UNIVERSITY OF LEICESTER

Leicester Medical School

Neurology and Special Senses

Student Handbook: 2026 -7

Including Student Workbooks

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Code of Conduct and Consent

General Conduct

Courtesy to patients, their partners and families and to members of nursing, midwifery and medical staff is essential at all times and reflects your professionalism. You must introduce yourself adequately in all clinical areas, and dress appropriately.

Your identity badge must be worn at all times; members of staff may deny you access to clinical areas if you are not wearing an ID.

Attendance

The code of conduct for Phase 2 clearly states that 100% attendance of time-tabled sessions is expected. In that respect, your clinical attachment is to be treated in the same way as full-time paid employment.

Where your absence is not avoidable (sickness etc), please inform your clinical team/Tutor, as well as the Undergraduate Coordinator.

In the absence of an adequate apology/sick notification etc, failure to attend sessions will result in an unsatisfactory grade for attendance.

Course Structure

Welcome to the Neurology and Special Senses block. This 7-week block includes an induction week followed by 1 week of Ophthalmology, Neurology, ENT, and Dermatology. During the final week, you will receive a mixture of activities from all specialities including some mandatory teaching sessions arranged by your Undergraduate Coordinator. There will be an End of Bock Assessment during the final week comprising of a single best answer (SBA) exam paper followed by feedback and your overall sign-off. You should make yourself available to attend your feedback session and should not make any early travel arrangements. In this workbook, each speciality has their own section outlining your objectives, clinical opportunities and resources including those on Blackboard.

In addition to the end of block exam we will be releasing a short answer question (SAQ) exam paper. This will be uploaded to blackboard on the Monday of the final week of the block and we encourage all students to replicate exam conditions when answering these questions. The answers to these questions will be released later in the week and this will be communicated to you by your undergraduate coordinator. This paper is for your benefit only and to practise answering NSS SAQs before your end of year final exams. It will not count toward your NSS block result.

During your final week you will also be timetabled to attend a NSS simulation half day, dates and times will be communicated to you by your undergraduate coordinator. During this half day you get to experience OSCE style scenarios for all four of the NSS specialties. You gain invaluable experience with using the same equipment that will be used during your end of year OSCEs as well as having the opportunity to ask any questions related to the equipment/scenarios that you may have.

My Knowledge Map (MKM)

During your time in Neurology and special senses you will be expected to complete and submit forms on My Knowledge Map (MKM) throughout the duration of the block. By recording your attendance, which clinical areas you have been to, and any strengths or areas of improvement you noticed in your knowledge. You should aim to submit a form for every theatre, Clinic & Face to face teaching session, this helps to provide an insight into how well you have engaged during your time

on the block. Miss Nagini Sarvananthan will review your MKM accounts, along with your end of block exam mark to help grade you with an overall block grade. These grades will be shared during your one-to-one feedback session at the end of the block should you be selected for a session.

Reading List

You can find copies of the Neurology and Special Senses Reading List here:

https://rl.talis.com/3/leicester/lists/0FBE6676-0C58-DF7F-0ABE-C302A314BA0D.html

Online Resources:

This is a link to the new online radiology learning resource which we will go live with later this week, feedback from a core group of students has been overwhelmingly positive. There are still a number of talks in the pipeline that will appear in the next couple of weeks but the core topics are completed, in particular there will be more talks in paediatrics, MSK, cancer care and related to the vertical themes.

https://www.lmsradiology.org/home

Learning Outcomes, Aims and Outcomes

Throughout Phase 2, you will work towards the learning outcomes of the **Leicester Medical School Curriculum** (see the Phase 2 Curriculum Guide), to attain the core knowledge, skills and behaviours defined in the **Medical Licensing Assessment (MLA) Content Map** and to achieve the **GMC Outcomes for Graduates**. You should refer to these documents and resources regularly to guide your learning.

The aims and objectives listed below support these outcomes and align with the Leicester Medical School Curriculum, MLA Content Map and GMC Outcomes for Graduates. They show how you can use your Neurology and Special Senses Placement to achieve these outcomes and goals.

Special Senses – ENT and Ophthalmology

Aims

The placement(s) aim to equip students to:

- Take a history and carry out an appropriate examination in a patient with an ENT or ophthalmic problem
- Understand the impact of dysfunction or loss of a special sense for a patient and their carer, and the resources required to manage the disability
- Understand acute, common, and important ophthalmic and ENT disorders, especially those that have systematic features or appear in other parts of the course

Objectives (Ophthalmology)

By the end of the placement(s) you should be able to:

Demonstrate your ability to identify the important causes for the symptoms of:

- ocular discomfort
- visual disturbance
- a red eye
- ocular discharge
- an abnormal pupil

Take an appropriate history to reach a provisional diagnosis

Elicit selectively normal and common abnormal signs in the eyes to test diagnostic hypotheses, in particular you should be able to:

- test and record visual acuity in adults and children
- assess a patient for the presence of squint by means of the corneal reflexes and cover testing
- examine the fundus with a direct ophthalmoscope
- examine visual fields by confrontation
- examine the ocular media of both adults and children by means of the red reflex

- distinguish between ophthalmic complaints requiring immediate referral, those which require referral but are not urgent and those with can be managed by the newly qualified practitioner
- Describe the management of complications and organ damage of chronic conditions where the eye is potentially involved, such as visual problems associated with diabetes, thyroid disease and hypertension

Objectives (ENT)

Demonstrate your ability to identify the important causes of

- nasal blockage
- rhinitis
- epistaxis
- deafness
- pain in the ear
- pain in the throat
- difficulty swallowing
- swelling of the neck
- hoarseness
- disturbance of balance

By taking an appropriate history to reach a provisional diagnosis

Elicit selectively normal and common abnormal signs in the ears, nose and throat including the use of an otoscope and a tuning fork to test diagnostic hypotheses

- use investigations selectively to confirm diagnostic hypotheses
- formulate a simple management plan including an assessment of the need for referral

Neurology

Aims

This placement aims to ensure that students are able to take a history and perform a competent neurological examination of a patient presenting with a neurological problem. Students should be familiar with investigation and management of common neurological conditions.

The placement aims to develop diagnostic reasoning skills in reference to neurological symptoms.

A number of the learning objectives below will be achieved in the Integrated Care Block (year 4) or the Foundation Assistantships (year 5).

Objectives

By the end of the course students should be able to:

Identify the important neurological causes, appropriate investigations and management for:

- headache
- dizziness or vertigo
- fits

- unconsciousness
- incoordination, gait disturbance and impaired balance
- movement disorders
- focal or generalised weakness
- speech, swallow and language disturbance
- disturbances of sensation and neuropathic pain
- urinary or faecal incontinence
- visual disturbance
- cognitive problems, changes in personality or behavior

Be aware that neurological dysfunction can be non-organic in nature

With regard to the Neurological Examination:

- perform a focused but thorough neurological examination
- perform a rapid screening neurological examination
- perform a neurological examination on patients with an altered level of consciousness
- recognise and interpret abnormal findings from the neurological examination

With regard to the use of investigations:

- recognise common indicators for lumbar puncture, EEG, CT , PET-CT and MRI in patients with neurological disease
- describe in detail the performance of a lumbar puncture
- interpret abnormalities in CSF

Use anatomical knowledge to localise neurological lesions and so be able to differentiate between lesions in:

- cerebral hemispheres
- cerebellum, brain stem and cranial nerves
- basal ganglia
- spinal cord – the importance of a spinal level
- nerve root/plexus
- peripheral nerve (the commoner forms of mononeuropathy, polyneuropathy, and mononeuritis multiplex)
- neuromuscular junction – myasthenia gravis
- muscle – myositis, muscular dystrophies

With regard to potential emergencies recognize, evaluate and provide initial management or referral for the following:

- raised intracranial pressure
- subarachnoid haemorrhage
- meningitis/encephalitis
- status epilepticus
- spinal cord or cauda equina compression
- head trauma
- acute respiratory distress
- temporal arteritis
- acute bulbar palsy

Dermatology

Aims

Dermatology is also included in several places through the curriculum, including Phase 1 and Primary care in Year 3. Cancer Care and Child Health in Year 4 can also expose students to dermatology presentations. The dermatology teaching in this block will allow students to revisit topics and consolidate their learning from other blocks.

Objectives

History and Examination:

- Take a history and examine a patient presenting with a dermatological condition.
- Describe and record a skin rash/lesion accurately.
- Demonstrate patient-centredness, holism and the ability to recognise the impact of disease
- Be able to recognise that dermatological conditions appear differently depending on skin pigmentation.
- Recognise the dermatological manifestations of systemic disease

Demonstrate knowledge of common dermatological conditions :

- skin infections and infestations
- Bacterial: impetigo, folliculitis, cellulitis
- Viral: Herpes simplex, chicken pox, shingles, viral warts, molluscum contagiosum
- Fungal: dermatophytosis, candidiasis
- Parasitic: scabies and lice infestations
- Eczema
- acne vulgaris
- psoriasis
- allergic rashes and urticarial
- benign and malignant melanocytic lesions
- Describe the epidemiology and pathophysiology of benign, premalignant and malignant melanocytic lesions
- Apply the above knowledge to assess a patient presenting with a pigmented lesion, using a holistic approach, including full skin examination
- Recognise that excision comprises the investigation and treatment of choice and that sampling is not appropriate
- non-melanoma skin cancers (NMSC)
- Describe the epidemiology and pathophysiology of premalignant lesions: actinic keratosis and Bowen's disease
- Describe the epidemiology and pathophysiology of basal cell carcinoma and squamous cell carcinoma

Acute and Emergency Dermatology:

- Know the principles of diagnosing and managing patients with the following presentations, including referral to appropriate specialties:
- Acute (and chronic) urticarial rashes
- Drug reactions
- Eczema with secondary infection (including eczema herpeticum)
- Necrotising fasciitis
- Staphylococcal scalded skin syndrome

Management of dermatological conditions:

- Demonstrate knowledge of therapeutics for common skin disorders
- Demonstrate knowledge of the British Association of Dermatologist Handbook
    - https://cdn.bad.org.uk/uploads/2024/04/15113327/Derm_Handbook_3rd-Edition-_Nov_2020.pdf
- Manage common skin conditions presenting to primary care.
- Apply a stepwise approach to treating skin problems ensuring that exacerbating factors have been addressed
- Prescribe safely topical treatments accounting for (where relevant) potency, formulation, body site, adverse effects, interactions, contraindications and patient preference including:
- Emollients and soap substitutes
- Corticosteroids
- Antibiotics
- Antifungals
- Phototherapy
- Retinoids
- Methotrexate
- Ciclosporin, Azathioprine
- Biologics

Neurology: Student Workbook

Updated August 2026

Neurology

Introduction

Welcome to your Neurology placement! Neurology deals with the diagnosis and management of patients with diseases of the central and peripheral nervous system and neuromuscular conditions. You already have a sound knowledge base to work with from phase I and we hope you will find it an exciting challenge to learn how to put this knowledge into clinical practice. Neurology is often detective work, and a systematic and logical approach are needed and will underpin the teaching in this phase.

It is important that you take this opportunity to clerk and examine as many patients as possible as this is the only way you will become proficient at, and comfortable with, examining patients and you will start to get a feel for what is normal as well as abnormal. You will also find how to direct the patients clearly in examination to gain the information you need.

You will find neurology cases are common in the outpatient clinics and acute neurology, stroke, and medical wards as well as on the rehabilitation wards so there should be plenty of opportunity for practice and experience.
There may be opportunities to present a case to one of the doctors, but it is good practice to try to present cases to one another as well as you will soon realise that it is not an easy skill to acquire.

This handbook contains pointers to what you need to know but you will need find more details in your reading.

Clinical Skills

Listed below are the clinical skills that you need to acquire in this block:

1. Take a full NEUROLOGICAL HISTORY to include physical, psychosocial and social aspects to allow you to formulate some hypotheses as to what maybe causing the patient's symptoms.

2. Summarise the history to clarify the problem and allow clear communication.

E.G. A 34 year old nurse presented with a 4 week history of gradually progressive sensory and motor symptoms affecting her legs, with a sensory level at the umbilicus and some urgency of micturition. She has a history of previous optic neuritis 5 years ago.

3. Perform a NEUROLOGICAL EXAMINATION.
    - Neurological examination will be modified in patients who are unconscious or uncooperative.
    - It is essential to recognise and interpret any abnormal findings as you go along.

Before you examine do you have some ideas as to where in the nervous system the problem may be to help you guide a more focussed examination? Use the examination as a tool to answer your questions to yourself and help you solve the case

4. Use ANATOMICAL KNOWLEDGE to localise neurological lesions.
    - Be able to differentiate between lesions in:
        - Cerebral Hemispheres - lobes in the cerebrum
        - Cerebellum
        - Brainstem and Cranial Nerves
        - Basal Ganglia
        - Spinal cord - localisation of spinal level
        - Nerve root and or plexus
        - Peripheral nerves
        - Neuromuscular junction
        - Muscle
        - Understand the existence of non-organic (

Peripheral nerves can be affected in different distributions:
-Peripheral neuropathy, often length dependant e.g. diabetic or alcoholic peripheral neuropathy
-Individual peripheral nerve palsies e.g. carpal tunnel syndrome
-Nerve root and plexus lesions

What is characteristic about lesions at each of these neurological levels? e.g. in a peripheral neuropathy you might expect a glove and stocking distribution of sensory loss and lower motor neurone signs.

5. Using all of the above to formulate a DIFFERENTIAL DIAGNOSIS.
6. Understand the use of INVESTIGATIONS
    - These will include indications for lumbar puncture (LP), EEG, EMG and scanning procedures to include CT, MRI and PET scanning.
7. Formulate an appropriate MANAGEMENT PLAN

You will Identify the important neurological causes for the symptoms of:

- Focal or generalised weakness
- Incoordination
- Gait disturbance/ impaired balance
- Movement Disorders especially Parkinson's Disease
- Urinary or faecal incontinence
- Dizziness/ Vertigo
- Visual symptoms including double vision
- Speech and language disturbances
- Bulbar symptoms - speech and swallowing
- Cognitive problems
- Behaviour and personality change
- Headache
- Neuropathic pain syndromes
- Sensory disturbances
- Blackouts/ Loss of consciousness

How many classic neurological gait patterns can you describe?

Monocular double vision is not neurological – if it occurs, then the lesion must be in the eye or it could be a functional symptom.
When patients complain of monocular double vision on one side they sometimes mean there is double vision looking to that side – make sure you clarify what they mean.
Very subtle double vision, where the images are overlayed but only slightly displaced, is sometimes perceived as blurring by the patient.

The pattern of sensory disturbance will give you a big clue as to where the lesion is. If in doubt map it out carefully on examination and draw the area of sensory disturbance on a diagram of the body to see if it fits a dermatome, peripheral nerve, glove and stocking, cape or half-cape, saddle …….

Be aware that neurological dysfunction can be non-organic in nature

You should be able to recognise, evaluate and manage the following common neurological disorders:

Potential Emergencies:

- Raised intracranial pressure
- Subarachnoid haemorrhage
- Meningitis / Encephalitis
- Status epilepticus
- Acute Stroke
- Spinal cord or cauda equina compression
- Head trauma
- Acute respiratory distress
- Temporal arteritis
- Acute bulbar palsy

Protocols for common, serious, emergency scenarios are usually available for staff on hospital intranet sites e.g. you can find advice on UHL intranet for status epilepticus, SAH and others.
Advice also available in NICE Guidelines.

Less acute but common disorders:

- Epilepsy
- Migraine and other common headache disorders
- Idiopathic Parkinson's Disease and Parkinsonism
- Neuromuscular weakness (including MND, GBS, MG, muscular dystrophies)
- Multiple sclerosis
- Dementia syndromes including Alzheimer's disease
- Polyneuropathy (including diabetic neuropathy, GBS)
- Lumbar and Cervical spondylosis
- Bell's palsy
- Carpal tunnel syndrome and Ulnar nerve palsy
- Radiculopathies
- Essential tremor
- Intracranial space occupying lesions (Including haemorrhages)
- Cerebral Palsy

In addition, there are less common, but important, neurological conditions and syndromes of which you should be aware. It is advised that you look up the following:

- Horner's Syndrome
- Cerebellopontine angle lesions e.g. acoustic neuroma
- Subacute combined degeneration of the spinal cord
- Individual cranial nerve palsies - physical signs and clinical causes
- Hypoxic ischaemic encephalopathy
- Trigeminal neuralgia
- Wernicke's encephalopathy

Which cranial nerves are often damaged after surgery for acoustic neuroma?

Neurology Examination.

Clinical signs in neurology can be subtle so the examination must be done carefully – comparing side to side can be very useful.
If you are not sure have a look at the 2 videos of neurological examination available on blackboard that demonstrate correct technique.
https://blackboard.le.ac.uk/webapps/blackboard/content/listContentEditable.jsp?content_id=_1984116_1&course_id=_11294_1

Comprehensive neurological examination.

Be able to perform the following parts of the neurological examination and be able to identify and interpret the significance of abnormal findings.

A. Mental Status
   i) Level of alertness (GCS, AVPU)
   ii) Attention and concentration
   iii) Orientation
   iv) Language function (fluency, comprehension, repetition, naming)
   v) Memory (Short and long term)
   vi) Calculation
   vii) Visuospatial awareness

B. Cranial Nerves
   i) Olfactory Nerve - sense of smell
   ii) Optic Nerve - visual acuity, fundoscopy
   iii) Oculomotor Nerve - eye movements
   iv) Trochlear Nerve - superior oblique muscle
   v) Trigeminal Nerve - facial sensation, muscles of mastication
   vi) Abducens Nerve - lateral rectus muscle
   vii) Facial Nerve - muscles of facial expression
   viii) Auditory Nerve – hearing
   ix) Glossopharyngeal Nerve - pharyngeal sensation, taste posterior tongue
   x) Vagus Nerve - movement soft palate, pharynx , larynx
   xi) Accessory Nerve - movement sternomastoid and trapezius
   xii) Hypoglossal Nerve - movement of tongue

What is the difference between upper and lower motor neurone facial weakness?

C. Motor Function
   i) Gait
   ii) Muscle bulk, wasting
   iii) Fasciculation
   iv) Involuntary movements
   v) Tone - resistance to passive movement
   vi) Power - muscle strength
   vii) Coordination - limb movements, balance, Romberg test

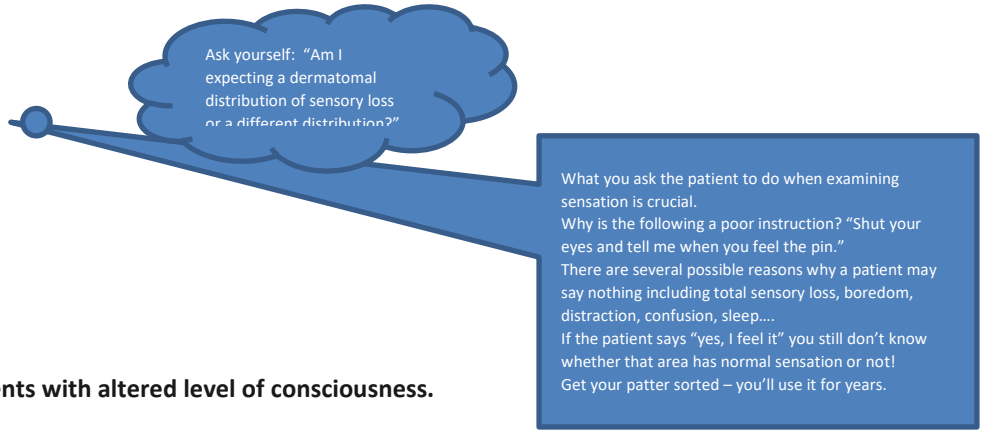
It is not the details of the motor examination that will help you understand the case but whether you are finding upper or lower motor neurone signs.
Don't get bogged down in detail – see the bigger picture!

what is the difference between spasticity and lead pipe rigidity?

How would you examine a patient who you suspect might have a cerebellar lesion?

D. Reflexes
   i) Deep tendon reflexes - biceps, triceps, brachioradialis, knee jerk, ankle jerk
   ii) Plantar responses
   iii) Jaw jerk
   iv) Gag reflex

E. Sensation
   i) Pain and temperature
   ii) Proprioception
   iii) Vibration
   iv) Light touch



Neurological examination in patients with altered level of consciousness.

You should know how to conduct a neurological evaluation of an unconscious or semi-conscious patient who cannot cooperate with the examination. This will involve examination of:

1. Level of consciousness
2. Pupil responses
3. Fundoscopy
4. Brain stem reflexes - corneal, vestibulo-ocular (doll's eyes), gag reflex
5. Deep tendon reflexes
6. Plantar response

Neurological examination in patients with Parkinson's Disease.

Specific conditions.

Epilepsy

What is the definition of epilepsy?

What are the essential features in the history to suggest a seizure?

How would you investigate someone with suspected seizures?

Seizures can occur in different forms depending on the cause and site of electrical discharge in the brain. What is the difference between a generalised and partial seizure?

International League Against Epilepsy (ILAE) Classification of seizure types basic version.

<table>
<tr><th colspan="2">Focal Onset</th><th>Generalised onset</th><th>Unknown onset</th></tr>
<tr><td>Aware</td><td>Impaired awareness</td><td>Motor
• Tonic-clonic
• Other motor

Non-motor (absence)</td><td>Motor
• Tonic-clonic
• Other motor

Non-motor (absence)</td></tr>
<tr><td colspan="2">Motor onset

Non-motor onset</td><td></td><td>Unclassified</td></tr>
<tr><td colspan="2">Focal to bilateral tonic-clonic</td><td></td><td></td></tr>
</table>

Patient based learning exercises and video links

Here are some videos of patients experiences in epilepsy (you may need to log in with your username and password to view these videos):

- Temporal Lobe Epilepsy - https://speakingclinically.co.uk/videos/temporal-lobe-epilepsy/
- Absence Seizures - https://speakingclinically.co.uk/videos/absence-seizures/
- Non-convulsive Status Epilepticus - https://speakingclinically.co.uk/videos/non-convulsive-status-epilepticus/

Think about personal safety, medication, triggers, lifestyle, work, recreation, contraception, pregnancy

What management advice would you give to a patient (and their relatives) who has been diagnosed with epilepsy?

Think about first aid and patient safety. More detailed advice may be appropriate depending on relationship with the patient.

What are the regulations with regard to driving?

