/dysphonia, distal airway spread in a minority and rare malignant transformation.

5. Final rapid-recall table

Stem	Answer
Commonest cause of congenital stridor	Laryngomalacia
Laryngomalacia + failure to thrive	Consider supraglottoplasty
Laryngomalacia source-reported intervention rate	10–15%
Congenital web	5% of congenital laryngeal lesions in Bullet Points in ENT
Saccular cyst	Stridor 90%; respiratory difficulty 55% in the same source

4. Management algorithm (cont.)

B. Suspected retained foreign body in a stable child

- Keep under appropriate observation and avoid unnecessary oral intake while awaiting definitive assessment.
- Involve ENT, respiratory and anaesthetic teams as appropriate.
- Rigid bronchoscopy is commonly used for removal of tracheobronchial foreign bodies in children.
- Flexible bronchoscopy has selected diagnostic/therapeutic roles depending on the object, site, child and local expertise.
- After removal, inspect for additional fragments, mucosal injury and complications.

5. Delayed diagnosis: complications

- Persistent cough and wheeze.
- Post-obstructive pneumonia.
- Atelectasis and bronchiectasis.
- Granulation tissue and airway stenosis.
- Respiratory failure or sudden complete obstruction.

6. Integrated airway anatomy — clinically relevant points

- Infant airway is smaller; a small reduction in radius can markedly increase airflow resistance.
- The larynx is relatively cephalad in infancy; the epiglottis is proportionally larger.
- The cricoid is a complete cartilaginous ring; clinically important stenosis may occur at the subglottis.
- Infants have limited respiratory reserve and can deteriorate quickly.
- Airway management depends on age, size, pathology, equipment and operator expertise; fixed tube-size rules do not replace clinical assessment.

7. Rapid differentiation: croup versus epiglottitis/supraglottitis

Feature	CROUP (ACUTE LARYNGOTRACHEOBRONCHITIS, VIRAL LARYNGOTRACHEOBRONCHITIS)	Epiglottitis/supraglottitis
Cough	Characteristically barking	Often absent/not prominent
Voice	Hoarse	Muffled voice may occur
Drooling	Usually absent	Concerning feature
Swallowing	Usually preserved	Dysphagia/odynophagia common
Position	Often tolerates lying down	May sit forward/tripod
Appearance	Variable, often non-toxic	May be toxic or severely distressed
Immediate approach	Assess severity; treat according to croup guidance	Airway emergency; controlled specialist airway plan

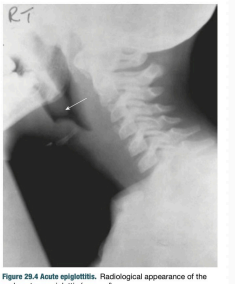


Figure 29.4 Acute epiglottitis. Radiological appearance of the oedematous epiglottis (arrowed).

Bacterial Laryngotracheobronchitis (Bacterial Tracheitis)

Synonyms: Pseudomembranous croup, membranous laryngotracheobronchitis, neonatal necrotising tracheobronchitis.

- Definition: Rare, potentially life-threatening upper-airway infection with mucopurulent secretions and sloughing of respiratory epithelium.
- Age: Mean age approximately 4–8 years; older than typical viral croup.
- Sex: Male predominance.
- Risk factors: Down syndrome, immunodeficiency; often follows viral respiratory infection.
- Pathology: Thick, adherent mucopus + pseudomembranes in subglottis/trachea → ineffective cough clearance → airway obstruction.
- Commonest bacterial isolate: Staphylococcus aureus.
- Other organisms: H. influenzae, Moraxella catarrhalis, Streptococcus pneumoniae, Pseudomonas aeruginosa.
- Viral association: Influenza A (including H1N1); metapneumovirus and other respiratory viruses.

Clinical clues

- High fever and toxic appearance.
- Worsening stridor and respiratory distress.
- Poor or absent response to corticosteroids and nebulised adrenaline/epinephrine—key distinction from uncomplicated croup.
- No single clinical, radiological or laboratory finding reliably distinguishes it from croup.

Diagnosis

- Confirmatory test: direct laryngotracheobronchoscopy (LTB).
- Findings: subglottic/tracheal pseudomembrane, thick mucopurulent secretions and debris extending into bronchi.
- Obtain tracheal cultures for targeted antibiotic therapy.