DVLA Assessing Fitness to Drive – a guide for medical professionals:
https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/965900/MIS828_interactive_020321_Final.pdf

List 5 causes of secondary epilepsy:

Antiepileptic drugs are used to treat patients who have recurrent seizures. The choice of drug will be determined by the type of epilepsy and features of the individual patient (e.g. age, sex). They may have side effects and interact with other drugs. They should be used with caution in women of childbearing age due to potential teratogenic effects and interaction with the oral contraceptive pill. Antiepileptic drugs are started at low dose and titrated upward until seizure freedom occurs or highest recommended dose or side effects occur. Some drug doses need to be monitored by intermittent blood tests.

N.B. Sodium valproate should **NEVER** be prescribed to women of childbearing potential outside of a tertiary referral centre where it will only be used as a last resort and under strict conditions regarding contraception.

Recently MHRA have released new guidance on the use of sodium valproate in men.

Look up MHRA guidance on use of sodium valproate in both women and in men.

Listed are the more commonly used antiepileptic drugs.

For each, indicate:

a) For which type of epilepsy, they are indicated
b) Side effects and contraindications
c) Major drug interactions
d) Requirements for blood monitoring

Drug Name	**Indication**	**Side Effects and Contraindications**	**Interactions**	**Blood Monitoring**
Levetiracetam				
Lamotrigine				
Sodium Valproate				
Topiramate				
Carbamazepine				
Phenytoin				
Phenobarbitone				

Status epilepticus

This condition is a medical emergency. It is defined as a single seizure lasting more than 30 minutes or recurrent without regaining consciousness. The risk of permanent brain damage increases the longer the condition persists. Status can occur in patients with pre-existing epilepsy, but may be the presenting feature of new intracranial pathology.

Look up the protocols for the management of status epilepticus and describe the appropriate management and drugs that can be used.

N.B. most hospitals publish protocols for the management of serious medical emergencies such as status epilepticus on their intranet sites

Reflect on a case of epilepsy that you have seen. Describe the presentation and clinical management. What was the medium to long-term impact upon the patient?

Additional Materials:

NICE guidelines Epilepsy Diagnosis and Management: https://www.nice.org.uk/guidance/cg137

If you want to test your understanding and knowledge of epilepsy here are some cases (you may need to log in with your username and password to view these videos):

Case Study 267 – https://leicester.capsule.ac.uk/case/4542

Case Study 281 – https://leicester.capsule.ac.uk/case/4549

Case Study 770 – https://leicester.capsule.ac.uk/case/22594

Case Study 772 – https://leicester.capsule.ac.uk/case/22616

Headache

Headache is a common disorder. The key to establishing the cause of the headache is to take a comprehensive and focussed history. Whilst most headaches are self-limiting and do not require investigation, others can be disabling without accurate treatment or are indicative of serious underlying pathologies requiring urgent investigation and treatment.

Chronic benign headaches may, nevertheless, have a significant impact on quality of life, affecting work, relationships and, sometimes leading to depression.

A comprehensive headache history should include:

1. Mode of onset
2. Duration
3. Nature of headache - sharp, dull, throbbing
4. Site of headache - localised, variable, generalised
5. Pattern and timing. - including time of day, relation to menstruation
6. Provoking and relieving factors e.g. coughing, posture
7. Associated symptoms including aura
8. Drug history - particularly analgesic usage

Medication overuse can lead to chronic headache which resolves when the drug is withdrawn. Examples of drugs which can cause this problem are:

1. Paracetamol, aspirin or NSAIDs used for 15 days per month or more
2. Triptans, opioids or ergot preparations for 10 days a month or more

95% of headaches which present in general practice are benign in aetiology and do not require further investigation. Referral for investigation is indicated in patients who present with “Red flags”.

Red flags 

Red flags are specific forms of headache presentation that may signify serious underlying pathology and require further, urgent investigation. These can be listed as follows:

1. Thunderclap headache
2. Associated fever
3. Meningism +/- non blanching skin rash.
4. Raised intracranial pressure – headache on waking and worse on lying down, worse on coughing, papilloedema
5. New neurological deficit
6. New cognitive dysfunction
7. Personality change
8. Impaired or deteriorating conscious level
9. Recent head injury
10. New onset headache in elderly - giant cell arteritis
11. Significant change in pattern of chronic headache
12. History of malignancy or impaired immunity
13. Postural headaches – i.e. worse on lying, worse on standing (e.g. post LP headache)

For the following headache conditions describe:

- Characteristic clinical manifestations, investigations (if any), management including drug treatment where appropriate, possible complications and prognosis.

Conditions	Clinical manifestations	Investigations	Management	Complications	Prognosis
Tension headache					
Migraine					
Medication overuse headache					
Cluster headache					
Subarachnoid haemorrhage					
Space occupying lesion					
Meningitis					
Idiopathic intracranial hypertension					
Temporal arteritis					
Post traumatic headaches					
Sinusitis					
Acute angle closure glaucoma					
Venous sinus thrombosis					

Watch the videos of patients experiences with different causes for their headaches (you may need to log in with your username and password to view these videos):

- Bacterial Meningitis: https://speakingclinically.co.uk/videos/bacterial-meningitis/
- Migraine: https://speakingclinically.co.uk/videos/common-migraine/
- Post Concussion Syndrome: https://speakingclinically.co.uk/videos/post-concussion-syndrome/
- Chiari malformation with syringomyelia - https://speakingclinically.co.uk/videos/chiari-malformation-with-syringomyelia/
- SpeakingClinically: The woman whose fitness run didn't do what it said on the tin - https://speakingclinically.co.uk/videos/sudden-headache-probably-migraine/

Additional Materials:

NICE guidelines on Headache: https://www.nice.org.uk/guidance/cg150

Recommended – easy to use and clear!

BASH (British Association for the Study of Headache) Guidelines: https://www.bash.org.uk/guidelines/

If you want to test your understanding and knowledge of headache here are some cases (you may need to log in with your username and password to view these videos):

a) Case Study 17 - https://leicester.capsule.ac.uk/case/3427
b) Case Study 49 – https://leicester.capsule.ac.uk/case/4601
c) Case Study 61 – https://leicester.capsule.ac.uk/case/4620

Lumbar puncture

When performing an LP always consider measuring CSF opening pressure using a manometer.

Examination of the CSF is indicated in a limited number of clinical situations. Lumbar puncture should not be performed in patients with focal neurology or raised intracranial pressure without prior neurological imaging.

Clinical circumstances in which CSF examination may be helpful include:

1. Suspected subarachnoid haemorrhage
2. Suspected meningitis/encephalitis
3. Immunological disorders such as multiple sclerosis or Guillain-Barré Syndrome

Describe the typical CSF findings in the following:

Bacterial meningitis	
Viral meningitis	
Tuberculous meningitis	
Subarachnoid haemorrhage	
Guillain-Barré Syndrome	

Neurodegenerative conditions are usually asymmetric in the early stages.

Idiopathic Parkinson's DIsease (IPD)

Idiopathic Parkinson's Disease (IPD) is a progressive degenerative disorder characterised by neuronal loss in the brainstem and basal ganglia. There is loss of dopaminergic neurones in the substantia nigra that leads to inadequate dopamine transmission. The characteristic neuropathological finding is Lewy Body formation in affected neurones. The condition is usually sporadic in nature, but some genetic variants do exist.

Diagnosis.

IPD is essentially a clinical diagnosis and is one of the commonest causes of the clinical syndrome of Parkinsonism. The Brain Bank Criteria are the gold standard for diagnosis and the essential clinical features are:

How would you examine a patient with PD to demonstrate the characteristics of the condition and what words would you use to describe your findings?

Step 1. Diagnosis of Parkinsonian Syndrome:

- Bradykinesia
- At least one of the following
    - Muscular rigidity
    - 4-6 Hz rest tremor
    - postural instability not caused by primary visual, vestibular, cerebellar, or proprioceptive dysfunction

Step 2. Exclusion of other causes of parkinsonism.

Step 3. Supportive prospective positive criteria for Parkinson's disease
Three or more required for diagnosis of definite Parkinson's disease in combination with step one:

- Unilateral onset
- Rest tremor present
- Progressive disorder
- Persistent asymmetry affecting side of onset most
- Excellent response (70-100%) to levodopa
- Severe levodopa-induced chorea
- Levodopa response for 5 years or more
- Clinical course of ten years or more

IPD is a slowly progressive disease and not all these features are seen in the early stages. To diagnose Parkinsonism only bradykinesia and rigidity are essential elements. Tremor may be predominant in some patients but absent in others. Characteristic gait and facial hypomimia develop as the disease advances. Other 'non-motor ' symptoms can also be prominent in some patients.

Watch the videos of patients experiences with Parkinson's Disease(you may need to log in with your username and password to view these videos):

- The man who dropped a brand new casserole dish - https://speakingclinically.co.uk/videos/parkinsons-disease/
- The woman whose stiffness was helped by deep brain stimulation - https://speakingclinically.co.uk/videos/parkinsons-disease-3/

List some non-motor symptoms of IPD:

As IPD progresses the symptoms and signs worsen. The disease can be staged using standardised rating scales such as the Hoehn and Yahr scale and Unified Parkinson's Disease Rating Scale (UPDRS).

Drug treatment of IPD.

IPD cannot be cured but some of the symptoms are dramatically alleviated by drug treatment. A significant improvement with treatment will distinguish IPD from other causes of Parkinsonism. Since there is no test to confirm the diagnosis, response to treatment is used to confirm a suspected clinical diagnosis.

List the commonly used medications for treating IPD along with their mode of action and major side effects:

Medication	Mode of action	Major side effects

Nausea and vomiting are common side effects from drugs used to treat PD but are also common non-motor symptoms experienced by patients unrelated to medications. Many common antiemetic drugs can worsen parkinsonian symptoms or cause drug induced parkinsonism. Therefore, drugs such as haloperidol and metoclopramide should not be used due to their anti-dopaminergic activity. Domperidone is an anti-emetic which works on peripheral dopamine receptors (it does not cross the BBB so it cannot make PD worse) and is commonly used in PD related nausea. It would be used at low dose and for the shortest time possible in selected patients bearing in mind potential cardiac side effects.

Nearly all patients with IPD will respond well to drug treatment and, in fact, failure to respond should raise doubt about the accuracy of the diagnosis. However, as the disease advances, response to treatment becomes poor and side effects from the drugs can be disabling. In these circumstances non oral treatments are sometimes used.

Look up and list some of the non-oral treatments that can be used in these circumstances.

Supportive care in IPD.

Due to its long term and progressive nature, IPD sufferers need to receive support from a multidisciplinary team. This consists of:

- Medical specialist (Neurologist or Geriatrician)
- PD specialist nurse
- Physiotherapist
- Occupational therapist
- Speech and Language Specialist
- Dietician (later stages)
- Psychiatrist (sometimes)

We recognise 4 distinct phases as the disease evolves:

1. Early stage - Soon after diagnosis, symptoms are mild and normal life possible
2. Maintenance stage - Good response to treatment and no major disability
3. Advanced stage - Poor response to drugs with motor side effects
4. Palliative stage - Unable to live independently and in need of multidisciplinary support

As IPD progresses and the degenerative process worsens, the response to treatment becomes less effective and the patient will develop adverse effects which may be both motor and non motor.

Describe the complications that are seen in advancing IPD:

Parkinsonism

Parkinsonism is a syndrome which manifests as bradykinesia and rigidity plus or minus tremor. IPD is common, but other causes of this syndrome must always be considered. Management and need for investigation is different to IPD and will depend upon the cause. Some relatively rare neurological conditions may mimic IPD and are known as Parkinson plus syndromes including progressive supranuclear palsy and multiple system atrophy. You should know that these exist but need not study each in detail.

List the clinical features of the following conditions that can be mistaken for IPD:

Vascular pseudoparkinsonism	
Drug-induced Parkinsonism	
Benign essential tremor	
Dementia with Lewy bodies	
Progressive Supranuclear Palsy	
Multiple System Atrophy (Shy-Drager Syndrome)	

Additional Materials:

NICE guidelines Parkinson's Disease: https://www.nice.org.uk/guidance/ng71

If you want to test your understanding and knowledge of Parkinson's Disease here are some cases (you may need to log in with your username and password to view these videos):

- Case Study 152 – https://leicester.capsule.ac.uk/case/5703
- Case Study 662 – https://leicester.capsule.ac.uk/case/4629

Motor Neuron Disease (MND)

MND is a progressive, degenerative neurological disease with rapid progression and poor prognosis.

Degeneration occurs in both upper and lower motor neurones (anterior horn cells) in the spinal cord and brainstem. The condition, therefore, most commonly presents with mixed upper and lower motor neurone signs although there are some less common subtypes. Bulbar palsy can lead to speech and swallowing problems and respiratory failure can occur when respiratory muscles are involved. Sensory pathways, special senses and micturition are not involved. Dementia can be seen in some variants. The condition is usually sporadic, but the condition is inherited as an autosomal dominant in 10% of cases.

Diagnosis

The diagnosis should be suspected in patients who present with mixed upper and lower motor neurone signs. It is confirmed using electromyography (EMG).

Mixed UMN and LMN signs might be wasting and fasciculation in muscles at the same location as brisk reflexes. In the case of mixed bulbar palsy the tongue may be waster and fasciculating ("bag of worms") but, at the same time, it can be slow, stiff and spastic, also possibly associated with a positive jaw-jerk.

Watch the video of a patients experience (you may need to log in with your username and password to view these videos):

- The man with slurred speech whose muscles rippled - https://speakingclinically.co.uk/videos/amyotrophic-lateral-sclerosis/

Treatment

MND is incurable. The rate of progression can be mildly slowed by the use of riluzole (a glutamate inhibitor). Symptomatic treatment can be provided for palliative complications such as bulbar palsy and respiratory failure.

Prognosis

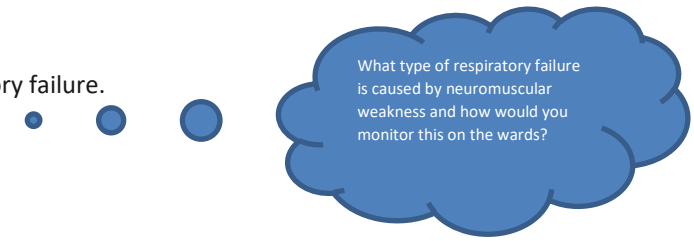
The prognosis is poor and will depend upon the clinical presentation. Average survival time is 3 years. The clinical variants of MND are:

- Amyotrophic Lateral Sclerosis
- Progressive Bulbar Palsy
- Progressive Muscular Atrophy (LMN) – rare
- Progressive Lateral Sclerosis (UMN)– rare

Describe the clinical features and prognosis for each of these variants:

Amyotrophic Lateral Sclerosis	
Progressive Bulbar Palsy	

Two major effects of MND are to cause dysphagia and respiratory failure.



Describe how these can be managed:

Additional Materials:

NICE Guidelines Motor Neurone Disease: https://www.nice.org.uk/guidance/ng42

Multiple Sclerosis (MS)

MS is an acquired, immune-mediated inflammatory, demyelinating, condition that affects both the brain and spinal cord. It does not affect the peripheral nervous system.

MS is commoner in women than men and typically occurs in temperate regions. Symptoms typically commence in early life (20s and 30s) in the form of visual or sensory disturbances. Weakness and clumsiness of the limbs combined with bladder symptoms are common. Typically, the early symptoms will relapse and remit but over time progressive disability will occur.

There are 3 main patterns of clinical presentation:

1. Relapsing remitting
2. Secondary progressive
3. Primary progressive

Watch the videos of patients experiences with Multiple Sclerosis (you may need to log in with your username and password to view these videos):

- The man who got into the bath but couldn't get out – https://speakingclinically.co.uk/videos/multiple-sclerosis/
- The woman with double vision and ataxia – https://speakingclinically.co.uk/videos/multiple-sclerosis-3/
- The woman who thought that her trousers were rubbing – https://speakingclinically.co.uk/videos/primary-progressive-multiple-sclerosis/

Look up and describe the difference in these 3 variants.

Relapsing remitting	
Secondary progressive	
Primary progressive	

Modes of clinical presentation of MS.

1. Optic neuritis
2. Double vision
3. Sensory disturbance in face or limbs
4. Weakness due to upper motor neurone involvement
5. Impaired balance or clumsiness of the limbs
6. Neuropathic pain (e.g. trigeminal neuralgia)
7. Bladder symptoms
8. Cognitive impairment (although this usually occurs late)

What is your method for examining visual fields to confrontation? Would you pick up a large central scotoma that is characteristic of this condition?

Describe the presentation of a patient with:

a. Optic neuritis

b. Trigeminal neuralgia

Aetiology of MS.

The cause of MS is unknown. It is thought to be an abnormal immune response to environmental triggers such as low vitamin D levels and EBV infection in people who are genetically predisposed. It leads to immune-mediated demyelination in CNS neurones and ultimately nerve cell degeneration. Initially, after the acute inflammatory stage, neurones may remyelinate and recover leading to clinical remission. Recurrent episodes eventually lead to neuronal degeneration that, in turn, will produce a permanent clinical deficit.

Diagnosis of MS.

The clinical diagnosis is based upon the presence of multiple lesions of the CNS occurring both in time (recurrent episodes) and space (different sites). It should be made by a consultant neurologist and be based upon the 2010 McDonald criteria:

1. Lesions consistent with an inflammatory process
2. No alternative diagnosis
3. Multiple lesions in time and space (Relapsing remitting MS)
4. Progressive neurological deterioration for 1 year (Primary progressive MS)

A relapse maybe diagnosed if a person:

1. Develops new symptoms
2. Has rapid worsening of existing symptoms
3. Symptoms last for more than 24 hours in the absence of infection or other cause after a stable period of one month or more

Investigations in MS.

Helpful investigations are:

1. MRI of brain or spinal cord - will demonstrate demyelination plaques
2. Immunoelectrophoresis of CSF - will show oligoclonal bands of IgG

Treatment of MS.

MS cannot be cured. It is an immune condition and will respond to treatments which modify the immune system. MS damages the central nervous system and other drugs can be used to modify the symptoms of MS.

Treatment options are therefore:

1. Treatment for an acute relapse
2. Treatments that can modify disease progression long term
3. Symptomatic treatments

Look up and describe drugs that might help the following symptoms in MS:

Spasticity	
Neuropathic pain	
Depression	
Incontinence	
Fatigue	
Paroxysmal symptoms	

Drugs which alter the long-term progression of MS are known as disease modifying drugs. Look up some of these drugs and describe how they may work and their potential side effects.

Disease modifying drugs	Mechanism of action	Side effects

How may pregnancy affect a patient with known MS? What advice would you give to a woman who is contemplating pregnancy?

Additional Resources:

NICE Guidelines Multiple Sclerosis: https://www.nice.org.uk/guidance/cg186

Neurology Resources

- Blackboard
  - Videos relating to cranial nerve examination and peripheral nerve examination https://blackboard.le.ac.uk/webapps/blackboard/content/listContentEditable.jsp?content_id=_1984116_1&course_id=_11294_1
  - Chapters relating to different topics in neurology are available.
- Recommended Text Books (available in Leicester University library)
  - Clinical Neurology: a primer, Peter Gates. Churchill Livingstone.
  - Lecture Notes Neurology, Lionel Ginsberg. Wiley-Blackwell.
  - Understanding Neurology: a problem oriented approach, John Greene & Ian Bone. Manson Publishing.
  - Crash Course (3rd edition): Neurology. Christopher Turner

Online neurology text books (available on Leicester University website):

Crash Course Neurology Updated Edition - E-Book
Yogarajah, Mahinda;Horton-Szar, Daniel;and more Elsevier Health Sciences 2015

Understanding Neurology : A Problem-Oriented Approach
Bone, Ian;Metcalfe, Richard;and more Taylor & Francis Group 2007

Fundamentals of Neurology : An Illustrated Guide
Mattle, Heinrich;Mumenthaler, Marco Thieme Medical Publishers, Incorporated 2016

Oxford Handbook of Neurology : Oxford Handbook of Neurology (2nd Edition)
Manji, Hadi;Connolly, Seán;and more Oxford University Press USA - OSO 2014

Clinical Cases Uncovered
Macleod, Malcolm;Simpson, Marion;and more John Wiley & Sons, Incorporated 2011

Clinical Neurology (7th Edition)
Simon, Roger P.;Aminoff, Michael J.;and more McGraw-Hill Professional Publishing 2009

Lecture Notes: Neurology
Ginsberg, Lionel John Wiley & Sons, Incorporated 2010

Neurology : A Clinician's Approach
Tarulli, Andrew Cambridge University Press 2010

Neurology
Westover, M. Brandon Wolters Kluwer Health 2016

Case-Based Neurology
Singh, Anuradha Springer Publishing Company 2011

Practical Neurology
Biller, José Wolters Kluwer 2017

Essential Neurology
Wilkinson, Iain;Lennox, Graham Blackwell Publishing Ltd. 2005

Localization in Clinical Neurology
Brazis, Paul W.;Masdeu, Joseph C.;and more Wolters Kluwer Health 2011

Special Senses (ENT and Ophthalmology): Student Workbook

Updated August 2026

Aims & Objectives

Aims (Special Senses – ENT and Ophthalmology)

The placement(s) aim to equip students to:

Take a history and carry out an appropriate examination in a patient with an ENT or ophthalmic problem

Understand the impact of dysfunction or loss of a special sense for a patient and their carer, and the resources required to manage the disability

Understand acute, common, and important ophthalmic and ENT disorders, especially those that have systematic features or appear in other parts of the course

Objectives (ENT)

By the end of the placement(s) you should be able to:

Demonstrate your ability to identify the important causes for the symptoms of:

- ocular discomfort
- visual disturbance
- a red eye
- ocular discharge
- an abnormal pupil
- by taking an appropriate history to reach a provisional diagnosis
- elicit selectively, normal and common abnormal signs in the eyes to test diagnostic hypotheses, in particular:
- test and record visual acuity in adults and children
- assess a patient for the presence of squint by means of the corneal reflexes and cover testing
- examine the fundus with a direct ophthalmoscope
- examine visual fields by confrontation
- examine the ocular media of both adults and children by means of the red reflex
- distinguish between ophthalmic complaints requiring immediate referral, those which require referral but are not urgent and those with can be managed by the newly qualified practitioner
- Describe the management of complications and organ damage of chronic conditions where the eye is potentially involved, such as visual problems associated with diabetes, thyroid disease and hypertension

Demonstrate your ability to identify the important causes of

- nasal blockage
- rhinitis
- epistaxis
- deafness
- pain in the ear

- pain in the throat
- difficulty swallowing
- swelling of the neck
- hoarseness
- disturbance of balance
- by taking an appropriate history to reach a provisional diagnosis
- elicit selectively normal and common abnormal signs in the ears, nose and throat including the use of an otoscope and a tuning fork to test diagnostic hypotheses
- use investigations selectively to confirm diagnostic hypotheses
- formulate a simple management plan including an assessment of the need for referral

Objectives (Ophthalmology)

By the end of the course, you should be able to

1. Demonstrate your ability to identify the important causes for the symptoms of:
    - ocular discomfort
    - visual disturbance
    - a red eye
    - ocular discharge
    - an abnormal pupil
2. By taking an appropriate history, to reach a provisional diagnosis, use investigations appropriately to confirm the diagnostic hypothesis and formulate a simple management plan (the workbook ophthalmology cases provide some good examples – please work through the cases).
3. Elicit selectively, normal and common abnormal signs in the eyes to test diagnostic hypotheses, in particular:
    - test and record visual acuity in adults and children
    - assess a patient for the presence of squint by means of the corneal reflexes and cover testing
    - examine the anterior segment and external eye with a pen torch, perform the swinging light test for a relative afferent pupillary defect
    - examine the fundus with a direct ophthalmoscope
    - use safely mydriatic and fluorescein diagnostic drops
    - examine visual fields to confrontation
    - examine the ocular media of both adults and children by means of the red reflex

4. Distinguish between ophthalmic complaints requiring immediate referral and those that require referral but are not urgent
5. Discuss the extent and causes of preventable blindness worldwide

The curriculum for undergraduate ophthalmology is updated and available on the Royal College of Ophthalmologists website.

https://www.rcophth.ac.uk/wp-content/uploads/2022/03/Curriculum-UG-RCOphth-220309.pdf

Ophthalmology Placement Introduction

Welcome to your Ophthalmology placement! We hope that you enjoy your time with us. This workbook is intended for all students currently on their Ophthalmology placement.

Information for Students on Ophthalmology placement

Your Ophthalmology placement makes use of key resources to direct your learning. It is anticipated that you will attend clinical activities which will include outpatient clinics with a clinician or orthoptist and ophthalmology theatres. It is also anticipated that you will review the lectures on blackboard, you will fill out your Ophthalmology case studies in this workbook. There will be opportunities for you to discuss the workbook answers either with your own peers or clinicians you meet during your placement. There will be an opportunity for you to meet with your assigned Ophthalmologist (UHL) or the lead Ophthalmologist (out block) to ask questions, review any areas of learning and to consolidate your learning. Both you and the Ophthalmologist should also complete the sign off to record what was discussed.

Timetable/Clinical Activity

The administrator at each site will contact you prior to the start of your placement to guide you in terms of your timetable of activities for your allocated site. Please be aware that self-directed learning via the workbook and blackboard are also a vital part of your learning for Ophthalmology.

The start times for clinics and operating theatres may differ from when the consultation with the clinician first takes place. The ophthalmology patients have to undergo vision testing, orthoptic assessments, visual field testing and optical coherence tomography (OCT) or other investigations prior to the consultation. On arrival in clinic, if the clinician has not started seeing patients, please ask a member of staff if you can observe the pre clinic assessments as they form part of your curriculum learning. **For operating theatre sessions, please ask a member of staff on the ophthalmology suite if you can speak to patients allocated for your list, then join the surgeons on the preoperative ward round which usually takes place 30 minutes prior to the theatre start time.**

Workbook

The first part is a collection of case studies which you are encouraged to work through during your placement and to discuss any difficult questions with the clinicians you meet either during seminar teaching or in your clinical sessions. The second part of this workbook provides an overview of anatomy and physiology and some clinical aspects of ophthalmology, but should not replace self guided study and reading around the subject.

Blackboard

The Ophthalmology placement makes large use of Blackboard. Please logon and visit the Ophthalmology area of Blackboard where you will find key information. This includes a series of videos of lectures covering key topics delivered by Ophthalmologists. There are also videos of ophthalmology clinical skills techniques.

Borrowing of Ophthalmoscopes

UHL based students will be sent an email regarding loan of ophthalmoscopes. Students undertaking placement at Northampton and Lincoln should borrow an ophthalmoscope from the Clinical Skills Unit there. It is important that all loaned equipment is returned promptly in order to enable the next group of students to obtain the ophthalmoscopes for their placement. The medical school will be informed of any students failing to return the equipment as instructed.

Keeler Prize

The Keeler Prize will be awarded to the highest ranked Leicester student in the Royal College of Ophthalmologists Duke Elder Prize Examination. The exam is usually held in March of each year. More information is available from www.rcophth.ac.uk or by contacting Victoria Smith (Victoria.smith@uhl-tr.nhs.uk)

Please feel free to contact us if needed.

Case Studies

Case Study 1

A 47 year old woman is a frequent attender at your General Practice. She has a 2 year history of bilateral itchy eyes and crusting lid margins which tend to be worse in the mornings. She has come to see you today, complaining of a foreign body type sensation in her eyes.

What is your diagnosis?

On examination, her eyelid margins are red and inflamed and on fluorescein staining you find that she has punctate staining on the inferior third of the cornea.

What is your initial advice about managing the condition?

What further medical treatment may be appropriate?

Case Study 2

A 27 year-old man comes into the emergency Department. He is complaining of severe eye pain, watering and sensitivity to light, having splashed oven cleaner in his eye.

What is your immediate management?

Which type of chemical injuries are more severe and why?

On examination you see the following:

- Red eye, expect for pallor of vessels close to the limbus in the interpalpebral fissure
- Cloudy cornea
- Hazy view of Iris detail

What is the cause of the unusual conjunctival injection?

CONT OVERLEAF….

What further treatment is used to treat this condition?

What are the long-term complications?

Does this patient need urgent referral to eye casualty? Please explain your answer:

Case Study 3

A 71 year old man is referred by his optometrist with reduced visual acuity associated with a substantial change in his spectacle prescription over the last six months. He tells you he is having problems driving at night and often gets glare when looking at oncoming vehicles.

What are the most likely causes for his symptoms?

How can his change in prescription be explained?

You decide that he is a suitable candidate for surgery, what operations are available for his eye condition?

At roughly what level of vision do most people undergo surgery?

CONT OVERLEAF….

The patient asks you what the operation involves, write down here what you would tell him:

What complications of surgery would you warn him of?

What percentage of world blindness does this condition account for – according to WHO?

List the other commonest causes of blindness world-wide and the treatment & public health measures that can be taken to treat or prevent them:

<u>Case Study 4</u>

A 75 year old woman presents complaining of difficulty reading for the past two months.

What is the commonest cause of registration as blind in the UK?

On further history taking she explains that she finds it hard to see her sons face clearly.

What two investigations can be used to investigate the diagnosis?

What are the two subtypes of this disease?

How can you differentiate between them?

What treatment in available?

Case Study 5

A 70 year-old woman presented to A&E with a one day history of sudden, painful loss of vision in the left eye, headaches and nausea developing over the course of 24 hours.

Name a likely diagnosis?

You find out that she has a history of being long sighted, needing reading glasses first at the age of 40 years. On examination what classic signs would you look for?

What medical treatment would need to be immediately initiated?

Give two systemic medications or diagnostics drugs that could induce this condition.

Case Study 6

A 45 year-old man who is asymptomatic, is referred to you by his optician with probable chronic simple glaucoma

What is the definition of Glaucoma?

What risk factors would you ask about in your history?

What are the characteristics of the visual field loss?

On examination what other features would support your diagnosis?

What classes of medication would you use to treat Mr Jones. Please give an example of each.

What laser and surgical treatment is available if medical therapy is ineffective?

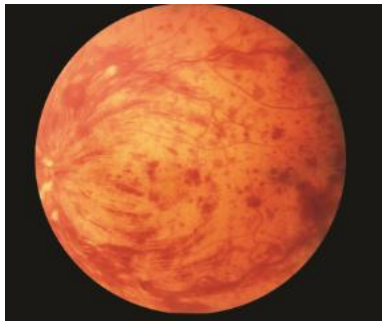
What are the principles of Glaucoma surgery?

Case Study 7

A 70 year old man comes in complaining of painless loss of vision in his left eye.
His past medical history includes type 2 diabetes (diet controlled) and hypertension.
He has had one myocardial infarction three years ago. On examination you find that he has a left relative afferent pupillary defect.

Name 3 conditions in which you would see a RAPD.

On Ophthalmoscopy you see the following:



https://www.asrs.org/content/images/cms

There are widespread retinal haemorrhages and a few soft exudates in the left fundus.
The Right eye is normal.
What is the diagnosis?

What are the possible outcomes for this patient without treatment?

Case Study 8

A 34 year old woman known to have Rheumatoid arthritis presents with severe aching pain in her right eye and a headache for the past four days. The pain is worse when she moves her eyes and she has not been able to sleep as a result of the pain.

On examination, her eyes are very tender and she has mild photophobia.

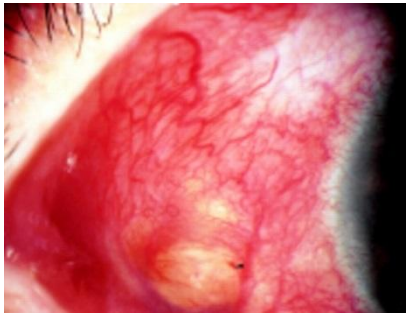


Image taken from the British journal of Ophthalmology

There is injection of the scleral vessels with a 4mm diameter area of pallor
What is the most likely diagnosis?

What is the cause of the pallor within the area of redness?

This may be a rare presentation of a more serious underlying systemic disease.
Name 2 systemic non-infectious associations of the condition, other than rheumatoid arthritis.

What investigations would you like to request to exclude underlying pathologies?

How do you treat her presenting condition?

Case Study 9

Helen, a 4 year girl is referred to clinic for an eye check. On slit lamp examination you see flare and cells in the anterior chamber. You instill dilating drops.

What does the picture show?

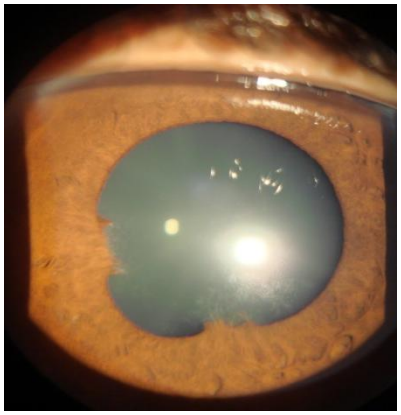


Image taken from Visoncarecentre.com

What is your Ophthalmological diagnosis?

What underlying disease do you suspect?

What treatment is required for her eyes?

What long-term ophthalmological complications may develop?

Case Study 10

A 60-year-old lady on treatment for controlling her systemic hypertension for the last 20 years presents to eye casualty with clinical symptoms and signs suggestive of allergic conjunctivitis.

What would you expect to find on examining the fundus

Will her vision be affected?

Case study 11

A 38-year-old lady presents with headaches and episodes of transient blurring of vision. Her fundus appearance is as below (similar in both eyes).

What are the next steps in management of this patient?

What is the visual prognosis for this patient if the systemic condition is adequately treated?

Clinically Applied Eye Anatomy

The following sections are designed to highlight the anatomy of the eye, relating structure to function. We have attempted to relate this to clinical practice as far as possible. This is not a substitute for a textbook! It should, however, explain some key concepts and allow you to gain a depth of understanding when reading about ocular pathology in order to meet your objectives.

In order to use this section you will need to refer to pictures illustrating the anatomy of the eye using your anatomy textbook.

Functional and Applied Anatomy & Physiology of the Eye

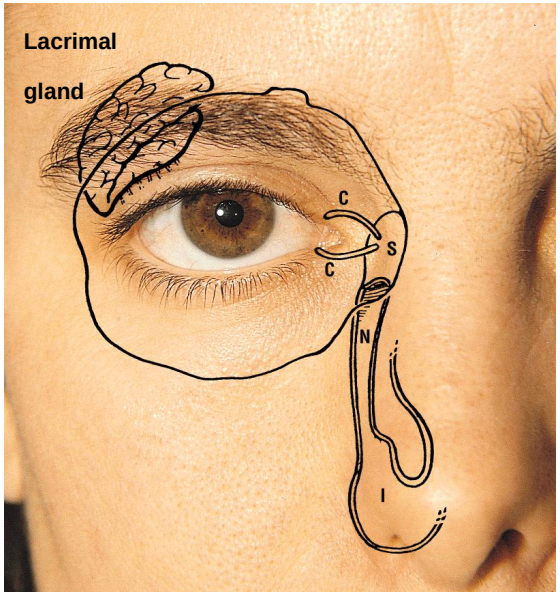
Facial injuries may involve the bones of the orbit and the surrounding structures such as the eyeball, nerves, blood vessels and air sinuses. It is important to have knowledge of the eye and its environment. This will require study of the bony orbit, the muscles and neurovascular structures associated with the eye and the structure of the eyeball.

The orbit is a pyramidal cavity in the facial skeleton which contains the eyeball and its associated muscles together with a number of nerves and blood vessels. The periosteum (periorbita) lining the bones of the orbit forms the fascial sheath of the eyeball and is continuous at the optic canal and supraorbital fissure with the periosteal layer of the dura mater; over the orbital margin and through the infraorbital fissure, it is continuous with the periosteum covering the external surface of the cranium. Around the anterior orbital rim, it is continuous with the orbital septum.

Apart from the lateral wall, the remaining walls of the orbit, particularly the medial and inferior, are thin and may fracture easily.

Eyelids and the Lacrimal System

The front of the eye is exposed and the eyelids and lacrimal glands are important protective mechanisms. The eyelids, which protect the cornea and the eyeball from injury, keep the cornea moist by the covering it with a tear film. On their inner surface, the eyelids are lined by palpebral conjunctiva that is reflected onto the eyeball where it is continuous with the bulbar conjunctiva overlying the anterior surface of the eye. Movement of the eyelids occurs via the orbicularis oculi to close the eye, and levator palpebrae superioris muscles to open the eye, innervated by the facial and oculomotor nerves respectively.



The eyelids are supported by a thick sheet of dense fibrous tissue (tarsal plate).

The tarsal plate contains the tarsal (meibomian) glands, a modified sebaceous gland secreting lipid, which is discussed further below. The eyelid margin has 2 to 3 rows of eyelashes which are more numerous on the upper eyelid. The tarsal glands open at the lid margin. Modified sweat glands (*glands of Moll*) and sebaceous glands (*glands of Zeiss*) also open at the lid margin. Obstruction to the tarsal glands can lead to a painless swelling of the eyelid, known as a *chalazion (sometimes called a meibomian cyst or internal hordeolum)*. A 'stye' or *hordeolum* can also result from acute infection of a gland of Moll, Zeiss or the eyelash follicle. If this is the case it is called an external *hardeolum*.

Lacrimal apparatus

The lacrimal apparatus consists of **lacrimal glands, lacrimal ducts** and **lacrimal canaliculi** (**C**).

The gland, which secretes one of the components of tears, lies in a fossa on the upper lateral part of the orbit. The lacrimal fluid entering the conjunctival sac through the lacrimal ducts passes into the lacrimal lake at the medial angle of the eye from which it drains to the lacrimal sac (**S**). The fluid passes to the nasal cavity through the **nasolacrimal duct** (**N** which opens into the nasal cavity (into the inferior meatus – I) from which it passes into the nasopharynx and swallowed. As there are no anastamotic pathways for drainage of tears, any obstruction to this system will give rise to an overflow of tears in the eye, *epiphora*. Further, stagnant tears in the sac may predispose to infection.

Tear film

Tears 'wet' the ocular surface and a tear film is regenerated every time blinking occurs. We need tears to provide a smooth ocular surface for light rays to be refracted uniformly and also provide lubrication to prevent friction between ocular surfaces during lid closure. In addition, the tears possess antibacterial properties.

The tear film has 3 components:

Surface lipid layer – secreted by the meibomian (tarsal) glands

Middle aqueous layer – secreted by the lacrimal gland and accessory lacrimal glands

Inner mucus layer – secreted by goblet cells of the conjunctiva and the epithelial cell surface

Dry eyes

The tear film prevents the ocular surface from 'drying out'. If this were to occur, the epithelial surface of the cornea and conjunctiva would become damaged. The patient would experience grittiness and discomfort of the eye.

The lipid layer of the tear film plays a very important role. This layer prevents evaporation of the underlying aqueous component. Any deficiency of the tarsal glands e.g. obstruction to their openings, would lead to increased evaporation of tears. Subsequently, the eye becomes 'dry' and there is a risk of damage to the ocular surface and predisposition to infection.

Another way in which 'dry eyes' can occur is that of reduced production of tears. In this case it is important to review the patient for any suggestions of autoimmune disease such as Sjogren's syndrome, rheumatoid arthritis and others. Also, it is important to assess their medications to determine if these could be contributing to reduced tear production e.g. antihistamines.

Indirect trauma or injury that displaces orbital contents is called a "blowout fracture".

Injury to the facial nerve may result in paralysis of the orbicularis oculi muscle. This prevents the eyelids from closing fully and loss of protective blinking of the eye. As a consequence of this, the cornea becomes dry and is left unprotected from dust and other small particulate material. Irritation of the eyeball results in excessive tear formation and failure of blink results in an ineffective lacrimal pump..

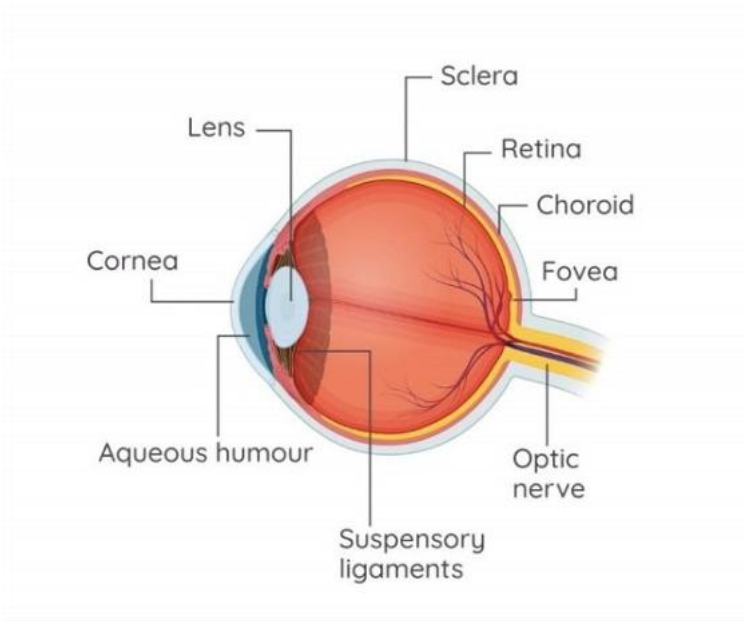
If facial palsy is combined with reduced corneal sensation, the risk of sight threatening corneal ulceration is high.

The Eyeball

The eyeball has three layers. The outer protective layer, which comprises the sclera and the cornea, is fibrous and provides attachment for the extraocular muscles. The middle coat comprises the choroid, ciliary body and iris. It has a rich network of blood vessels. The inner layer is the retina consisting of optic and non-visual parts.

The sclera forms the bulk of the fibrous layer of the eyeball, its anterior part visible through the conjunctiva as the "white of the eye". The cornea is the transparent part of the fibrous coat. The choroid, which is a dark membrane between the sclera and the retina, forms the largest vascular layer of the eyeball and terminates anteriorly as the ciliary body. The ciliary body (which is muscular as well as vascular) connects the choroid with the iris.

The eyeball has two chambers. The **anterior chamber** is the space between the cornea and the iris. The **posterior chamber** is the space between the iris and the ciliary body and the **lens.** The ciliary body secretes the **aqueous humour that** fills the chambers of the eye.



https://www.studyclix.ie/leaving-certificate/biology/higher/eye-and-ear/the-eye/boost/structure-and-functions-of-the-eye

The iris, which lies on the anterior surface of the lens, is a thin contractile diaphragm with a central aperture (the **pupil**) for transmission of light; two muscles (sphincter and dilator pupillae) control the size of the pupil. The lens, which lies posterior to the iris, is a transparent biconvex structure enclosed in a capsule and is attached to the ciliary body by the suspensory ligaments. Contraction of the muscle fibres in the ciliary body changes the shape of the lens.

The cavity behind the lens is filled by **vitreous humour** (a transparent jelly-like substance) that supports the lens and holds the retina in place. The retina, which receives the visual light, consists of a **neural layer** (light-receptive) and a **pigmented layer**. In the posterior part (called the **fundus**) of the eye is a circular depressed area, the **optic disc** where the **optic nerve** enters the eyeball. The optic disc contains nerve fibres and no photoreceptor cells. Just lateral to the optic disc lies a small area (macula lutea) of the retina with photoreceptor cells specialised for highest acuity of vision.

The **fovea centralis,** which is a depression in the centre of the macula, is the area of most acute vision. The retina is supplied by the central artery of the retina and drained by the corresponding vein.

In some diseases/disorders (eg tumours in the orbit) the eyeball protrudes, a condition known as proptosis. In some conditions (eg thyroid eye disease) the lids are pulled wider open; this is called lid retraction and can occur with or without proptosis.

Dirt and other extraneous particles can cause corneal abrasions that result in sudden pain and excessive tears. Sharp objects may cause lacerations of the cornea. Injury to the sensory nerve supply (from the ophthalmic division of the trigeminal nerve (5th cranial nerve) to the cornea renders it vulnerable to foreign particles.

During old age, the lens becomes harder and more flattened and these changes slowly reduce the focusing capacity (presbyopia). Some elderly people develop partial or complete opacity of the lens (cataract).

Blunt trauma to the eyeball may result in haemorrhage into the anterior chamber of the eye (hyphema); a large accumulation of blood in the chamber can cause high intra-ocular pressure and staining of the cornea.

Since the optic nerve is surrounded by meninges with CSF in the subarachnoid space, increase in the CSF pressure may compress the optic nerve. This in turn compomises the blood vessels supplying the retina, the vein being occluded before the artery. Slow venous return causes oedema of the retina. The normally depressed optic disc then forms a papilla (papillodema). This is easily seen during ophthalmoscopy. Continued compression of the optic nerve in such conditions may lead to visual impairment.