Superficial IH is typically bright red and raised; deeper lesions may appear bluish or as a subcutaneous swelling. Clinical appearance and evolution help distinguish them from malformations.

Which lesions need treatment?

Treatment is considered when there is:

- Airway compromise or suspected airway IH.
- Visual-axis obstruction or risk of amblyopia.
- Feeding impairment, ulceration, significant pain or bleeding.
- High risk of permanent disfigurement or functional impairment.
- Large segmental facial/scalp lesions, which may signal associated structural anomalies.
- Rare systemic complications, including high-output cardiac failure with extensive disease or consumptive hypothyroidism with extensive hepatic IH.

VASCULAR MALFORMATIONS: LYMPHATIC, VENOUS, CAPILLARY AND AV

Tumour versus malformation

Feature	Infantile haemangioma	Vascular malformation
Nature	Vascular tumour	Abnormal vessel development
At birth	Often absent/subtle	Usually present, though may be subtle
Natural history	Proliferates, then involutes	Grows with the child; does not spontaneously involute in the same way
Flow	Depends on lesion and phase	Classified as low-flow or high-flow
Treatment	Propranolol for many high-risk/problematic IH	Depends on vessel type, flow, extent and symptoms



Diagnosis and management

Clinical situation	Management principle
Small, uncomplicated IH	Observation with follow-up and caregiver education
High-risk IH	Early specialist referral; assess site, size, growth phase and functional risk
IH requiring systemic treatment	Oral propranolol is first-line for most problematic IH when appropriate and medically supervised
Thin superficial residual lesion	Topical timolol may be considered for selected small, superficial IH
Ulcerated lesion	Wound care, pain control and specialist-directed therapy
Persistent residual deformity after involution	Selected laser or surgical correction after specialist review

Propranolol: beta-adrenergic blockade; reduces vascular tumour activity and promotes regression. Initiation requires assessment of contraindications and feeding/illness precautions because hypoglycaemia, bradycardia, hypotension and bronchospasm are important risks. It should not be started unsupervised.

Lymphatic malformations (LM)

1. Definition and epidemiology

- **LM = low-flow vascular malformation** due to abnormal embryological development of the lymphatic system; exact mechanism remains uncertain.
- Abnormal collection of variably sized lymphatic spaces and channels replaces the normal interconnected lymphatic network with one-way valves.
- **75% occur in the head and neck; may extend into the thorax.**
- **Incidence: approximately 1 : 5,000.**
- Account for **6% of paediatric soft-tissue masses**.
- Approximately **50% present at birth;** 90% diagnosed by 2 years.
- Grow proportionately with the child; **unlike infantile haemangioma, they do not undergo true spontaneous involution**.

2. Cyst-size classification

Type	Definition	Clinical relevance
Macrocystic	A few large cysts; volume >2 cm^3	Generally responds better to treatment
Microcystic	Numerous smaller cysts	More difficult to treat
Mixed	Combination of macro- and microcystic components	Common in practice

3. Location-based classification

Type 1 — below mylohyoid

- Anterior and posterior triangles of the neck.
- More often predominantly macrocystic.
- Generally better prognosis.

- Aminoglycosides, particularly gentamicin, may cause vestibular dysfunction; some agents also cause cochlear toxicity.
- Platinum-based chemotherapy can cause ototoxicity.
- Ask specifically about medication exposure; assess hearing and vestibular function where indicated.

6. Persistent imbalance and ataxia

Acute cerebellar ataxia

- Often follows viral illness in young children, with acute gait instability.
- Exclude posterior fossa tumour, intoxication and other serious causes before diagnosing a benign post-infectious condition.

Hereditary and syndromic causes

- **Friedreich ataxia:** progressive ataxia with associated neurological/systemic features.
- **Charcot–Marie–Tooth disease:** peripheral neuropathy, distal weakness and gait disturbance.
- Refsum disease: retinitis pigmentosa, neuropathy, hearing loss and ataxia; potentially treatable metabolic disorder.
- **Chiari malformation:** may cause imbalance and other neurological manifestations.
- CHARGE, Pendred and Usher syndromes: consider with congenital hearing loss and associated syndromic features.
- NF2: vestibular schwannomas may cause hearing and balance symptoms.