During embryonic development, the layers of the retina are separated and fuse during the foetal period. Although the pigmented layer is firmly fixed with the choroid, its attachment to the neural layer is not firm. A tear in the retina may allow fluid between the two layers causing a detached retina.

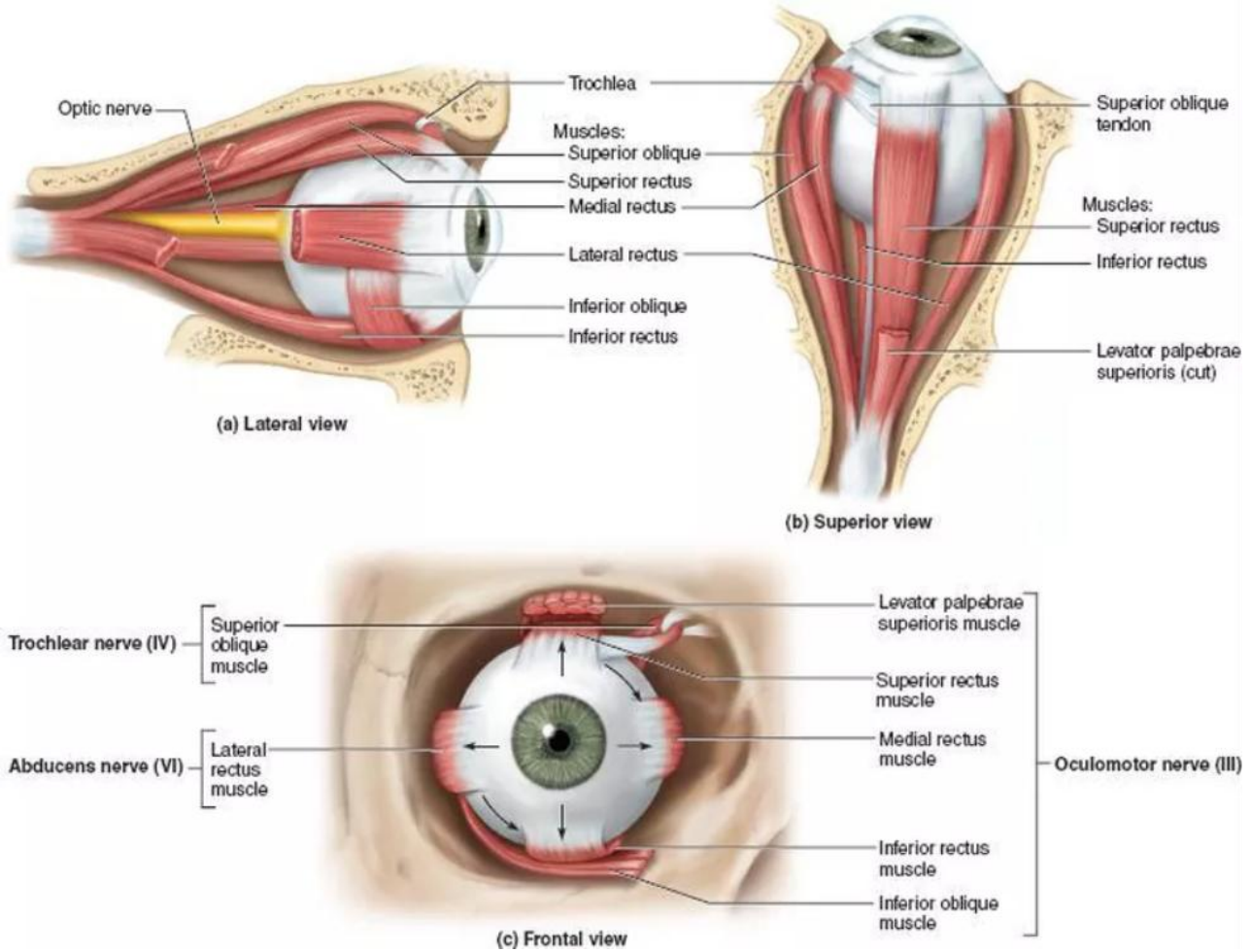
The Ocular Muscles, Nerves and Vessels

The muscles of the orbit are the **levator palpebrae superioris (LPS)** and four **recti** (superior - **SR**, inferior, lateral and medial) and two **oblique** (superior - **SO** and inferior). The muscles act together to move the eyelids and the eyes and are supplied by the **oculomotor, trochlear (TN)** and **abducent** (3rd, 4th, & 6th cranial nerves respectively) **nerves**. The recti muscles arise from a fibrous cuff, the **common tendinous ring**, that surrounds the optic canal and attach to the sclera on the anterior half of the eyeball. The oblique muscles work synergistically with the recti.

Complete oculomotor palsy affects most of the ocular muscles, the levator palpebrae superioris and the sphincter pupillae. The superior eyelid droops (ptosis) and cannot be raised voluntarily because of the unopposed orbicularis oculi supplied by the facial nerve.
One or more ocular muscles may be paralysed by head injury or brainstem disease affecting the nerves (CN 3,4 or 6), resulting in double vision (diplopia).
Blockage by emboli of branches of the central artery of the retina results in sudden painless partial or complete loss of vision in the area supplied beyond the blockage.
Central retinal vein occlusion presents with slower onset variable extent of visual loss.

In addition to the **optic nerve** (**ON** – 2nd cranial nerve), several branches (i.e. lacrimal, frontal, nasociliary and ciliary nerves) of the **ophthalmic division of the trigeminal nerve** (5th cranial nerve) supply structures in the orbit.

The arteries supplying the orbit are mainly from the **ophthalmic artery** which gives off the **central artery to the retina**. The veins of the orbit are tributaries of the **ophthalmic veins** that drain into the cavernous venous sinus lying within the cranial cavity.



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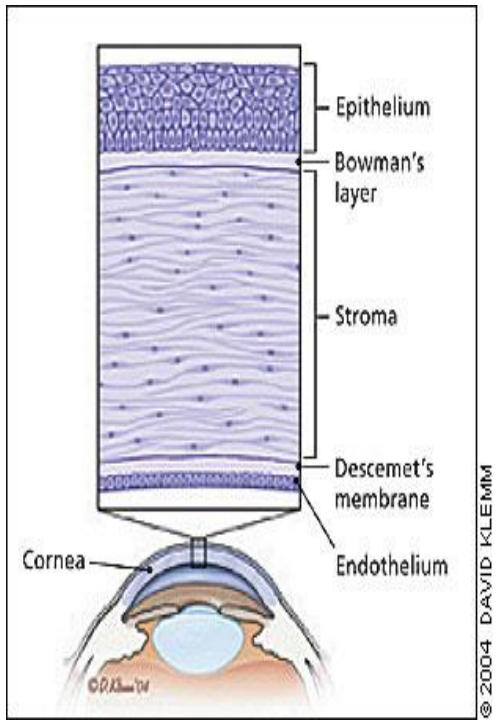
The Cornea, Intra Ocular Pressure and the Uveal Tract



Cornea

The cornea is a transparent, avascular structure which separates the anterior chamber posteriorly and the tear film anteriorly. It has 3 main roles:

1. Maintaining transparency (so that we have a clear window through which to see)
2. Ocular protection (including the corneal reflex)
3. Refraction of incoming light (along with the overlying tear film)

The cornea is divided into 5 layers: Epithelium, Bowman's membrane, Stroma, Descemet's membrane and Endothelium. We shall briefly discuss the roles of the above structures in achieving the functions of the cornea below.

Epithelium

This is a non-keratinized stratified squamous layer approximately 5-7 cell layers thick. The cells migrate from the basal layer to the surface and are eventually sloughed off into the tear film. When the epithelium is damaged cells must migrate from the basal layer to cover the wound. Cells also migrate from the periphery to the centre of the cornea. When the corneal epithelium is damaged, cells on the surface spread out to cover the defect before an increase in mitotic activity at the basal layer replenishes the cellular deficit. The total time for regeneration of the corneal epithelium is quoted as between 3 to 14 days, being longer for central epithelial defects. Corneal stem cells are also located at the periphery of the cornea at the 'limbus'; also explaining why central corneal wounds heal more slowly than more peripherally located defects.

Bowman's Layer

This is an acellular layer and separates the overlying epithelium from the underlying stroma. It is important to recognise as any trauma extending below this level will result in corneal scarring as only the epithelium is regenerated.

Stroma

This connective tissue layer forms the bulk of the cornea. It consists of an arrangement of regularly orientated collagen fibres (type 1 predominantly). The regularity with which these are arranged helps to explain the transparency of the cornea. Keratinocytes (fibroblasts) are found in-between these collagen fibres and help tether them together as well as other functions. Corneal oedema is associated with visible lines in the stroma (striae). These are visible corneal stromal lamellae.

Descemet's membrane

This separates the corneal stroma anteriorly from the endothelium posteriorly. It acts as the basement membrane to the endothelium.

Endothelium

The main role of the endothelium is to maintain hydration in order for the cornea to remain transparent. It is a layer of simple squamous endothelium that does not regenerate. Any cellular loss is replaced by changes in shape and size of adjacent cells to cover the deficit. At birth we have 3000-4000 cells/mm^2 and this steadily falls to 2000 cells/mm^2 in old age. A level of less than 800 cells/mm2 leads to corneal oedema and poor vision. Cellular loss may be accelerated by trauma, surgery and exposure to UV radiation.

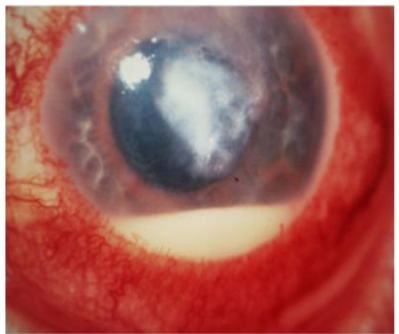
Corneal innervations

The cornea has a rich nerve supply derived mainly from the ophthalmic branch of the trigeminal nerve (via long ciliary nerves). In some case there is innervation via the maxillary branch which supplies the inferior cornea.

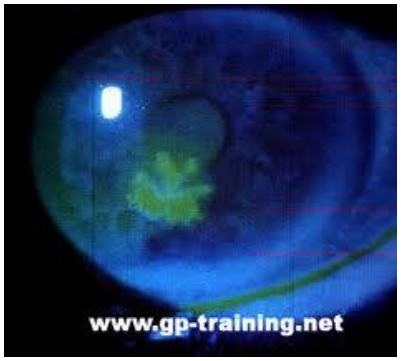
Infective keratitis (kera – cornea)

For infection to occur there is normally the need for disruption to the epithelial surface. Potential triggers for this include contact lens wear, trauma, 'dry eyes', and pre-existing corneal disease amongst others. Also, systemic conditions leading to an immunocompromised state will make infection more likely.

A loss of epithelium exposes free nerve endings causing severe pain. The inflammation also leads to increased circumcorneal vascularity and a red eye. There is associated excess tear production. As the corneal surface and tear film is disrupted, along with possible corneal oedema, there is a resultant drop in visual acuity. There may also be photophobia and a mucus or mucopurulent discharge. Examination may reveal a white deposit in the cornea (corneal infiltrate) and a collection of pus behind the cornea in the anterior chamber (hypopyon). Looking at the anterior chamber more closely may reveal 'cells' and 'flare' which are leucocytes and protein respectively derived from the leaky iris blood vessels responding to the infection.



a) Image illustrating hypopyon



b) Image illustrating corneal epithelium staining

Aqueous humour and Intraocular Pressure (IOP)

Aqueous humour is responsible for maintaining an adequate pressure in the eye. IOP is a balance between the rate of production of aqueous and the rate of drainage.

Aqueous is produced by the ciliary processes of the ciliary body. It then flows up between the iris and anterior surface of the lens and through the pupil. After passing through the pupil it flows out through the drainage angle of the eye, between the cornea and the peripheral iris, through the trabecular meshwork then into the Canal of Schlemm. Aqueous then drains into the episcleral vessels and finally into the systemic venous circulation. However, about 10-20% of aqueous is drained via an 'unconventional' or 'uveoscleral' route. Here, instead of aqueous passing into the trabecular meshwork it passes into the root of the iris and/or ciliary muscle before draining into the scleral vascular system. This is important as it has pharmacological implications as we shall see below.

IOP is calculated by measuring the force needed to flatten a pre-determined area of the corneal surface using a *tonometer*. The greater the force needed to flatten the cornea, the higher the pressure. The 'normal' range of IOP is 11-21 mmHg. Above 21 is considered to be ocular hypertension. (This measurement does have a correction factor – the corneal thickness – ask your ophthalmologist about this whilst in clinics during your placement.) It is important to note that having high intra-ocular pressure does not equate to a diagnosis of glaucoma. Indeed, a diagnosis of glaucoma can be made with normal IOP. However, high IOP is a significant risk factor for the development of glaucoma.

Autonomic control of IOP

This is via adrenergic receptors. Cholinergic mechanisms have little direct effect on aqueous production.

Alpha 2 receptors – stimulation reduces IOP by reducing aqueous production and may increase uveoscleral outflow

Beta 2 receptors – stimulation increases IOP by increasing aqueous production

Drugs targeting IOP

Once a decision has been reached to lower IOP we can choose from several drugs in order to achieve this. Ideally we would choose a drug that can be used at its lowest concentration and frequency of application has minimal side effects and produces the greatest therapeutic effect. We shall briefly discuss some potential topically used medications below:

CLASS	EXAMPLES AND REGIMEN	MECHANISM OF ACTION
Beta blockers	Timolol, Carteolol OD-BD	Reduce production of aqueous (see above)
Alpha agonists	Apraclonidine, Brimonidine BD	Reduce production and small increase in drainage of aqueous
Prostaglandin analogues	Latanoprost, Bimataprost ON	Increase uveoscleral outflow of aqueous
Carbonic Anhydrase Inhibitors	Dorzolamide, Brinzolamide TDS	Reduces production of aqueous
Parasympathomimetic	Pilocarpine QDS (when used as monotherapy)	Increases outflow of aqueous by ciliary muscle contraction opening trabecular meshwork

Topical preparations can also be combined, for example, Cosopt – (timolol +dorzolamide).

Lens

The crystalline lens is positioned posterior to the iris and anterior to the vitreous which help to maintain its position suspended by the zonules. The zonular fibres (suspensory ligaments) arise from the ciliary body and attach to the surface of the lens.

The lens helps to refract light and although its refractive power is less than that of the cornea; the lens can vary the amount it refracts light by changing its shape – *accommodation*. We require increased accommodation for near tasks. The lens sits in a capsule – a thickened basement membrane that completely envelopes the lens. As we age the degree to which the lens is able to accommodate diminishes - a process known as *presbyopia*. The elasticity of the lens together with atrophy of the ciliary muscle contributes to this. We can overcome this drop in accommodative ability by prescribing convex lenses to aid patients with their near work. This is most notable from the age of 40-45 onwards.

The lens is covered with epithelium and the epithelial cells undergo mitosis and eventually form lens fibres. As new fibres are laid down subcapsularly the lens continues to grow throughout life, with the newest layers on the surface and the older layers moving progressively deeper. This also results in the outer lens cortex being soft whereas the central nucleus of the mature lens is very hard. Lens fibres are tightly packed and the arrangement is highly organised – helping the lens to maintain its transparency.

Cataract

Any opacity (loss of transparency) of the lens is known as cataract and may result from disruption to the lens fibre configuration, capsule or epithelium. The commonest cause for cataract is increasing age. However, cataract can occur in the newborn and it is vital that this is detected as early as possible and referral made to a specialist; in order to enable the best possible vision to be obtained from the affected eye.

Other causes for cataract in younger patients include:

Trauma - direct injury to lens (penetrating), electric shock/lightning, blunt trauma

Drugs – steroids (systemic and topical), amiodarone, phenothiazines

Systemic disease - diabetes mellitus, myotonic dystrophy, neurofibromatosis type 2

Morphological types of cataract and symptoms

Along with general symptoms of reduced vision and difficulty with everyday tasks certain cataracts are associated with more specific changes:

NAME	LOCATION	PATIENT SYMPTOMS
Subcapsular	Directly under the lens capsule. Granular or plaque like appearance.	Near vision affected more than distance as opacity at nodal point of eye.
Nuclear	Involving lens nucleus. Yellowish-brown due to deposition of urochrome pigment.	Patient becomes myopic due to increase refractive index of lens. Colours appear less well saturated and more yellow/brown
Cortical	Cortex of lens. Radial spokes in the periphery.	Give rise to astigmatic changes. Patients troubled more in dark when pupil dilates and exposes more of cataract.

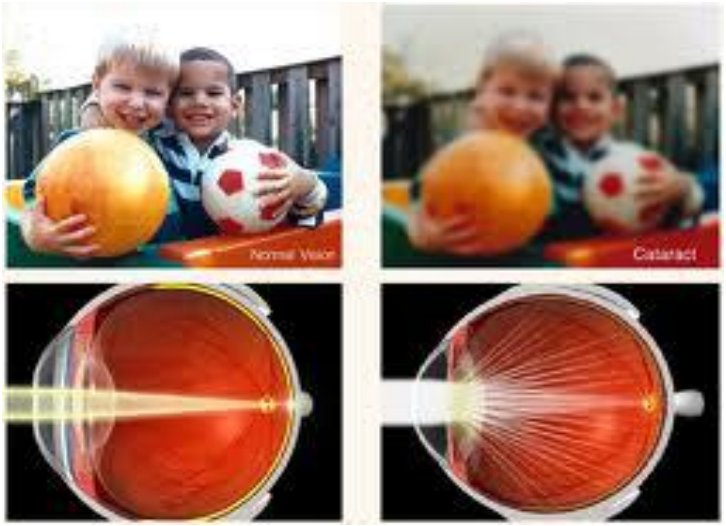


Image illustrating a) Normal vision vs What patients see through a cataract, b) Normal passage of light to the retina, b) Light passing through an opacified lens to the retina

Cataract surgery – Phacoemulsification

During this placement you will have the opportunity to see patients with cataracts. It is important that you try to gain an understanding of this patient journey.

Phacoemulsification is the most common technique for cataract surgery in the United Kingdom. It is usually performed under local anaesthetic and the patient must be prone. Entry to the eye is made via the limbus (peripheral cornea) using a self-sealing incision. A circular incision is made in the anterior lens capsule – *capsulorrhexis*. The cataractous lens is removed using ultrasound and replaced with a plastic lens calculated to correct the patients' refractive error. This is an over-simplification but will hopefully help you when visiting ophthalmic theatres!

The Uveal Tract

The uveal tract consists of the Iris, Ciliary body and the Choroid. These structures are pigmented and vascular. We shall briefly discuss the relevant parts of their anatomy below.

Iris

The iris is a pigmented structure and divides the eye into the anterior and posterior chambers. The iris is anterior to the lens and is attached by the root of the iris at the *irido-corneal* angle where it merges with the ciliary body and trabecular meshwork.

The iris controls the amount of light entering the eye by varying the size of the aperture in the middle of the iris – the pupil. In conditions of low light or sympathetic activation the pupil dilates. This is called *mydriasis* and allows more light into the eye. The dilator pupillae muscle allows the pupil to dilate. It is orientated radially and therefore contraction pulls the pupil outwards and enlarges the pupil diameter.

In conditions of bright light the pupil constricts. This is called *miosis*. The sphincter pupillae allows the pupil to constrict. It encircles the pupillary margin and thus contraction will bring the edges of the pupil closer together resulting in a narrowing of the pupil diameter. It is stimulated via parasympathetic activation, the fibres of which are carried in the inferior division of the 3rd nerve.

Armed with the above knowledge it should now be simple to determine the effects of the following drugs on the pupil:

Tropicamide – antimuscarinic – mydriasis

Phenylephrine – sympathomimetic – mydriasis

Pilocarpine – muscarinic agonist – miosis

With age the dilator pupillae becomes atrophic and the sphincter papillae more fibrotic, resulting in relative miosis of the pupil in the elderly.

Ciliary Body

The ciliary body is also a pigmented structure and its primary roles are those of accommodation, aqueous humour production and providing attachment for the lens zonules. The ciliary muscle helps to control accommodation by way of the attached zonules (suspensory ligaments) (see section on lens) which travel up and attach to the equator of the lens. When the ciliary muscle contracts it moves forwards and inwards, thus slackening the tension on the zonules and increasing the power of accommodation.

The ciliary processes are found on the anterior part of the ciliary body. Aqueous humour is produced by the ciliary processes and secreted out into the posterior chamber (see section on Aqueous humour and IOP).

Choroid

The choroid is found lining the posterior part of the eye, extending from the ciliary body to the optic nerve lying between the retina and the sclera. As with the iris and ciliary body it too is a pigmented structure. The choroid has several important functions:

1. Allows nerves and vessels to reach the anterior eye by passing through the choroid
2. Removes waste product from the outer retina (area closest to choroid)
3. Supplies essential nutrients to the outer half of the retina
4. Absorbs any light passing through the retina, thus preventing it from reflecting back and interfering with vision

The choroid attaches to the retina by way of a basement membrane known as Bruch's membrane. This is important in the pathophysiology of Age Related Macular Degeneration. Moving outwards we then come to the rich capillary bed of the choroid – *choriocapillaris*. It is this layer that supplies the outer half of the retina.

Uveitis

Having defined the uveal tract above it should now be easy to define uveitis – inflammation of the uveal tract. We can anatomically classify uveitis using the following terms:

Anterior uveitis - inflammation affecting the iris (*iritis)* +/- the ciliary body (*iridocyclitis*)

Intermediate uveitis - inflammation of the posterior part of the ciliary body and nearby peripheral retina and choroid

Posterior uveitis – inflammation of the retina and choroid

Panuveitis – inflammation of the whole uveal tract

Anterior uveitis

The patient complains of photophobia, pain, reduced vision and a watery eye. Examination reveals circum-corneal injection and sometimes cellular deposits on the corneal endothelium (keratic precipitates; see picture above). Within the anterior chamber there are cells and flare (protein), derived from leaky iris blood vessels. In the subacute or recurrent cases there may be posterior synechiae – adhesions between the inflamed iris and anterior lens capsule. This is important because if the adhesions became significant they would obstruct the passage of aqueous humour which flows up between the iris and anterior lens capsule. We therefore prescribe antimuscarinic eye drops (cyclopentalate) to prevent posterior synechiae from occurring. These drops also help with pain derived from spasm of the ciliary muscle as they paralyse the ciliary muscle – *cycloplegia*. Topical steroids are then used to dampen down the inflammation.

Illustration of keratic precipitates in anterior uveitis

Vitreous

The vitreous cavity fills 2/3 of the volume of the eye. It is attached to the posterior lens capsule, and to the retina, at several points, posteriorly. It holds the retina in place and supports the lens. It is approximately 99% water and is 3 times more viscous than water. If, during cataract surgery, the posterior lens capsule is disrupted, it will lead to disruption of the anterior membrane of the vitreous allowing flow of vitreous into the anterior chamber, increasing the risk of retinal detachment. The vitreous degenerates and contracts with age, fluid filling the potential space between the retina and vitreous. The vitreous may then detach from the retina where it is less weakly bound. A vitreous detachment may predispose to a retinal detachment, especially in areas where vitreous attachments to the retina are firmer just behind the ciliary body.

Retina

The retina is the innermost layer of the eyeball. It has two parts derived embryologically from the optic cup. The first is the inner neural retina and the second is the outer retinal pigment epithelium (RPE). There is a potential space between these two layers - *subretinal space.* The subretinal space between the neural and RPE retina 'fuses' during early foetal life. The RPE is firmly attached to Bruch's membrane (see choroid above). The neural retina is only attached firmly at the optic nerve head and anteriorly at its termination the *ora serrata*. Trauma and other mechanisms can lead to this separation reforming – *retinal detachment.* Patients will complain of flashing lights and floaters as well as possible visual field defects due to disruption of afferent nervous pathways.

During your time in this placement you may encounter various terms for parts of the retina. It is important that you familiarise yourselves with the following terms – posterior pole, macula/fovea, optic disc and peripheral retina.

Neural Retina

The neural retina is concerned with receiving light stimulus and transforming this into nervous impulses. These impulses travel along ganglion cell axons in the optic nerve, eventually reaching the visual cortex, where they are processed.

There are several neural cell types in the inner retina. However, important cells which we shall discuss briefly are photoreceptors – namely rods and cones. Rods are responsible for sensing contrast and motion. Rods are not good for detail, 'see in black-and-white' and are also assist seeing in darker environments. Cones sense fine detail and colour vision. Disruption to photoreceptor physiology will lead to specific visual problems. You may wish to read about rod-cone-dystrophies and colour vision anomalies to find out more.

Blood supply

The retina is highly metabolically active and thus requires a good blood supply to meet these demands. The inner 2/3 of the retina is supplied by the central retinal artery whilst the outer 1/3 is supplied by the choroidal blood supply. The central retinal artery divides into 4 and each supplies a segment of the retina. These arteries are end-arteries. Retinal arteries overlie retinal veins and in certain situations eg. Hypertension, characteristic changes (nipping) can be seen where these vessels cross. The superior and inferior temporal retinal arteries arch over the posterior pole.

It is important to note that the macula has a very dense capillary network, except at the fovea – the *capillary free zone*. This region is dependent on the underlying choriocapillaris.

Retinal Artery Occlusion

From the above we know that retinal arteries are end-arteries. Therefore, if there is obstruction to an artery there will be insufficient anastomotic supply for tissue survival. In a branch or central retinal artery occlusion the retina becomes pale and oedematous. Changes are irreversible within 1-2 hours. However, in a central retinal artery occlusion the fovea retains a red colour due to being supplied by the choriocapilaris. Patients will complain of painless and sudden loss of vision. Eventually, the inner retina suffers irreversible ischaemia and patients have a persisting visual defect.

Visual pathway

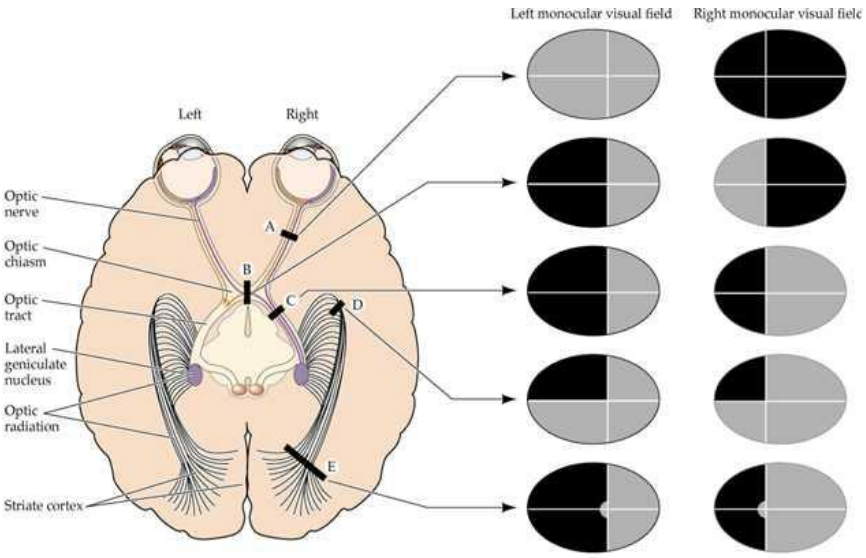
From elemental physics we note that light travels in a straight line. Therefore, light that strikes the temporal retina must come from the nasal visual field and the converse applies for the nasal retina. This simple rule will help when faced with visual field defects and determining locations of lesions causing them, or vice versa.

The temporal and nasal fibres come together at the optic nerve and travel as one to the optic chiasm. Here, the nasal retinal fibres (temporal visual field carrying fibres) decussate (cross over). This means that the right optic tract will carry temporal retinal fibres from the right eye and nasal retinal fibres from the left eye. This puts all the fibres carrying information from one hemi-field together. For the right optic tract this would mean carrying all information from the left half of the visual field. The remainder of the visual pathway maintains this hemi-field organisation of fibres. The optic tract synapses at the

Lateral Geniculate Nucleus (LGN). After the LGN the optic radiations arch through the temporal and parietal lobes and terminate at the occipital cortex, (visual cortex). Optic radiations passing through the parietal lobe carry superior retinal fibres from one hemi-field of vision. For example, the right parietal lobe carries superior temporal fibres from the right eye and superior nasal fibres from the left eye. These fibres carry information from the inferior left visual field. Fibres travelling through the temporal lobe are the inferior retinal fibres and would carry information from the superior visual field respectively.

It is important to note that after decussation at the optic chiasm fibres represent one hemi-field of vision. Therefore, any lesion posterior to the chiasm will not, by definition, cross the vertical midline of the visual field. See if you can prove this to yourself! See overleaf.

Imaging showing the visual pathway and possible patterns of visual field loss, *taken from: http://www.utdallas.edu/~tres/integ/sen4/8_04.jpg*



Pupil reflexes

Afferent pupillary fibres travel with the rest of the retinal fibres but leave the visual pathway just before the LGN. After leaving the visual pathway they synapse with the pretectal nucleus. From here, fibres travel to BOTH Edinger-Westphall nuclei (3rd nerve nucleus). This is important to note as it demonstrates why stimulation of pupillary afferent fibres in one eye causes a bilateral motor pupillary response. Efferent fibres leave the 3rd nerve nucleus, travel to the ciliary ganglion (via the inferior division of the oculomotor nerve) and finally to the sphincter pupillae, causing pupillary constriction.

RAPD (Relative afferent pupillary defect)

Stimulation of the right afferent pupillary fibres will cause bilateral pupillary constriction. If the right optic nerve (RON) is completely severed we would have no afferent conduction from the right eye. This would mean we get no direct or consensual response from shining a light in the right eye. A consensual pupillary response would remain in the right eye when light is shone into the left eye. This occurs because the efferent system is still intact and can still be innervated by afferent fibres from the left eye innervating both right and left 3rd nerve nuclei. However, what would happen if the RON was damaged incompletely? We would get a partial pupillary response when light is shone into the right eye (as there is a reduction in afferent innervation) and a normal right pupillary response when light is shown in the left eye (as the right eye efferent system is intact). This may be subtle and difficult to determine and we can exaggerate this response by swinging the light from the right eye to left eye and back again several times, whilst observing the pupil we are shining light into. If both afferent arms of the pathway are intact we would expect the constriction response to be equal and maintained as we swing our light. If the RON is damaged incompletely and we present the light from the left eye to the right eye, the right eye would not constrict but rather dilate!

This is a right RAPD. This is because the afferent information from the left eye which no longer has light shone into it is demanding dilatation rather than constriction. The damaged afferent system of the right eye (into which we are shining light) is overpowered by the intact afferent system of the left eye and instead of constricting, the eye dilates.

Assessment of patients with ocular disease

Taking the History from a Patient with an Eye Problem

Identify the onset and duration of visual symptoms and whether visual function has an impact on the patient's lifestyle.

Visual loss: if present, distinguish between bilateral or unilateral, central or peripheral visual loss and acute or chronic

Discomfort: a foreign body sensation indicates corneal irritation e.g. corneal foreign body, foreign body under the upper lid, corneal abrasion, corneal ulcer, dry eye. Aching suggests a more posterior problem

Double vision: enquire if monocular ie the double image is still there if the unaffected eye is covered (often a rather non-specific symptom), or binocular diplopia
(indicates misalignment of the eyes: 1 image always disappears if one eye is covered)

Photopsia i.e. the sensation of localisable flashing light in 1 eye is an indicator of retinal traction in the affected eye.

The ability to localise an abnormal visual sensation suggests retinal pathology e.g. diabetic retinopathy, age related macular degeneration, retinal detachment etc etc.

Performing an Eye Examination

Check the visual acuity

Check the visual fields with the confrontation test

Observe both eyes, looking specifically for discharge, ptosis and lid lesions.

Examine the corneal reflexes and carry out a cover test to detect any misalignment of the eyes. Check the eye movements in all positions of gaze.

Examine the pupils and in particular look for any opacity within the normally black pupil, look for any irregularity or distortion of the pupil that could be caused by damage to the pupil or adhesions between the iris and the lens, and check for a relative afferent pupillary defect (RAPD).

Examine each eye with a pen torch looking for generalised or circumcorneal redness, corneal opacity such as a foreign body or abscess, any fluid level of blood or pus in the anterior chamber, and any distortion or irregularity of the pupil.

Use a direct ophthalmoscope to examine the red reflex in each eye to check opacities in the ocular media such as those caused by corneal oedema or other opacity, cataract, or vitreous haemorrhage.

Examine the optic disc, macula and retina with the direct ophthalmoscope, taking care to document both normal and abnormal findings (this is easier when the pupils are dilated).

Management of Eye Disease

Investigations

Visual field testing: An initial assessment of the visual fields can be done by confrontation testing. Precise delineation of the visual fields is done with a perimeter. Modern automated perimeters have statistical methods to monitor deterioration in the visual field over a series of visits and to give an estimate of the reliability of the results. You should be able to distinguish between a field defect suggestive of an ocular problem and one suggestive of a neurological chiasmal or post-chiasmal problem.

OCT- Ocular coherence tomography- uses a scanning laser ophthalmoscopy to demonstrate the retina and optic nerve at cellular level. It is essential in the management of macular disease and imaging the optic disc in glaucoma.

Fluorescein angiography involves intravenous injection of fluorescein which delineates the retinal vessels. A photographic record is made of the distribution of the dye in the retinal vessels, including any leakage from the retinal vessels over a 15 minute period.

Orthoptic assessment involves detailed measurement of the angle of deviation of the eyes in different directions of gaze as well as assessment of binocular function and the assessment of visual acuity in small children.

Ultrasound, CT and MRI are used to image the eye and orbit.

Ophthalmic Conditions and their Treatment

External eye and acute disorders of the anterior segment of the eye.

You should be familiar with the common causes of a red eye. By the end of the course you should be able to recognise conjunctivitis, corneal abrasion, corneal foreign body, keratitis, iritis, and orbital cellulitis. You should particularly know about common eye disorders associated with atopic disease, thyroid disease, urogenital disease, ENT disease and rheumatoid disease. You should know about the treatment of common conditions causing a lump on the eyelid. You should be able to treat simple cases of external eye disease and know when it is appropriate to refer.

Chronic visual loss

You should be able to distinguish the characteristic visual disabilities in patients with glaucoma, cataract, age related macular degeneration, retinitis pigmentosa and hemianopia from cerebrovascular vascular disease. You should understand the distinction between painful acute angle closure glaucoma and chronic simple glaucoma. You should be aware of the systemic side effects of some eye drops for chronic simple glaucoma.

Cataract

You should be able to detect cataract and be able to detect an intraocular lens with a pen torch. You should be aware of the relationship between cataract and ageing and diabetes. You should have some idea of the level of vision at which cataract surgery may be considered and of appropriate expectations from cataract surgery. You should be able to detect congenital cataract and understand the importance of early referral of congenital cataract for the prevention of blindness in childhood.

Diabetic Retinopathy and Age Related Macular Degeneration

You should be able to recognise diabetic retinopathy and understand the implications of your clinical findings. You should have a broad understanding of factors that may contribute to the development of diabetic retinopathy, the scope of treatment and the significance of diabetic retinopathy as a cause of preventable blindness in the UK. You should be able to detect age related macular degeneration and understand in which patients there may be scope for treatment.

Paediatric ophthalmology

You should understand the causes and treatment of amblyopia, and how the commonest causes of treatable visual loss in children are detected and treated. You should read about the importance of red reflex testing in infancy and causes of an abnormal reflex.

Ophthalmic manifestations of genetic eye disease, neuro-ophthalmology, malignant disease, systemic inflammatory disease and rheumatoid disease

You should have some understanding of the ophthalmic features of the major genetic, neurological, malignant, and systemic inflammatory diseases which may present with eye signs. In particular you should appreciate the wider medical and genetic significance of retinoblastoma, retinitis pigmentosa, optic neuritis, amaurosis fugax, homonymous and bitemporal field defects, and inflammatory eye disease.

Epidemiology

By the end of the course you should have a broad understanding of the impact and causes visual loss has in the UK and the extent and cause of preventable blindness worldwide.

Mydriatic and Cyclopaedic Drops

Uses:

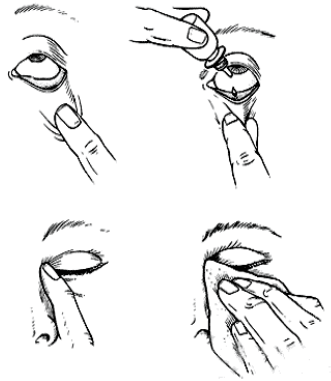
- ~ To dilate the pupil for visualisation of the retina
- ~ In the management of children with amblyopia
- ~ Use in refraction of children for the prescription of glasses

Types:

Name	Dose	Mechanism	Length of Action	Contraindications
Atropine	1-2 drops of 0.5% or 1% solution	Parasympatholytic- Anticholinergic that blocks the response of the iris sphincter muscles and the accommodative muscles of the cillary body	1-2 weeks	Hypertension Untreated Narrow Angle Glaucoma
Cyclopentolate	1-2 drops of 1% or 0.5% solution	Parasympatholytic- Anticholinergic that blocks the response of the iris sphincter muscles and the accommodative muscles of the ciliary body	Effect in 25-75 mins Recovery over 6 – 24 hrs	Untreated Narrow Angle Glaucoma Untreated anatomically narrow angle Allergy
Tropicamide	1-2 drops of 1% solution	Parasympatholytic- Anticholinergic that blocks the response of the iris and ciliary muscle.	Effect within 15-20 mins Recovery over 4-8 hrs	Allergy Untreated Narrow Angle Glaucoma
Phenylephirine	1-2 drops of 2.5% and 10% solution	Sympathetic agonist - Stimulation of the iris dilation muscle	3-6 hrs	Avoid in children Untreated Narrow Angle Glaucoma

Side Effects:

- ~ Whitening of the eyelids due to vasoconstriction, this will resolve as the drops wear off.
- ~ Atropine can cause redness of the face and a warm sensation to the touch, this is not dangerous but a lower dose should be considered.
- ~ All mydriatics will sting the eyes for a few seconds after instillation.
- ~ Warn the patient that they cannot drive until the blurring effect of the drops has worn off - this is very important.



Instillation:

- ~ Require: Bottle of drops, Tissues, (Assistant)
- ~ Preparation: Hand washing prior to instillation
- ~ Consent: Inform patient of what the procedure will involve, that they cannot drive until the effect has ceased, what the effects will be (dilation +- stinging sensation on application). Check for allergies and contraindications
- ~ Application: Ask the patient to look up and pull the inferior eyelid down to form a well, apply one drop from a vertical position. Allow the patient to blink, wipe away any excess
- ~ Documentation: Record the type of drops, which eye, how many drops and time of instillation. Time, date and sign.

Fluorescein Drops

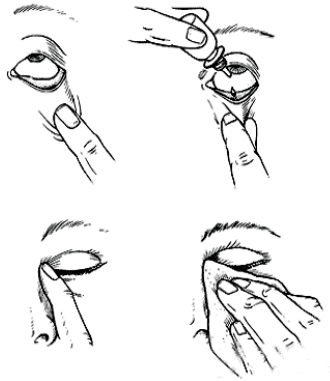
Uses:

- ~ Diagnostically to highlight defects in the corneal epithelium
- ~ Diagnostically to assess tear drainage in children with congenital nasolacrimal duct obstruction
- ~ Can be used as an investigation when measuring the intraocular pressure (tonometry)
- ~ Can be administered combined with a local anaesthetic as a 0.25% solution with oxybuprocaine HCl or proxymetacaine HCl

Name	**Dose**	**Mechanism**	**Length of Action**	**Contraindications**
Fluorescein	1-2 drops of 1% or 2% solution	Fluorescein, a precursor of the eosins, temporarily stains any cell it enters therefore marking any damaged areas.	Skin discolouration may last 6-12 hours	Allergy

Side Effects:

- ~ Warn patients about staining of the skin and clothes
- ~ If patient is wearing/will be putting in contact lenses warn that the Fluorescein may discolour them (do not replace soft contact lenses for at least 1 hour)



Instillation:

- ~ Require : Bottle of drops, Tissues, (Assistant)
- ~ Prepare: Wash hands
- ~ Consent: Inform patient of what the procedure will involve; what the effects will be (mild stinging sensation on application and temporary yellow/green discolouration of the eye). Check for allergies and contraindications. Ensure they are not wearing soft contact lenses
- ~ Application: Ask the patient to look up and pull the inferior eyelid down to form a well, apply one drop from a vertical position. Allow the patient to blink, wipe away any excess.
- ~ Documentation: Record the type of drops, which eye, how many drops and time of instillation. Time, date and sign.

List of important ophthalmological presentations and conditions

The workbook does not cover all conditions within ophthalmology. Below is a list of eye diseases or symptoms related to ocular disease which you should attempt to read using ophthalmology references which are listed in the Phase 2 reading list.

Please note: the columns below are not linked. Each column is simply a separate list of presentations or conditions you should be familiar with.

Presentations	**Conditions**
Acute loss of vision	Acute glaucoma
Allergies	Benign eyelid disorders
Diplopia	Blepharitis
Eye pain/discomfort	Cataracts
Eye trauma	Central retinal arterial occlusion
Facial/periorbital swelling	Chronic glaucoma
Flashes and floaters in visual fields	Conjunctivitis
Foreign body in eye	Diabetic eye disease
Gradual change in or loss of vision	Infective keratitis
Loss of red reflex	Iritis
Red eye	Macular degeneration
Squint	Optic neuritis
Visual hallucinations	Periorbital and orbital cellulitis
	Retinal detachment
	Scleritis
	Thyroid eye disease
	Uveitis
	Visual field defects

Vocabulary of Terms Relating to the Eye (for reference)

Accommodation	The adjustment of the eye for seeing at different distances, accomplished by changing the shape of the lens through action of the ciliary muscle, thus focusing a clear image on the retina.
Agnosia	Inability to recognise common objects despite an intact visual apparatus.
Albinism	A hereditary deficiency of pigment which affects the eye by causing failure of pigmentation of the retina, iris and choroid. Many different mutations in a number of different genes may cause albinism. In X linked ocular albinism the eye is the only organ affected to a significant extent.
Amaurosis Fugax	Transient recurrent unilateral loss of vision, commonly caused by embolic occlusion of the central retinal artery.
Amblyopia	Reduced vision due to disuse of the eye in childhood with no demonstrable organic defect.
Aniridia	Congenital absence of the iris.
Anophthalmos	Absence of a true eyeball.
Anterior Chamber	Space filled with aqueous bounded anteriorly by the cornea and posteriorly by the iris.
Aphakia	Absence of the lens.
Aqueous	Clear, watery fluid that fills the anterior and posterior chambers.
Astigmatism	Refractive error which prevents the light rays from coming to a single focus on the retina because the cornea has a greater curvature in one plane compared to another (i.e. the corneal curvature resembles the surface of a rugby ball).
Binocular Vision	Ability of the eyes to focus on one object and to fuse the two images into one.
Blepharitis	Inflammation of the eyelids.
Blepharospasm	Involuntary spasm of the lids.
Blindness	In the USA, the usual definition of blindness is corrected visual acuity of 6/60 (20/200) or less in the better eye, or a visual field of no more than 20 degrees in the better eye. In the UK blindness is defined as the level of visual impairment such as to prevent any work for which vision is essential. In a patient with normal visual field, an acuity of 3/60 or worse is generally taken to meet the criteria for registration as blind.

Blind Spot	'Blank' area in the visual field, corresponding to the light rays that come to a focus on the optic disc.
Buphthalmos	Large eyeball in infantile glaucoma.
Canaliculus	Small tear drainage tube at the medial end of the upper and lower lid leading from the puncta to the common canaliculus and then to the tear sac.
Canthus	The angle at either end of the eyelid aperture; specified as outer and inner or medial and lateral.
Cataract	A lens opacity.
Chalazion	Granulomatous inflammation of a meibomian gland of the eyelid.
Chemosis	Conjunctival swelling from any cause.
Choroid	The vascular middle coat between the retina and sclera.
Ciliary Body	Portion of the uveal tract between the iris and the choroid. It consists of ciliary processes and the ciliary muscle.
Coloboma	Congenital cleft due to the failure of some portion of the eye or lids to complete growth.
Colour Blindness	Diminished ability to perceive differences in colour.
Concave Lens	Lens having the power to diverge rays of light; also known as diverging, reducing, negative, myopic, or minus lens.
Cones and Rods	Retinal photoreceptor cells. Cones are concerned with visual acuity and colour discrimination; rods, with peripheral vision under decreased illumination.
Conjunctiva	Mucous membrane which lines the posterior aspect of the eyelids and the anterior sclera.
Convergence	The process of directing the visual axes of the eyes to a near point.
Convex Lens	Lens having power to converge rays of light and to bring them to a focus; also known as converging, magnifying, hyperopic, or plus lens, denoted by the sign (+).
Cornea	Transparent portion of the outer coat of the eyeball forming the anterior wall of the aqueous chamber.
Corneal Graft (keratoplasty)	Operation in which a section of opaque cornea is replaced with transparent cornea.