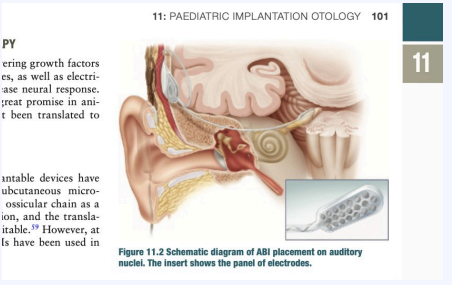


Figure 11.2 Schematic diagram of ABI placement on auditory nuclei. The insert shows the panel of electrodes.

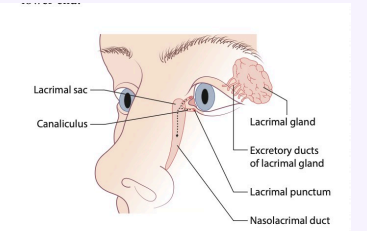


Figure 25.1 The lacrimal system, comprising the lacrimal gland, the lacrimal puncta, canaliculi, lacrimal sac and nasolacrimal duct.

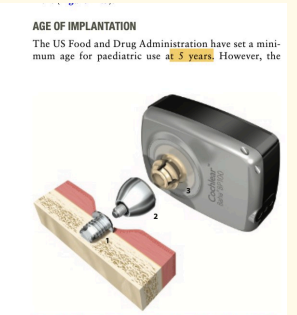


Figure 11.3 Diagram of percutaneous bone-anchored hearing aid with titanium fixture in the bone (1), abutment (2) attaching to the fixture and hearing aid (3) (placed later and removable by patient).



Figure 11.1 Internal part of cochlear implant (Cochlear N512).

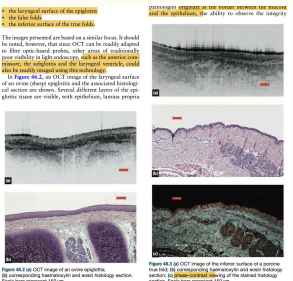


Figure 46.2 (a) OCT image of an ovine epiglottis. (b) corresponding haematoxylin and eosin histology section. Scale bars represent 150 µm.

Figure 46.3 (a) OCT image of the inferior surface of a porcine true fold. (b) corresponding haematoxylin and eosin histology section. (c) phase-contrast view of the stained histology section. Scale bars represent 150 µm.

Polarization-sensitive OCT (PS-OCT)

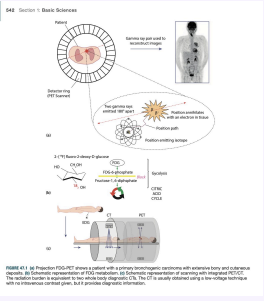
Additional principle: Measures changes in light polarization caused by tissue properties, particularly birefringence.

- Organized collagen fibres alter polarization in a predictable manner.
- Random scattering alters polarization less predictably.
- Potential application: indirect assessment of vocal-fold lamina propria layers and collagen organization.
- Vocal-fold scarring changes collagen density/orientation and disrupts lamina propria biomechanics → reduced pliability and impaired voice quality.
- PS-OCT may help localize scars and characterize tissue architecture, but these interpretations require further validation.

PET–CT: PRINCIPLES AND SQUAMOUS CELL CARCINOMA

Core principle

- PET: functional/metabolic imaging using a positron-emitting radiotracer.
- CT: anatomical localization and attenuation correction.
- PET–CT: fused metabolic and anatomical images; more accurate localization than PET alone.



- Axial resolution: approximately 1–15 µm.
- Penetration depth: approximately 2–3 mm in non-transparent tissue.
- Images can be obtained in situ, non-destructively and in real time.
- Fibre-optic probes permit integration with endoscopes, catheters and surgical instruments.

Laryngeal OCT findings

- Epithelium, basement membrane and lamina propria may be distinguished.
- Glands and vessels may be visible.
- Epithelial thickness and transparency can be assessed.
- Potential targets include hyperplasia, early keratosis, papilloma and early mucosal abnormalities.
- Imaging of the epiglottis, false folds and inferior surface of true vocal folds has been described.