Cover Test	A method of determining the presence of misalignment of the eyes.
Crystalline Lens	The natural lens of the eye.
Cycloplegic	A drug that temporarily puts the ciliary muscle at rest, paralyses accommodation, and dilates the pupil.
Cylindrical Lens	A segment of a cylinder the refractive power of which varies in different meridians, used to correct astigmatism..
Dacryocystitis	Infection of the lacrimal sac.
Dioptre	Unit of measure of strength of refractive power of lenses or of prisms.
Diplopia	Double vision
Ectropion	Turning out of the eyelid so that the conjunctiva lining the inner surface of the lid is exposed to the exterior
Emmetropia	Absence of refractive error; thus an emmetropic individual does not need glasses unless they are old enough to need them for reading i.e. have presbyopia.
Endophthalmitis	Extensive intraocular infection.
Entropion	A turning inward of the eyelid, so that the eyelashes may come into contact with the cornea. This is a common and usually easily treated condition in the elderly in rich countries. In poor countries, Trachomatous conjunctivitis causes scarring and contraction of the conjunctiva lining the inner surface of the upper lid. and hence cicatricial entropion. This is a common cause of corneal damage and blindness in Africa.
Enucleation	Complete surgical removal of the eyeball.
Epiphora	Watering from the eye.
Esophoria	A tendency of the eyes to turn inward when binocular reflexes are disrupted by covering one eye (as in the alternate cover test).
Esotropia Evisceration	An inward deviation of the eyes. Removal of the cornea and contents of the eyeball, leaving the sclera behind.
Exenteration	Removal of the entire contents of the orbit, including the eyeball and lids.
Exophoria	A tendency of the eyes to turn outwards when binocular reflexes are disrupted by covering one eye (as in the alternate cover test).

Exotropia	A manifest outward deviation of one or both eyes.
Field of Vision	The entire area which can be seen without shifting the gaze.
Floaters	The sensation caused by small opacities moving in the vitreous.
Fovea	Depression in the macula adapted for most acute vision.
Fundus	The posterior of the eye visible through an ophthalmoscope.
Fusion	Co-ordination of the images received by the two eyes into one image.
Lacrimal sac	The dilated area at the junction of the nasolacrimal duct and the canaliculi.
Limbus	Junction of the cornea and sclera.
Long-sightedness	See Hypermetropia
Macula Lutea	The small avascular area of the retina surrounding the fovea.
Microphthalmos	Abnormally small eyeball.
Miotic	A drug causing pupillary constriction e.g. acetylcholine or pilocarpine.
Mydriatic	A drug causing pupillary dilation such as atropine, adrenaline or phenylephrine.
Myopia (Nearsightedness)	Refractive error in which the focal point for light rays from a distant object is anterior to the retina. The patient has blurred vision for distance. Myopia is most commonly associated with a large eyeball.
Nystagmus	An involuntary, rapid movement of the eyeball which may be horizontal, vertical, rotary, or mixed.
Ophthalmia Neonatorum	Conjunctivitis in the newborn, classically caused by gonococcus with devastating results. Nowadays, more commonly caused by chlamydial infection.
Optic Atrophy	Optic nerve damage characterised by optic disc pallor.
Dispensing Optician	Health Care professional who fits and sells spectacles prescribed by an optometrist or ophthalmologist.
Optometrist (Ophthalmic Optician)	Health Care professional who examines eyes, and prescribes glasses. Optometrists working in the community have a role in detecting eye disorders and referring to a GP or an ophthalmologist where appropriate. In addition they normally fit and sell glasses and contact lenses they prescribe. Some optometrists work in the hospital eye service supporting the role of ophthalmologists.

Ophthalmologist	Medically qualified doctor who can prescribe glasses and undertakes diagnosis and treatment of eye disorders. There are over 1000 consultant ophthalmologists in the UK, most of whom undertake eye surgery.
Orthoptist	Health care professional trained to detect measure and provide care for patients with ocular motility disorders and amblyopia. A large proportion of the work of an orthoptist is involved in the care of children with squint.
Oscillopsia	The subjective illusion of movement of objects that may occur with acquired nystagmus.
Palpebral	Pertaining to the eyelid.
Pannus	Infiltration of the cornea with blood vessels.
Papilloedema	Swelling of the optic disc, caused by with raised intracranial pressure.
Photocoagulation	A method of causing usually multiple areas of localised destruction of the retina for treatment of certain types of retinal disorders, e.g. diabetic retinopathy.
Posterior Chamber	Space filled with aqueous anterior to the lens and posterior to the iris.
Presbyopia	Blurred near vision requiring reading glasses, typically evident from the age of 45 years caused by progressive failure with age of the capacity to accommodate
Pterygium	A pathological triangular fold of tissue which extends from the conjunctiva over the cornea.
Ptosis	Drooping of the eyelid.
Puncta	External orifices of the upper and lower canaliculi.
Refractive Error (ametropia)	A defect (such as myopia, hypermetropia or astigmatism) that prevents light rays from being brought to a single focus on the retina
Retinal Detachment	A separation of the retina from the choroid. Patients with retinal detachment (which is more common in myopic individuals) typically complain of flashing lights (photopsia) and floaters followed by a black curtain spreading across the field of vision. Prompt surgical intervention is usually indicated.
Retinitis Pigmentosa	A hereditary degeneration of the retina.
Retinoscope	An instrument designed for the objective measurement of refraction.
Sclera	The white part of the eye - a tough covering which, with the cornea, forms the external protective coat of the eye.
Scotoma	A blind or partially blind area in the visual field.
Short-sightedness	see Myopia

Slitlamp	A microscope providing focal illumination for examination of the eye
Snellen Chart	Used for testing central visual acuity. It consists of lines of letters or numbers, in graded sizes so that a person with 'standard vision' able to resolve two limbs of a letter ''E'' subtending one minute of arc is able to read the 6 line at 6 metres. A patient able to read the top ''60'' line at 6 metres has an acuity of 6/60. American Snellen charts are calibrated in feet. Hence the term 20/20 vision which is equivalent to an acuity of 6/6.
Strabismus (squint, tropia)	Misalignment of the eyes.
Stye	Infection (usually staphylococcal) of an eyelash follicle
Sympathetic Ophthalmia	Inflammation in one eye following traumatic inflammation in the fellow eye.
Synechia	Adhesion of the iris to cornea (anterior synechiae) or lens (posterior synechiae) Iritis is the usual cause of posterior synechiae. In iritis associated with Juvenile Chronic arthritis the only indicator of severe sight threatening eye disease may be irregularity of the pupil caused by posterior synechiae.
Trachoma	Serious infectious keratoconjunctivitis due to repeated chlamydial infection in hot dry climates where flies are prevalent. Worldwide this is one of the commonest cause of blindness. see Entropion
Uvea (uveal tract)	The iris, ciliary body and choroid.
Uveitis	Inflammation of one or all portions of the uveal tract.
Visual Acuity	Measure of detailed central vision used for reading face recognition etc (see also Snellen chart)
Vitreous	Transparent, colourless mass of soft, gelatinous material filling the eyeball behind the lens.
Zonule	The numerous fine tissue strands which stretch from the ciliary processes to the lens equator (360 degrees) and hold the lens in place.

Ears, Nose and Throat

Learning Objectives and Aims

By the end of the course, you should be able to

- Take a relevant ENT history and carry out an appropriate examination on a patient
- Gain an understanding of the disability caused by different ENT pathologies and to have a clear idea of the most appropriate advice to give to the patient to cope with this and the relevant resources they might wish to call upon e.g. Hearing loss may require referral for a hearing aid, tinnitus counselling etc.
- Understand how ENT problems contribute to the management of pathology in other systems and vice versa.
- Overall, best holistic management of the patient is the desired outcome and the student needs to have an idea of which other expertise outside of ENT may be required to achieve this.

Students need to be able to identify ENT specific symptoms and have an idea of what the underlying pathology is likely to be. It is important that students are able to triage symptoms into those that demand immediate attention and those that are likely to indicate more long-term benign disease. (e.g. airway compromise – immediate attention, gradual long term hearing loss –elective treatment)

Students need to demonstrate their ability to identify the important causes of

- nasal blockage
- rhinitis
- epistaxis
- deafness
- pain in the ear
- pain in the throat
- difficulty swallowing
- swelling of the neck
- facial pain
- headache
- hoarseness

- ✓ by taking an appropriate history to reach a provisional diagnosis
- ✓ Show an ability to examine effectively the ear, nose and head and neck with minimal equipment
- ✓ Use investigations selectively to confirm diagnostic hypotheses
- ✓ Formulate a simple management plan

Students should be conversant with techniques in diagnosis, investigations and treatment of the following ENT conditions:

- Otitis Media with Effusion and childhood hearing loss in general
- Rhinitis – allergic and non-allergic
- Balance disorders – vestibular and non-vestibular
- Rhinosinusitis – acute and chronic
- Middle ear disease including infection, cholesteatoma, and chronic otitis media
- Deafness, conductive and sensorineural in adults
- Infections and Inflammation of the outer ear and external auditory meatus
- Voice disorders and their causes
- Lumps and bumps in the head/neck and how to effectively manage different types

ENT Placement Introduction

Welcome to your ENT placement! We hope that you enjoy your time with us. This workbook is intended for all students currently on their ENT placement.

Information for Students on ENT placement

Your ENT placement makes use of key resources to direct your learning. It is anticipated that you will attend clinical activities during your placement, during which time you will be able to complete the ENT sign off in this booklet. It is also anticipated that you will review the online resources which will be described to you at the induction lectures and, that you will fill out your ENT case studies in this workbook. Useful information can also be found on http://www.worldmedicaleducation.org/

Timetable/Clinical Activity

The administrator at each site will contact you prior to the start of your placement to guide you in terms of your timetable of activities for your allocated site. Please be aware that self-directed learning via the workbook and blackboard are also a vital part of your learning for ENT.

Borrowing of Otoscope

UHL based students can borrow an Ophthalmoscope/Otoscope from the Clinical Skills Unit, Level 1, Robert Kilpatrick Building. Students undertaking placement at Kettering and Lincoln should borrow an Ophthalmoscope/Otoscope from the Clinical Skills Unit there.

ENT Skills Sign Off

This sign off is for your guidance only. You will not be graded based on this sign off. You may have an opportunity (or have had an opportunity) to undertake some of these skills in the acute care block during your eye casualty/emergency slot.

PROCEDURE/EXAMINATION	Student Signature	ENT verification signature
EAR **By the end of Phase 2 you should be able to perform the following examinations:**		
Otoscopy		
Identify the eardrum		
Understanding the tuning fork tests		
Identify an abnormal eardrum, including glue ear and perforation of the eardrum		
Understand an audiogram and a tympanogram		
NOSE **By the end of Phase 2 you should be able to perform the following examinations:**		
Performing an anterior rhinoscopy using a nasal speculum and headlights		
Assess epistaxis and various nasal packing devices		
THROAT **By the end of Phase 2 you should be able to perform the following examinations:**		
Performing an examination of the oral cavity and oral pharynx using a headlight and tongue depressors		
An examination of the neck		
Ability to explain to patients the use of rigid and flexible endoscopy under local anaesthetic		

ENT Case Studies

Answers are located at the end of the ENT section of this workbook

Case Study 1

A 23 year-old female, previously fit and well, complains of unilateral otalgia and an offensive discharge that has persisted for 2 days. She has recently been on holiday to Egypt.

1. What is the most likely diagnosis?

2. What are are the most common organisms causing this condition?

3. She is planning to go on holiday again. What should you advise her about swimming and flying?

4. How would you expect the ear canal and tympanic membrane to appear?

5. What risk factors can predispose to this condition?

Case Study 2

A 12-year-old child presents to the A&E Department with his mother and complains of pain in his left ear after using ear buds.

1. What differential diagnoses would you consider?

2. Why would patients be advised not to 'clean' the ears with cotton buds?

3. You find that there is a small perforation antero-inferiorly in the child's tympanic membrane. How will you manage this patient?

Case Study 3

A 6-year-old child presents with his father to the emergency ENT clinic with a pyrexia and complaining of otalgia. You note he has seen the school nurse recently as staff feel he is not working as hard as he was earlier in the year.

1. What diagnosis is most likely?

2. Why might he not be performing as well in school of late?

3. How would you expect the tympanic membrane to appear?

4. You examine the ear and notice that one ear appears to protrude more than the other does. What complication are you worried about?

5. What common organisms are implicated in this condition?

6. List below the complications associated with infection in this part of the ear

Intracranial Complications	Intra temporal bone Complications
Meningitis Intracranial abscess Sigmoid sinus thrombosis	Bacterial labyrinthitis Facial paralysis Petrous apicitis Gradanigo's syndrome Citelli abscess

He has started on intravenous antibiotics but after 24 hours of treatment there has been no improvement. After a normal MRI of his head to look for intracranial complications, he is taken to theatre for a cortical mastoidectomy.

7. What important relations of the middle ear/mastoid are at risk?

Case Study 4

A 33 year-old lady presents with a one-year history of foul smelling discharge from her right ear and gradual deterioration in her hearing. On examination, there is a perforation at the attic, and a white substance occupies the area.

1. What is the most likely diagnosis?

2. What possible reasons are there for the deterioration in her hearing?

3. What results would you expect with tuning fork tests?

4. Post-operatively, you note that she is doing quite well, and she remains asymptomatic for the following two years. She returns some months later with similar symptoms and new onset vertigo. What possible explanations are there for her latest presentation?

Case Study 5

A 45 year-old man presents to you with sudden onset dizziness. He gets episodic room spinning with nausea, especially with certain head movements and lying in bed.

1. What is the most likely diagnosis?

2. What else would you want to know in the history?

3. What examination can you perform which might help establish the cause of his problems? What would you hope to see?

4. What manoeuvre can you carry out to help his symptoms?

Case Study 6

A 16-year old boy comes to the A&E Department with epistaxis which has spontaneously resolved. He claims to have injured his nose whilst re-enacting last night's UFC match with a friend. He is seen by the A&E SHO and is sent home with no follow-up. He returns to the emergency ENT clinic 4 months later complaining of a change in shape to his nose. On examination, there is an obvious "step," which looks like a saddle deformity.

1. Describe the likely underlying series of events that have resulted in this deformity, after the initial trauma.

2. How would you have identified, and therefore prevented this from happening on his initial presentation?

3. What recreational drug is commonly implicated in nasal septal defects, and what is its method of damage?

Case Study 7

A 36-year old man with chronic rhinosinusitis is admitted for Functional Endoscopic Sinus Surgery (FESS).

1. What medical treatment would you have expected them to have before considering surgery?

2. What are the potential complications of sinus surgery?

Case Study 8

A 50-year old man presents to A&E with Epistaxis. He has tried conservative measures at home including ice, and pinching the soft part of the nose with no success. On arrival, he is tachycardic, tachypnoeic, and actively bleeding from his left nose

1. How would you manage this patient?

2. His nose is eventually packed which initially controls the bleeding. He then starts to spit up fresh blood. Outline what steps you would take to stop a posterior epistaxis.

3. How would you manage this patient if packing and cautery of the bleeding vessels were to fail?

Case Study 9

A 74-year old man is referred to ENT with a right-sided craggy mass at the inferior border of his mandible. He is normally fit and well, smokes 20 cigarettes a day, and drinks regularly.

1. Describe the relations of the submandibular gland.

2. You are concerned that he may have a malignancy so after appropriate investigations you decide that he needs to have his submandibular gland removed along with any lymph nodes in the submandibular triangle. What complications of surgery would you mention in the consent?

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Case Study 10

An 80-year old man is referred with a left sided mass anterior to the ear. He complains of drooling, and his face is drooping on the left. He has several hard lymph nodes in his neck on examination.

1. What salivary gland is likely to be affected?

2. What features in the history suggest that this is a benign or malignant mass?

3. The patient is seen 6 months after surgery and there is no evidence of a mass. He still has facial weakness, but now complains that when he eats, his face begins to sweat profusely. What has happened and what is this known as?

4. When performing a neck dissection, what important structure in the posterior triangle is at risk of damage? If damaged, how might this affect the patient?

Case Study 11

A 60-year old man presents with a right sided neck nodule that moves on swallowing. On examination, he is warm and well perfused, tachycardic, and appears to be staring. There is an obvious asymmetry to his neck.

1. What are the most likely diagnoses?

2. How would you clinically differentiate a thyroglossal cyst from a thyroid mass?

3. What are the risks of carrying out thyroid surgery on someone who is hyperthyroid?

4. What are the complications of carrying out thyroid surgery on a euthyroid patient?

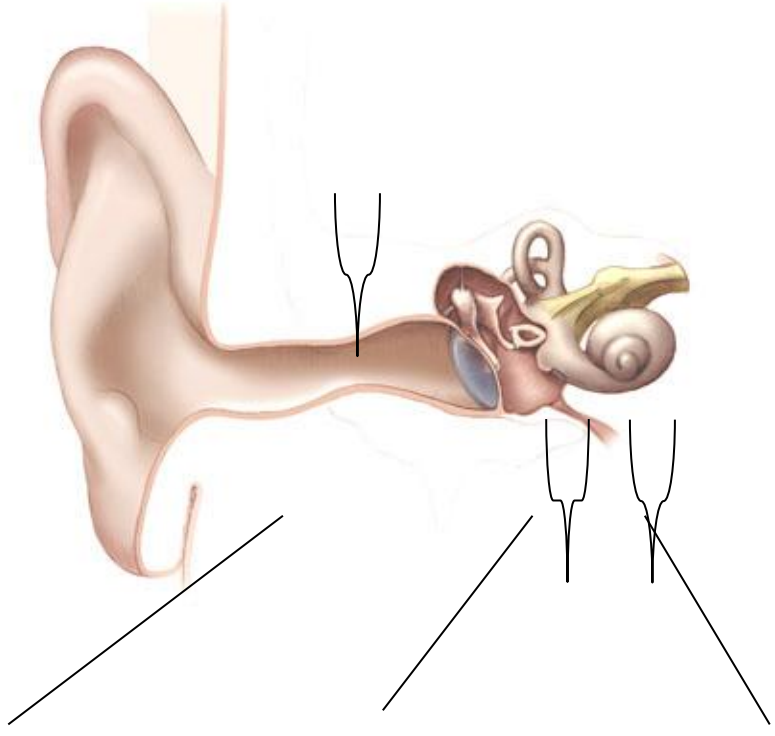
5. Describe the course of the recurrent laryngeal nerve on the left and on the right.

6. What is the most important blood test to check after a routine total thyroidectomy and what symptoms would suggest that this blood test was going to be low?

The Ear

Learning Objectives:

- To revise the gross anatomy of the ear, and the relevance of this to clinical practice.
- To understand the pathophysiology, and therefore management of common conditions involving:
    - The external ear
    - The middle ear
    - The inner ear



External Middle Internal

The anatomy of the ear is complex, especially when considered as a single entity. It is much more palatable when considered in three parts; external, middle and inner ear.

This chapter aims to cover the common clinical conditions in each of the three parts of the ear, thus supplementing the anatomy and physiology of the head and neck.

External Ear

Ear Trauma

This is a common occurrence, typically seen in sport-related injuries, but also as a result of violence and aggression involving a blow to the ear. Fortunately in the majority of uncomplicated cases, treatment can be administered quickly and often under local anaesthetic.

It is therefore important to know the sensory supply to the pinna.

- Upper lateral surface – CN V_3 – Auriculotemporal nerve.
- Lower lateral surface and medial surface – C3 – Greater auricular nerve.
- Superior medial surface – C2/C3 – Lesser occipital nerve.
- Auricular branch of vagus – External Auditory Meatus

Performing regional nerve blocks at each of the nerves mentioned above, anaesthetises the pinna and allows a variety of procedures to be performed.

i. **Lacerations**

Simple primary closure of the skin with sutures after adequate cleaning is all that is required in most cases. It is important to ensure that any exposed cartilage is covered with skin. If there is significant skin loss where primary closure will not be possible an opinion from a plastic reconstructive surgeon may be required.

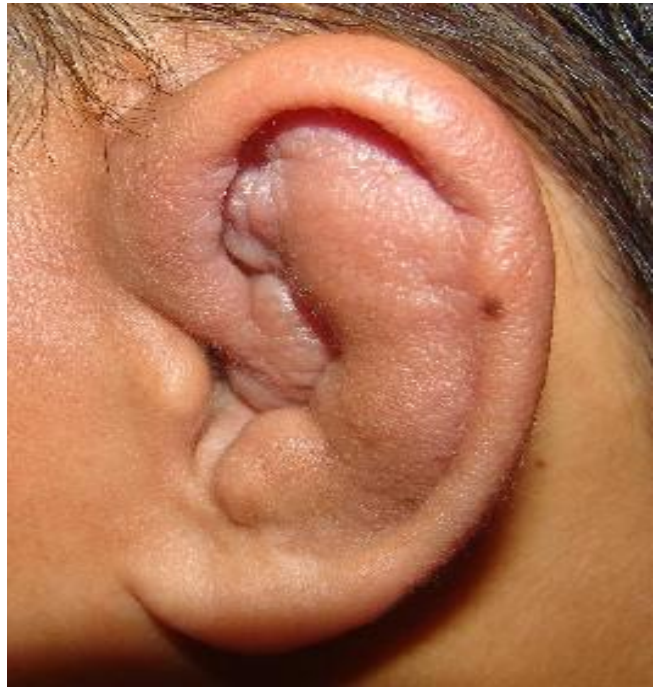
ii. **Bites**

Bites to the ear carry a significant risk of infection from skin commensals, or oral commensals from the offending creature/person. These require an appropriate history to ascertain likely organisms involved in potential infection, and the wound must be left open. Treatment includes thorough wound irrigation and antibiotics.

iii. **Haematoma**

Pinna haematomas cause disruption in the blood supply to the cartilage. Normally, the cartilage obtains nutrients via diffusion from vessels in the overlying perichondrium, and a disruption can therefore lead to a vascular necrosis.

There is a significant risk of associated deformity (cauliflower ear) and this therefore requires urgent drainage and pressure dressing application to prevent re-accumulation.



iv. **Tympanic Membrane Perforation**

The tympanic membrane can be perforated by direct or indirect trauma. This subsequently leads to:

- Pain
- Possible conductive deafness

Most perforations will heal by themselves, and therefore only require a conservative "watch and wait," approach with the patients following water precautions. In cases where the perforation does not heal after a period of 6 months or more the patient may benefit from surgical intervention in the form of a myringoplasty to repair the tympanic membrane if the perforation is causing problems.

Perforations can also be caused by otitis media.

(v) Haemotympanum

Trauma may cause a haemotympanum (blood in the middle ear) which may be associated with a temporal bone fracture. This can be seen through the tympanic membrane and is associated with a conductive hearing loss. It should be treated conservatively as it will settle with time but patients should be followed up to ensure that there is no residual hearing loss from damage to the ossicles.

Ear Infections (Otitis Externa)

This is inflammation of the skin lining the external canal, and is occasionally known as "swimmer's ear". It may be bacterial or fungal. When considering otitis externa (OE), it is useful to consider the external ear as a continuation of the rest of the skin. It is therefore inhabited by the same skin commensals that can be found elsewhere.

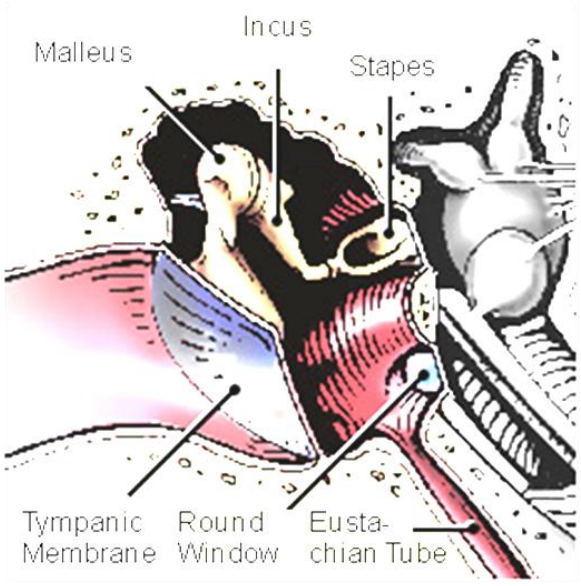
Patients normally present with a painful discharging ear and there is often a history of an itchy ear. The hearing maybe muffled from the discharge present in the ear canal.

Malignant otitis externa is a particularly aggressive infection mainly seen in diabetics or immunocompromised patients where the infection spreads from the soft tissue of the ear canal into the bone. It normally presents with chronic ear discharge despite topical treatment, a deep seated severe ear pain and sometimes cranial nerve palsies (most commonly CNVII). It has a significant mortality rate of around 10% despite aggressive treatment.

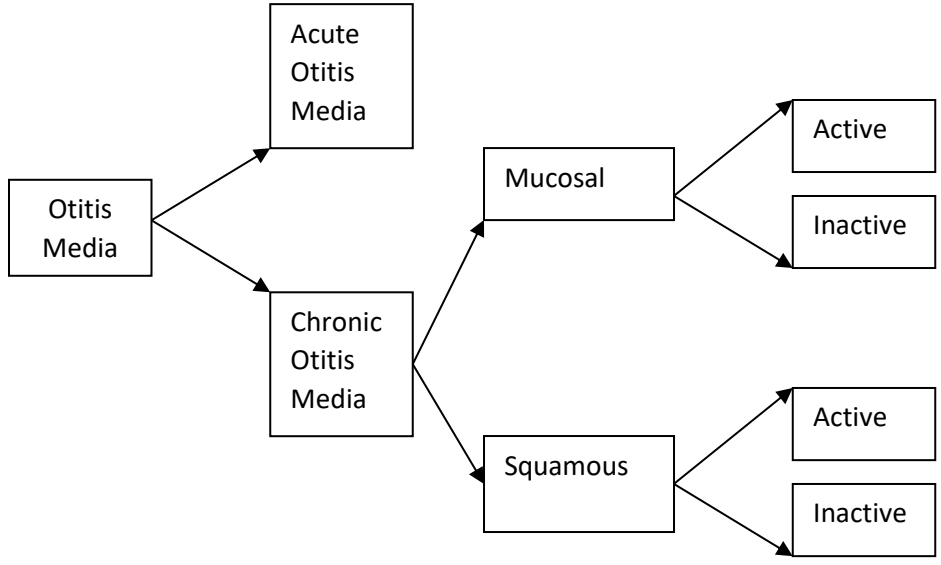
Management

- Topical eardrops empirically e.g. Gentamicin.
- Swab any discharge in resistant cases.
- Microsuction of pus/debris which enables the drops to get to the source of the infection
- In severe infection, a wick may be used to help hold the canal open and to allow topical treatment to diffuse through
- Fungal infections should be treated with topical antifungals as appropriate.
- Malignant otitis externa will require aggressive treatment with iv antibiotics as well as topical treatment for an extended period of time to eradicate infection

Middle Ear



Ear infections (Otitis media)



i. **Acute Otitis Media**

Acute otitis media (AOM) is an infection of the middle ear. Unlike the external ear, the epithelium lining the middle ear is respiratory epithelium, or pseudostratified columnar epithelium. The middle ear can therefore be regarded as a continuation of the upper respiratory tract, and is therefore susceptible to a similar variety of pathogens.

AOM is more common in childhood and is related to eustachian tube dysfunction. Common pathogens include Streptococcus pneumoniae, Haemophilus influenzae and Moraxella species.

Clinical Features

- Ear pain
    - caused by increased pressure in the tympanic cavity, in young children this may be evident by ear pulling.
- Discharge
    - the tympanic membrane may rupture with the pus from the middle ear discharging into the ear canal. When this happens the pain settles and the ear discharge starts.
- Fever

Management

- Conservative – Most patients can be managed conservatively with analgesia
- Medical – In severe or persistent cases oral antibiotics may be required
- Surgery – Recurrent AOM may be helped by grommet (ventilation tube) insertion

ii. **Chronic Otitis Media**

Chronic otitis media (COM) can be divided into active or inactive depending on whether the ear is discharging (active) or not (inactive). It can then be subdivided into mucosal disease or squamous disease. Active squamous disease is also known as cholesteatoma and inactive squamous COM is a retraction pocket which may develop into active disease (cholesteatoma). Active mucosal disease is where there is chronic discharge from the middle ear through a tympanic membrane perforation and inactive mucosal disease is where there is a tympanic membrane perforation but no active infection/discharge.

Mucosal COM is thought to develop from an episode of AOM where after rupturing of the tympanic membrane there is a failure to heal and active squamous COM is thought to develop when keratinised squamous cells are introduced into the middle ear from a retraction pocket or a perforation.

Active COM is associated with chronic ear discharge and often a conductive hearing loss. It can also be associated with complications associated with spread of the disease within the temporal bone and intracranially.

Management

Management of COM depends on whether there is cholesteatoma present (squamous disease) or not. If there is cholesteatoma present surgery is required to clear the cholesteatoma. The cholesteatoma will often grow from the middle ear into the mastoid bone and this therefore often requires surgery to the mastoid bone (mastoidectomy). If there is no cholesteatoma (mucosal disease) there is a chance that this may clear up with medical treatment (topical antibiotic drops and aural toilet). Often it is not known whether cholesteatoma is present in a chronically discharging ear and in this case the ear should be treated medically first and then if this fails surgery should be carried out when cholesteatoma may or may not be discovered. In surgery where there is no cholesteatoma found the aim is to repair the perforation and ensure good ventilation of the middle ear and mastoid bone but it is not necessary to remove every last trace of disease unlike cholesteatoma. Mastoid surgery is associated with a small risk of a facial nerve palsy, altered taste from damage to the chorda typani, CSF leak, tinnitus, vertigo and also of complete loss of hearing in the operated ear.

Otitis media with effusion (Glue ear)

This is a condition where fluid is present in the middle ear with an intact tympanic membrane and is related to eustachian tube dysfunction. It is more common in children but can occur in adults. In adults with an Otitis Media with Effusion (OME) (especially unilateral) it is very important to have a look at the post nasal space as tumours in this area can cause eustachian tube dysfunction leading to OME. OME is not painful but the middle ear may become infected which will lead to an acute otitis media which is painful.

Clinical features

- Middle ear effusion on otoscopy
- Conductive hearing loss
    - This may be associated with speech delay and problems at school

Investigations

- Tympanogram
    - Flat (Type B) Tracing with normal canal volume
- Pure tone audiogram
    - Conductive hearing loss

Management

- Conservative – most cases settle within 3 months
- Hearing aid
- Surgery - for prolonged hearing loss causing significant problems
    - Grommets (ventilation tubes)
    - +/- Adenoidectomy

Otosclerosis

This is a disease affecting the ossicles in the middle ear. The cause has both genetic and environmental components. In families where otosclerosis is prevalent it appears to follow an autosomal dominant transmission. Mature bone is gradually replaced with woven bone, and symptoms develop as the stapes footplate becomes fixed to the oval window.

Otosclerosis causing symptoms is thought to occur in 1-2% of the population and 85% of these will develop bilateral disease. Twice as many females are affected compared with males.

History

- Progressive hearing loss
- Often tinnitus
- Improved hearing in noisy surroundings during early stages of disease
- Family History

Examination

- Most commonly normal
- Rarely pink hue to the tympanic membrane – Schwartze's sign

Investigations

- Tympanogram
    - Normal type A trace
- Pure tone audiogram
    - Conductive hearing loss
    - A characteristic "Carhart notch" at 2kHz

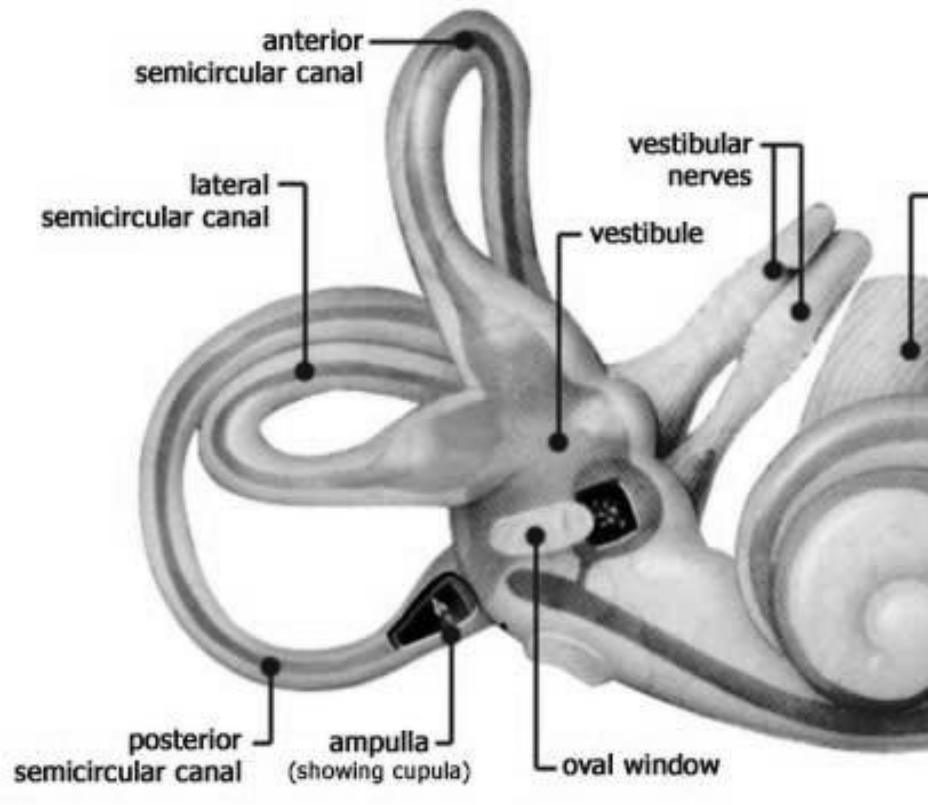
Management

- Conservative – hearing aid
- Surgery - stapedectomy

Inner Ear

The inner ear resides in the petrous part of the temporal bone and consists of a labyrinth of canals. It can be divided functionally into two parts:

- The vestibule and semicircular canals
- The cochlea



The membranous labyrinth is filled with endolymph, and this in turn is surrounded by the bony labyrinth. The membranous labyrinth is suspended in perilymph, contained within the bony labyrinth.

The perilymphatic system communicates with the subarachnoid space and CSF via the cochlear aqueduct. Perilymph fluid resembles CSF, and endolymph fluid resembles intracellular fluid.

The cochlea is responsible for the perception of hearing and is analogous in structure to the rest of the labyrinthine system but has a number of distinguishing features and functions;

- It has 2.5 turns around a bony core, the modiolus.
- The stapes articulates with the oval window, causing movement of perilymph, and a pressure change, compensated by the round window.
- Vibrations are transmitted through the endolymph to the tectorial membrane.
- Low frequency sounds are detected at the apex of the cochlea, and high frequency sounds at the base of the cochlear.
- Movement of the tectorial membrane causes movement of hair cells, and subsequent depolarisation of neuronal fibres allowing perception of sound.
- Transmission of information occurs via the cochlear nerve.

The vestibular system can be further divided into:

- Semicircular canals
- Utricle
- Saccule

There are three semicircular canals, each existing at 90° to the other.

The utricle and saccule detect movement in a linear fashion, rather than the rotatory mechanism as seen in the semicircular canals.

Simply put:

- **U**tricle – Hair cells point **U**p – Detect linear/horizontal movement
- **S**accule – Hair cells stick out to the **S**ide – Detect vertical movement

Good balance requires input from the vestibular system to be integrated centrally with proprioceptive and visual inputs and problems in any of these areas can cause problems with balance.

Vertigo

Vertigo is defined as the hallucination of movement and is associated with a problem with the vestibular system. It is essential to take a thorough history, and hence ascertain potential causes and treatment options. Causes may be due to central or peripheral vestibular pathology;

- Central causes:
  - Stroke
  - Migraine
  - Neoplasms
  - Demyelination eg. MS
  - Drugs

- Peripheral causes:
  - BPPV
  - Ménière's disease
  - Vestibular Neuronitis

i. BPPV

Benign Paroxysmal Positional Vertigo, as its name suggests, is vertigo occurring with particular head movements, which is benign in nature, and lasts a short amount of time, typically seconds. Unfortunately, this is often a very distressing condition for the patient, and at times, disabling.

The underlying cause are otoliths (crystals) in the semicircular canals (most commonly posterior) causing abnormal stimulation of the hair cells giving a hallucination of movement i.e. vertigo.

Diagnosis is by the Dix-Hallpike test and treatment is with the Epley manoeuvre. You should have the chance to see both of these during your attachment.

ii. Ménière's Disease

The precise aetiology of this is still unknown although many theories exist. The pathophysiology involves endolymphatic hydrops (increased fluid in the endolymphatic compartment).

Clinical Features

- Tinnitus in affected ear
- Episodic vertigo lasting minutes to hours associated with nausea & vomiting
- Fluctuating sensorineural hearing loss which over time becomes permanent
- Aural fullness

Initially in the course of the disease patients are well between attacks but as the disease progresses they may feel generally unsteady between attacks as they start to develop reduced vestibular function on the side of the disease and a progressive sensorineural hearing loss. Over time the disease often burns itself out so that the patient doesn't suffer from acute vertigo but will have reduced hearing and maybe generally unbalanced. If the vestibular system of the other ear is working well this can compensate for the bad ear to improve the overall balance but this can take time especially in the elderly.

Management

- Dietary – reduce salt, chocolate, alcohol, caffeine, chinese food
- Medical
  - Thiazide diuretics e.g. bendrofluazide
  - Betahistine
  - Vestibular sedatives e.g. prochlorperazine for acute attacks only

- Surgical
    - Grommet insertion
    - Dexamethasone middle ear injection
    - Endolymphatic sac decompression
    - Vestibular destruction using middle ear injection of gentamicin
    - Surgical labyrinthectomy – very rarely required

iii. **Vestibular Neuronitis**

In this condition inflammation in the inner ear causes severe incapacitating vertigo lasting several days associated with nausea and vomiting. During an attack patients will have horizontal nystagmus but an otherwise normal neurological examination. Treatment is symptomatic with vestibular sedatives during the acute episode and intravenous fluids if required. There is often an associated long term vestibular deficit after the acute episode which can lead to a generalised unsteadiness after the acute episode for a number of weeks while the brain compensates for this. If patients are suffering from prolonged poor balance from vestibular hypofunction in one ear from vestibular neuronitis or any other pathology they can be helped by vestibular rehabilitation exercises such as the Cawthorne-Cooksey exercises. After the acute attack patients should not take vestibular suppressants as this will delay their recovery.

Sudden onset sensorineural hearing loss

This is an otological emergency that may cause lifelong disability for the patient. Patients who have a sudden onset hearing loss need to be assessed to see whether it is sensorineural or conductive. This can be done by tuning fork tests or a pure tone audiogram if you have access to this.

Prognosis

1. 1/3 recovery to normal
2. 1/3 some recovery
3. 1/3 no recovery

Investigations

- Pure tone audiogram
- MRI scan
    - to exclude lesion along central auditory pathway. e.g. acoustic neuroma

Management

- Steroids – normally orally but can be injected into middle ear
- Anti-virals
- Other treatments are used e.g. hyperbaric oxygen, carbogen but are not widely practiced

Hearing Tests

A. Tuning fork tests

These tests help to differentiate a sensorineural hearing loss from a conductive hearing loss. This is extremely important as patients with sudden onset sensorineural hearing loss require urgent treatment and most of the time that these patients present you will not

have access to a pure tone audiogram. A 256 Hz or 512 Hz tuning fork should be used. These tests assume that there is normal hearing in the other ear.

i. Weber Test

The tuning fork is knocked against your knee or elbow and placed on the patient's forehead. The patient is asked if they can hear the noise on the left, right, centre or not at all. The tone produced from the tuning fork is transmitted via the bone of the skull to both cochlear.

- Sensorineural hearing loss – the tone will be heard on the opposite side to the hearing loss
- Conductive hearing loss – the tone will be heard on the same side as the hearing loss. This is because the conductive loss will block out background noise so relative to the other ear the tone will sound louder in that ear.

ii. Rinne test

The tuning fork is knocked against your knee or elbow and placed on the patient's mastoid for a few seconds and then lateral to the external meatus of the ear. By placing the tuning fork on the mastoid the tone is being conducted to the cochlear via the temporal bone and by placing the fork lateral to the ear the tone goes through the external canal and gets the benefit of the amplification from the external and middle ear. In a normal hearing patient the tuning fork will be heard louder when held lateral to the external auditory meatus, this is referred to as Rinne positive as opposed to Rinne negative where the sound is heard louder when the tuning fork is placed over the mastoid.

- Sensorineural hearing loss – Rinne +ve if heard at all because you are still getting the benefit of the amplification of the external and middle ear
- Conductive hearing loss – Rinne -ve as you are no longer getting the benefit of the amplification from the middle and external ear.

B. Pure tone audiogram (PTA)

This test assesses the hearing thresholds of patients at different frequencies and is the most widely used assessment of patient's hearing. It can be used from around the age of 4 upwards. It should be performed in a sound proofed booth. Tones are played to the patient at different frequencies to each ear. Each frequency is tested a number of times and the quietest tone that can be reliably heard is marked on the audiogram.

The frequency in hertz is on the x axis and the decibel scale on the y axis with 0 at the top and increasing decibels (loudness) as you descend the y axis. The higher the decibel number the louder the noise, so the higher the line on the pure tone audiogram the better the hearing. Anything above 20dB on the audiogram is considered normal. Air conduction and bone conduction thresholds are tested.

Air conduction is measured by playing the tone through headphones and assesses the whole auditory pathway. Bone conduction is measured by playing the tone through a bone conductor placed over the mastoid bone. This then passes through the bone straight to the cochlear bypassing the conductive part of the auditory system (external and middle ear) and therefore approximates to sensorineural hearing. Sound played through the bone conductor to one ear will not only travel to the cochlear on that side but also to the cochlear on the other side so if there is any discrepancy in the hearing between the ears the other side will need to be masked. This means playing a noise to the contralateral ear to prevent it from detecting the test tone. This is not such a problem for the air conduction unless there is a very large difference in air conduction thresholds between the two ears.

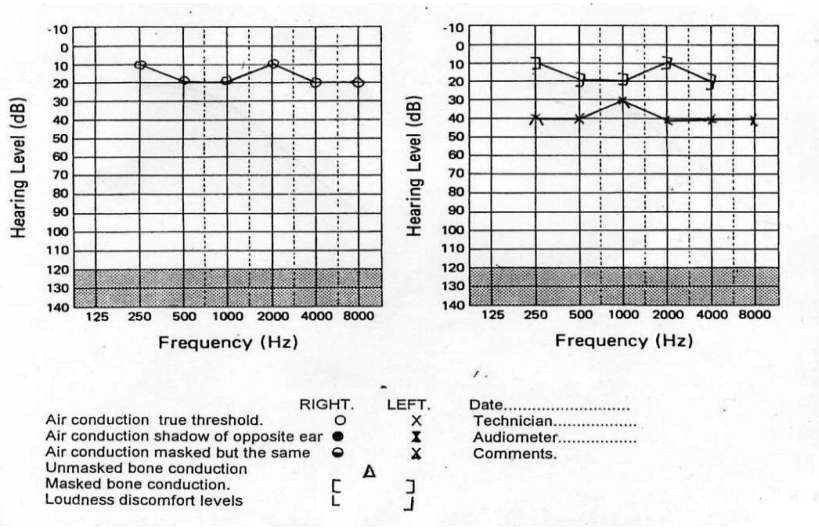
The thresholds are marked on the audiogram. Different marks are made on the audiogram depending on the ear being tested, whether bone conduction or air conduction is being recorded and whether masking is being used or not. This information will normally be on a legend beside the audiogram. Both ears may be marked on the same graph or they may be plotted on separate graphs next to each other when the right ear will be on the left hand graph and the left ear on the right hand graph.