Applications and limitations

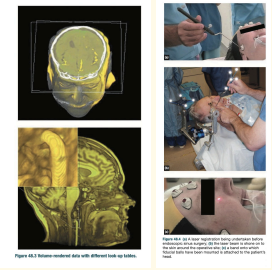
Application	Status / limitation
Laryngeal lesions and biopsy guidance	Promising correlation with histology; not a replacement for histological confirmation
Paediatric airway modelling	FD-OCT/LR-OCT explored for subglottic stenosis and localization of obstruction
Tracheal/cartilage assessment	Investigational
Parathyroid identification	OFDI and micro-OCT explored; not established as a universal replacement for frozen section
Cochlear implantation/stapes surgery	Small studies and experimental localization applications
Post-dilatation laryngotracheal stenosis	OCT combined with EBUS reported in small case series

Exam traps

- OCT uses infrared light, not ultrasound.
- Excellent resolution does not imply deep penetration.
- OCT is structural imaging, not a histological specimen.
- PS-OCT uses polarization/birefringence as an additional contrast mechanism.
- Evidence described in the source is predominantly observational, pilot or proof-of-concept; routine clinical roles remain limited.

FDG mechanism

1. Intravenous **^{18}F-fluorodeoxyglucose (FDG)** enters cells through glucose transport mechanisms.
2. Hexokinase phosphorylates FDG → FDG-6-phosphate.
3. Further metabolism is limited compared with glucose → tracer becomes relatively trapped.
4. Positron emission → annihilation with an electron → two photons emitted approximately 180° apart.
5. Coincidence detection reconstructs the PET signal; CT localizes the uptake anatomically.



ENT applications

- Endoscopic sinus surgery: difficult revision cases with distorted or absent landmarks; localization of the frontal recess during Draf II/III procedures.
- Skull base surgery: preoperative planning, bone-flap design, localization of small lesions and critical structures.
- Transnasal/transsphenoidal surgery: localization of the sella.
- Otology: localization of facial nerve, petrous apex lesions, internal auditory canal tumours and structures such as dura and jugular bulb.
- Surgical simulation: develops endoscopic hand–eye coordination and permits structured assessment of technical skills.

Embolic agents:

Agent	Type / permanence	Main features and uses	Key exam trap
Autologous blood clot	Temporary; particulate/clot	Occludes afferent arteries	Temporary occlusion
Gelfoam	Temporary; gelatin particles	Derived from pork-skin gelatin; pliable, can be cut to size; proximal vessel occlusion, often after distal particle embolization	Does not provide the same permanent occlusion as coils or liquid adhesives
Polyvinyl alcohol (PVA) particles	Permanent; non-spherical particles	Penetrate tumour vessels; widely used for preoperative tumour devascularization	Less predictable penetration than spherical particles; very small particles increase non-target ischaemia risk
Trisacryl gelatin microspheres — Embosphere®	Permanent; spherical particles	More predictable embolic penetration than non-spherical PVA; available in different sizes	Small particles penetrate distally but increase normal-tissue ischaemia risk

Registration and tracking

- Registration: aligns the preoperative image coordinates with the patient's actual position.
- Anatomical landmarks include the tragus, outer canthus and nasion.
- Landmark registration alone: approximately 3–4 mm error in the source.
- Adding 40–100 sampled points can improve accuracy to approximately 2–3 mm.
- Fiducial markers and surface scanning are alternative methods to improve registration.

Tracking method	Principle	Limitations
Optical/infrared	Tracks markers or instruments using a camera	Line-of-sight obstruction; relatively expensive
Electromagnetic	Tracks instruments within a magnetic field	Distortion from nearby metal and field interference
Ultrasound-based	Tracks position using ultrasound signals	Affected by temperature, air currents, echoes and delay
Inertial	Measures changes in motion/rotation	Slow positional changes may be measured poorly

INTERVENTIONAL NEURORADIOLOGY AND EMBOLIZATION

Core principle

Endovascular embolization selectively occludes abnormal vessels or a tumour's vascular bed using catheter-delivered embolic material. The aim is to reduce blood flow, control haemorrhage or treat selected vascular lesions.

Indications

- Preoperative devascularization of highly vascular tumours → reduce operative blood loss and facilitate resection.
- Arteriovenous shunts and vascular malformations.
- Refractory epistaxis.
- Selected lesions where surgery is contraindicated or palliation is required.
- Selected haemorrhage from head and neck malignancy.
- Other techniques described include percutaneous embolization, image-guided biopsy and targeted intra-arterial drug delivery.

74. PHARMACOLOGY AND ANAESTHETICS

74.1 Local Anaesthetics

Classification and mechanism

- Amides: lidocaine, bupivacaine.
- Esters: cocaine, benzocaine.
- Most local anaesthetics (LAs) are weak bases and exist as a protonated form at body pH.
- Block Na^+ channels in axons → prevent generation of action potentials (APs).
- Small-diameter nerve fibres are more sensitive to LAs:
  - Coarse touch and movement are usually spared initially.
- At high concentrations, LAs can depress other excitable tissues, e.g. myocardium.
- LAs are use-dependent: degree of block is proportional to rate of nerve stimulation.
- More drug molecules in protonated form enter Na^+ channels when they are open → greater inactivation.
- Opening of Na^+ channels causes Na^+ entry exceeding K^+ exit, further depolarizing the membrane and opening more Na^+ channels.
- LAs block Na^+ channels by preventing opening of their h-gates.
- Duration and potency relate to lipid solubility.
- Most amides cause vasoconstriction at low doses and vasodilatation at higher concentrations.
- Parallel use of a vasoconstrictor prolongs the effect.
- Amides are metabolized in the liver.
- Esters, except cocaine, are hydrolysed by plasma pseudocholinesterase.
- Cocaine causes vasoconstriction by blocking norepinephrine reuptake and potentiating sympathetic activity.

74.2 Neuromuscular Blocking Drugs

- Competitive blockers: atracurium; duration 15–30 minutes.
- Depolarizing blocker: suxamethonium; rapid onset, duration 3–7 minutes.
  - Drug does not dissociate rapidly from receptors at cholinergic nerve terminals.
  - Prolonged receptor activation is produced.
  - Endplate depolarization initially causes a brief train of muscle APs.
  - Neuromuscular block then occurs because:
    - Inactivation of voltage-sensitive Na^+ channels in the surrounding muscle fibre membrane prevents further AP generation.
    - Activated receptors transform to a desensitized state and become unresponsive to acetylcholine (ACh).
  - Initial muscle twitch may cause muscle pain the next day.

74.3 Autonomic Nervous System (cont.)

Receptor drugs

74.1 Local Anaesthetics (cont.)

Clinical effects and toxicity

- Main systemic effects are CNS effects: sedation and light-headedness; sometimes anxiety and restlessness.
- High toxic doses cause twitching and visual disturbances.
- Severe toxicity → convulsions, coma and cardiorespiratory depression.
- Hypersensitivity reactions may occur, especially with atopic patients.

Duration and maximum doses

- Lidocaine: rapid acting and most stable; duration of action 90 minutes.
- Bupivacaine: slow onset (30 minutes), but duration 8 hours.
- Maximum dose of lidocaine = 200 mg (500 mg with adrenaline); example: 5 mL of 2% lidocaine with adrenaline = 100 mg.
- Bupivacaine maximum = 60 mL of 2.5 mg/mL (0.25%) solution.

74.1.1 Moffat Solution

- Cocaine 10%:
  - 1 mL or 5%: 2 mL at 50 mg/mL = 100 mg (1.5 mg/kg maximum).
- Adrenaline 1:1000: 1 mL (maximum 100 mg/10 min, as shown in the screenshot).
- $NaHCO_3$: 2 mL.
- Risks:
  - Arrhythmias, especially with halothane.
  - Hypercapnia and hypertension.

74.1 Local Anaesthetics (cont.)

74.1.2 Laryngopharyngeal Local Anaesthesia

- Krause method: swab soaked in 4% lidocaine, held with Krause forceps in each piriform fossa for 1 minute.
- Percutaneous injection of LA at the greater cornu of the hyoid also anaesthetizes the superior laryngeal nerve.
- Sprayed/nebulized lidocaine.
- Benzocaine lozenge.

74.1.3 Ear Anaesthesia

- Auriculotemporal block: 2 mL lidocaine injected anterior to the meatus.
- Greater auricular nerve block: inject anterior and posterior to the mastoid tip.

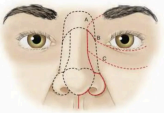
74.3 Autonomic Nervous System

- Sympathetic: norepinephrine at α and β receptors.
- Parasympathetic: ACh at muscarinic receptors.
- All ANS preganglionic fibres are myelinated and release ACh from nerve terminals → activate nicotinic receptors at ganglia.
- Postganglionic fibres are short in parasympathetic nerves.
- Sympathetic nerves synapse in paravertebral/prevertebral ganglia or abdominal plexuses.
- ACh action is greater at muscarinic receptors → parasympathetic effects predominate.

2. Incisions in ENT (cont.)

Incision	Main use in source
Blair	Parotidectomy
Modified Blair / Lazy-S	Parotidectomy
Modified facelift	Parotidectomy
Facelift / rhytidectomy	Parotidectomy
Mini incision	Parotidectomy
Gluck–Sorenson	Total laryngectomy
Dean	Parapharyngeal abscess
Cruciate	Retropharyngeal abscess
Kocher	Thyroidectomy

Modified Weber-Ferguson incision





- A-lynch extension;
- B- subciliary extension;
- C- infraorbital extension.

D – **Borle's extension** – ALLOWS HAREVEST OF TEMPORALIS FLAP

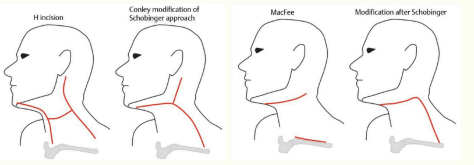
3. Functional MRI (fMRI) (cont.)

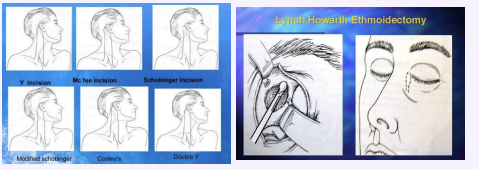
Topic	Condensed points
Relative versus absolute	BOLD fMRI measures relative signal changes between cognitive states in a session; PET can provide absolute tissue-metabolism measures

Gamma Knife Stereotactic Radiosurgery

Principle and equipment

- **Stereotactic radiosurgery (SRS):** computer-guided, highly focused radiation delivered to a defined target; usually a single high-dose session rather than fractionated radiotherapy.
- SRS systems include Gamma Knife, linear accelerator (LINAC) and proton-beam systems.
- Uses 3D localisation from CT/MRI/PET-CT, patient immobilisation, high-precision collimation and image-guided targeting.
- Radiation damages target-cell DNA → loss of reproductive capacity → gradual tumour control/shrinkage.
- Gamma Knife: fixed array of 201 cobalt sources in this source (other systems/texts describe 192–201); beams converge on the target.
- Multiple "shots" combine to create the treatment volume; steep dose fall-off limits irradiation of surrounding tissue.
- Source states targeting accuracy of 1–2 mm and treatment time of approximately 20 minutes to 2 hours.





3. Functional MRI (fMRI)

Topic	Condensed points
Definition	Functional neuroimaging using MRI to infer brain activity from local blood-flow changes during neuronal activation
BOLD principle	Blood Oxygenation Level Dependent contrast; active grey matter has increased blood flow and a higher oxyhaemoglobin:deoxyhaemoglobin ratio
Haemoglobin properties	Oxyhaemoglobin is diamagnetic; deoxyhaemoglobin is paramagnetic
Signal mechanism	Neural/synaptic activation → increased local perfusion → relative reduction in deoxyhaemoglobin → altered local magnetic susceptibility → increased BOLD signal
What it reflects	Indirect haemodynamic/metabolic response related to synaptic activity, rather than a direct recording of neuronal firing
Neurovascular coupling	Nitric oxide is the key mediator in the source; other mediators include K^+, H^+, adenosine, prostacyclin, noradrenaline and serotonin
Other functional modalities	EEG/MEG detect electrical or magnetic consequences of neuronal depolarisation; PET measures metabolic changes
Spatial resolution	Ability to distinguish nearby sites; determined by voxel size, reported as approximately 1–5 mm
Temporal resolution	Limited by sampling/repetition time (TR) and the haemodynamic response

Topic	Condensed points
Perfusion MRI	Measures local perfusion or associated small increases in blood volume during activation

Gamma Knife Stereotactic Radiosurgery (cont.)

Indications listed