There are two types of hearing loss – conductive and sensorineural.

i. Conductive

- this is caused by an impedance to hearing in the middle or external ear

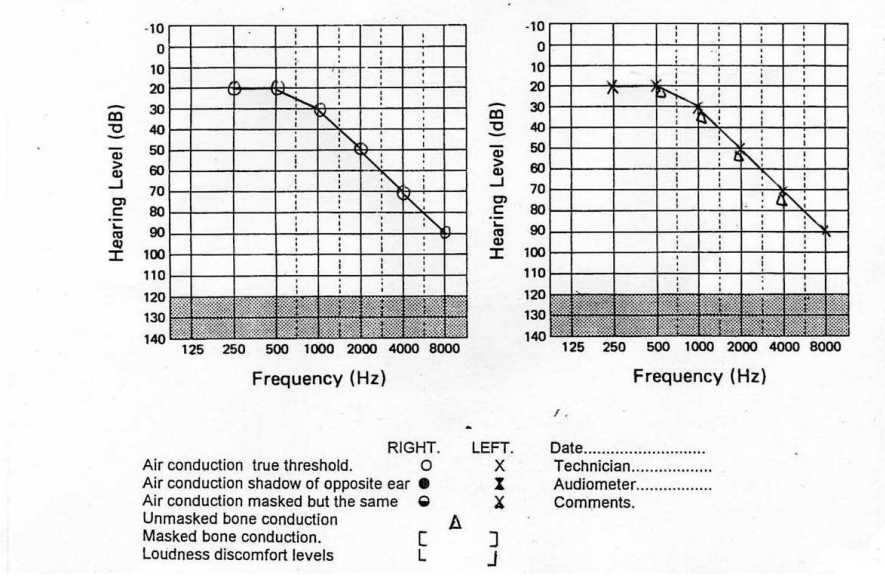
- e.g. wax, otitis media with effusion
- the audiogram has normal bone conduction and reduced air conduction thresholds i.e. an air bone gap



Pure tone audiogram showing normal hearing in the right ear and a conductive hearing loss in the left ear.

ii. **Sensorineural**

- this is caused by a problem from anywhere between the cochlear and the auditory cortex of the brain
- asymmetrical sensorineural hearing loss should be investigated with an MRI scan looking for lesions along the pathway e.g. acoustic neuroma (vestibular schwanoma)
- the audiogram will have reduced bone and air conduction thresholds i.e. no air bone gap



Audiogram showing a bilateral sensorineural hearing loss consistent with hearing loss seen in old age (presbycusis)

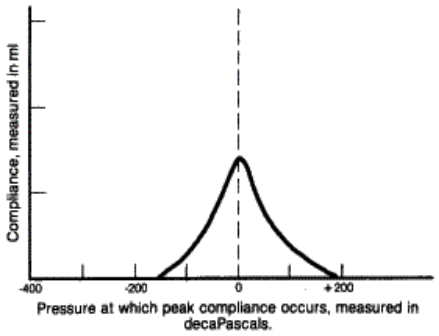
Patients may also have a mixed hearing loss with both conductive and sensorineural components.

C. Tympanogram

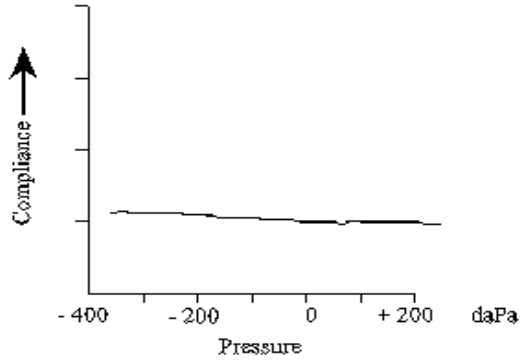
This is a simple test that measures compliance of the tympanic membrane and gives useful information about the tympanic membrane, the middle ear and eustachian tube function. It involves inserting a probe in the external ear canal and only takes seconds to perform. It can be done at any age as it takes minimal cooperation from the patient.

Compliance is measured along the y axis and pressure (daPascals) is measured on the x axis. The compliance of the tympanic membrane is then measured with varying amounts of pressure in the external ear canal which is sealed by the probe. The compliance peaks when the pressure in the canal equals that of the middle ear. Three types of tracing are classically seen

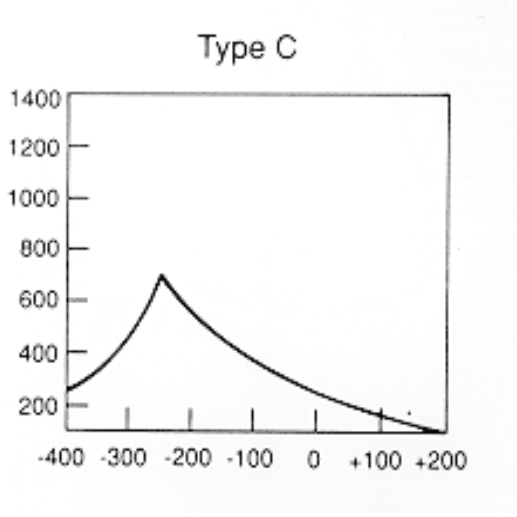
i. **Type A**
- Normal result
- Peak centred on 0 daPa on x axis



ii. **Type B**
- Flat tracing
- Suggests middle ear effusion or perforation
- By looking at the canal volume reading on the side of the tympanogram you can differentiate an effusion from a perforation. An effusion will have a normal canal volume (around 1cm^3 in adults) whereas in a perforation the volume being measured will be much larger as it is measuring the middle and external ear volume.



iii. **Type C**
- Suggests eustachian tube dysfunction
- The peak of the tracing has negative pressure



The Nose/Sinuses

Learning Objectives:

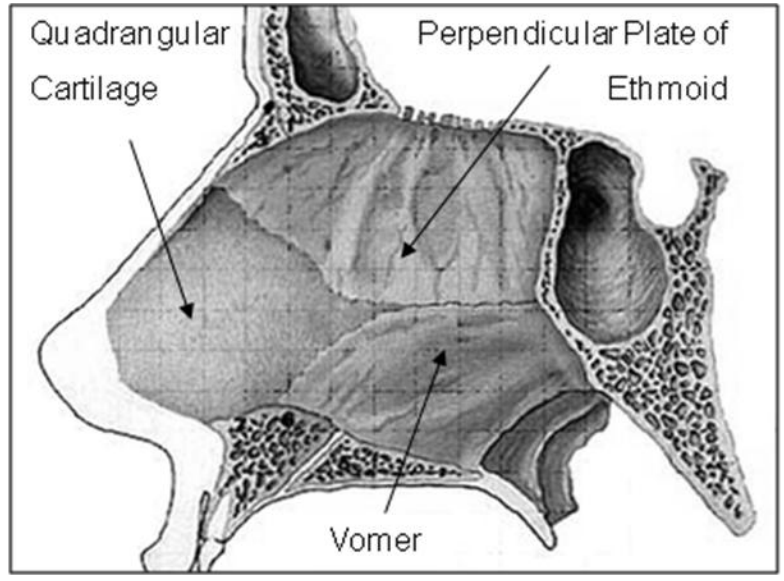
- To revise the gross anatomy of the nose and paranasal sinuses, and their clinical relevance.
- To understand the pathophysiology of common conditions affecting the nose and paranasal sinuses.

This chapter aims to cover the common clinical conditions you will see affecting the nose and related structures. Anatomy will briefly be revisited. The nose will be considered as one entity, with the paranasal sinuses being discussed separately, and a brief mention of the nasopharynx and its clinical relevance.

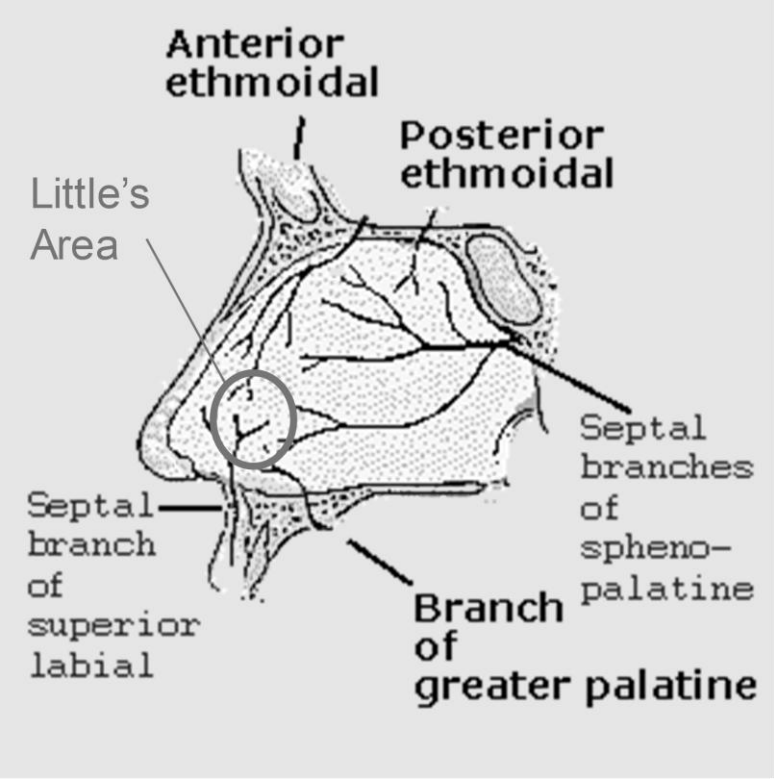
Anatomy of the Nose

External nose

The external nose receives support from bone (nasal bones) and a number of cartilages.



Blood Supply to the Nose



Nasal Septum

Little's area, also known as Kiesselbach's plexus, is a highly vascular area on the nasal septum. It receives a contribution from branches of the external carotid and internal carotid arteries.

This becomes important when considering the management of epistaxis, as haemorrhage may be from one, or a combination of these arteries.

Epistaxis

Epistaxis is bleeding from the nose, and in the vast majority of cases is relatively insignificant. In a small proportion however, it can present with massive haemorrhage and shock.

It is vital therefore to obtain a concise, but thorough history to establish the cause, and any perpetuating factors such as anticoagulant use, and hypertension.

Causes

Local

- Idiopathic – 85%
- Traumatic
- Iatrogenic
- Foreign Body
- Inflammatory – Rhinitis, Polyps
- Neoplastic

Systemic

- Hypertension
- Coagulopathies
- Vasculopathies
- Hereditary Haemorrhagic Telangiectasia/Osler-Weber-Rendu disease

Management

- ABC
- First Aid
    - Pinch soft part of nose
    - Head forward
    - Spit out (not swallow) any blood in mouth
- Examination
    - Locate source of bleed – Anterior or Posterior bleeding?

- Conservative
    - Cautery – silver nitrate or bipolar diathermy
        - If anterior bleed – with anterior rhinoscopy

        - If posterior bleed – with rigid endoscope
        - Topical adrenaline may help control bleeding before cautery
    - Nasal packing if cautery fails to control bleeding
        - Initially anterior pack
        - If continues bleeding into oropharynx anterior & posterior pack
- Surgical/Radiological
    - If nasal packing fails to stop the bleeding the following vessels can either be ligated surgically or embolised radiologically
        - Sphenopalatine
        - Anterior ethmoid (can not be embolised because comes from internal carotid artery)
        - External carotid (last resort)

Nasal Trauma

Nasal trauma commonly occurs following assault, and is also seen in sport, falls, and road traffic accidents.

Fracture of the nasal bones may be complicated in the following ways:

- Septal haematoma
- CSF leak with associated skull base fracture (rare)

Management

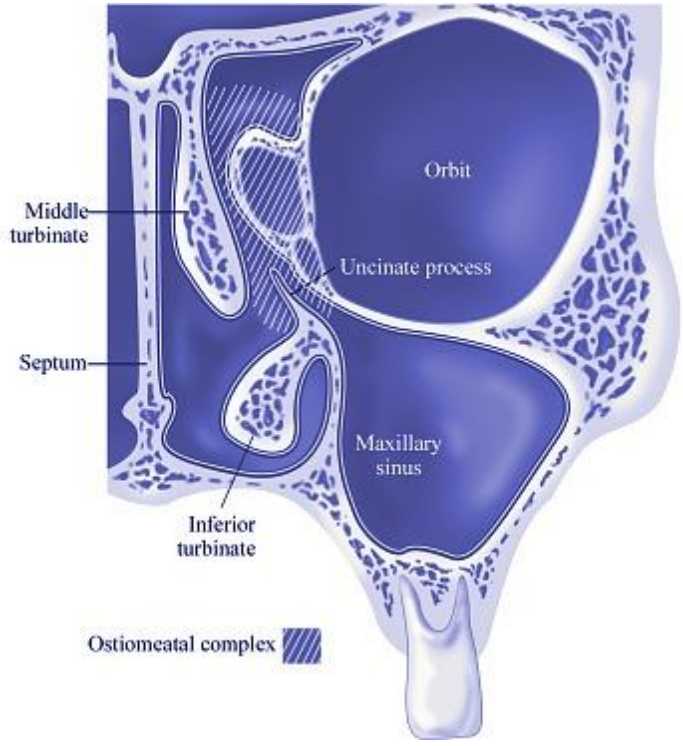
- ABC – epistaxis normally self-limiting
- Examine for septal haematoma
- No XR required
- If deviated nose consider Manipulation under anaesthetic (LA/GA) within 2 weeks of injury

Paranasal Sinus Anatomy

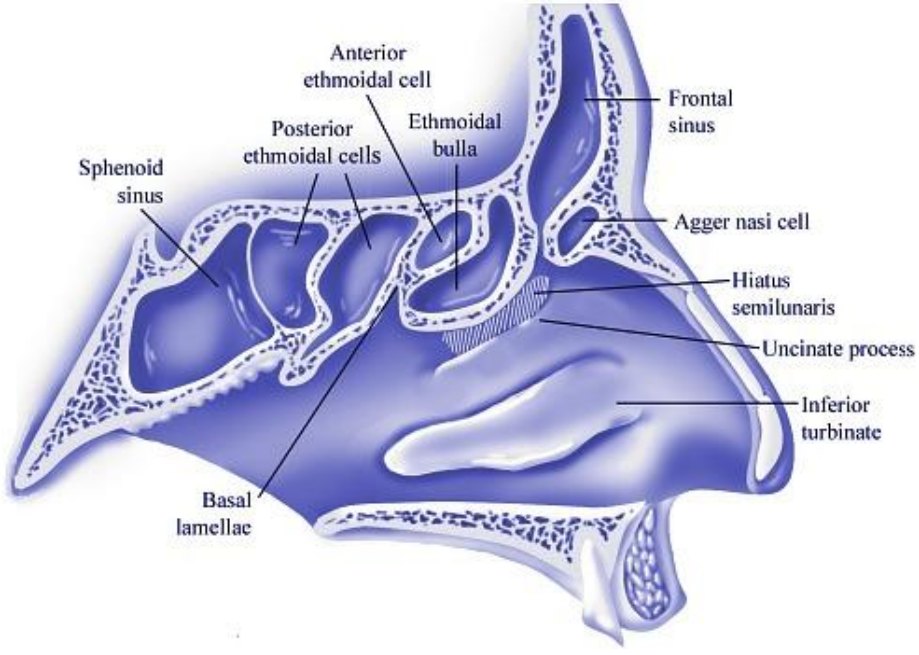
Knowing the relationship of the paranasal sinuses is particularly important when considering surgery.

Due to the intimate nature of structures within the head, there is a significant risk of injury to surrounding structures, especially when the surgeon is working with minimal access in such an enclosed space.

Structures of importance include the lamina papyracea forming the medial wall of the orbit, the anterior cranial fossa and the internal carotid artery



Site of Drainage	Paranasal Sinus
Spheno-ethmoidal recess	Sphenoid sinus
Superior meatus	Posterior ethmoid cells
Middle meatus	All other sinuses Frontal Maxillary Anterior ethmoid cells
Inferior meatus	Nasolacrimal duct



Complications Involving the Sinuses

- Surgery
- Damage to orbit – the orbit lies lateral to the ethmoid sinus and superior to the maxillary sinus and its contents are at risk during sinus surgery and in severe cases can cause loss of site
- Anterior Skull base – lies just above the sphenoid and ethmoid sinuses and can be breached during surgery which can cause a CSF leak and in the worst cases brain damage.

- Rhinosinusitis

- Orbit – infective rhinosinusitis can spread to the orbit to cause periorbital sinusitis which can be site threatening

- Intracranial infection – infective rhinosinusitis particularly of the frontal sinus can spread intracranially to cause meningitis and intracranial abscess formation

Rhinosinusitis

Definition

Inflammation of the nose and the paranasal sinuses characterised by two or more symptoms, one of which should be:

- Either nasal blockage/obstruction/congestion or nasal discharge
- (Anterior / posterior nasal drip):
- +/- facial pain/pressure
- +/- reduction or loss of smell

And either:

- Endoscopic signs of - Polyps, mucopurulent discharge, or oedema in middle meatus,

And/or:

- CT changes- Mucosal changes within the osteomeatal complex, or sinuses.

Classification

Rhinosinusitis can be classified into **Acute (ARS)** or **Chronic (CRS).**

- **Acute:**
    - < 12 weeks, complete resolution of symptoms.
    - ARS can be divided into viral, and non-viral.
- **Chronic:**
    - 12 weeks, without complete resolution of symptoms.
    - CRS can be divided into:
        - **CRS with nasal polyps**
        - **CRS without nasal polyps**

Acute Rhinosinusitis

Viral ARS (common cold) is caused mainly by Rhinovirus, and Influenza virus, normally with resolution of symptoms within 5 days.

Non-viral ARS is considered to be the persistence of symptoms after 5 days, caused mainly by super added bacterial infections such as Streptococcus pneumoniae, Haemophilusinfluenzae, and Moraxella catarrhalis.

Allergy and ciliary impairment can predispose to ARS.

Management

- Analgesia if required
- Nasal decongestants
- If persists longer than 5 days consider topical nasal steroids and oral antibiotics

CRS with or without nasal polyps

I. **Predisposing factors**

- Allergy: Atopy can predispose and is associated with CRS.
- Infections: Bacterial such as Staph aureus and streptococcus pnemonia, and fungal infections.
- Ciliary impairment; such as in Cystic fibrosis due to inability of cilia to clear mucus. Nasal polyps are present in about 40% of patients with cystic fibrosis.
- An anatomical abnormality, such as septal deviation and abnormal uncinate process leads to narrow infundibulum and occlusion of osteomeatal complex.
- Immunocompromised host
- Aspirin hypersensitivity.
- Atmospheric irritants: smoking, dusts, fumes.
- Hormonal: pregnancy and hypothyroidism, nasal congestion is high during pregnancy due to oestrogen and progesterone effect on nasal mucosal vascularity.
- Trauma: Surgical (oroantral fistula), nasal sinus fracture.
- Foreign body in the nose or sinuses
- Swimming and diving

Nasal polyps

A polyp is simply an abnormal mass which can be found in any part of the body. Nasal polyps associated with CRS are inflammatory polyps. They represent the extreme end of the spectrum of inflammation seen in CRS. They are normally bilateral and as long as there are no worrying signs from the history and examination do not require biopsy for histological diagnosis. All unilateral polyps require a biopsy for histological diagnosis.

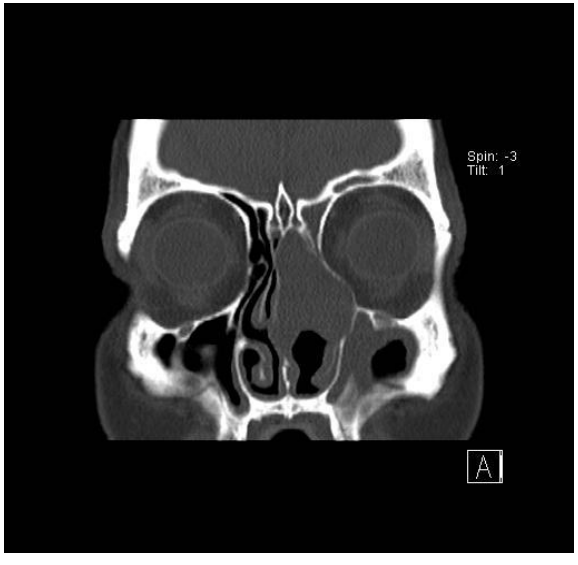
I. Investigations

- Skin prick tests if allergy suspected

- Radiology

    - Plain XR no longer used
    - CT Sinuses

        - If surgery planned
        - Atypical features to history or examination
        - Not good for diagnosis as large numbers of asymptomatic patients have changes in the sinuses on CT scanning



Coronal CT scan showing left sided rhinosinusitis with nasal polyps.

II. Management

For the majority of patients there is no cure for CRS and treatment is aimed at improving symptoms.

- Conservative
    - Avoidance of possible allergens
    - Nasal douching

- Medical

    - Antihistamines
    - Topical nasal steroids
    - Oral steroids(1 week course) in severe cases
    - Oral antibiotics

- Surgical treatment

    - Nasal polypectomy – very high rate of recurrence
    - Functional Endoscopic Sinus Surgery to improve ventilation/drainage of sinuses
    - Other procedures to improve nasal airways such as septoplasty, and reduction of inferior turbinates may be considered.

Allergic Rhinitis

Allergic rhinitis is an IgE-mediated, type 1 hypersensitivity reaction in the mucous membranes of the nasal airways. The disease is very common, affecting approximately 30% of the Western population and has a strong association with asthma. It can be either seasonal (summer hayfever) or perennial (sometimes with seasonal exacerbations). The commonest allergens include pollens, moulds, house dust mites and animal epithelia.

There is a new classification of **A**llergic **R**hinitis according to its **I**mpact on **A**sthma (**ARIA**), and other health issues. This is based on the **duration,** and **severity** of symptoms:

I. Duration of symptoms

- Intermittent: symptoms < 4 days per week, and less than 4 weeks
- Persistent: symptoms > 4 days per week, and more than 4 weeks

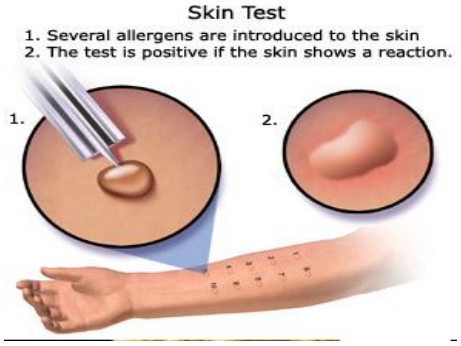
II. Severity of symptoms

- Mild: normal daily activities, and sleep. No troublesome symptoms
- Moderate to severe: Impairment of daily activities and sleep. Troublesome symptoms.

Allergic reaction leads to synthesis and release of arachidonic acid metabolites (prostaglandin D& leukotrienes) and mast cell degranulation to release histamine. The effect is to increase capillary permeability which leads to congestion, oedema, rhinorrhoea, sneezing and irritation.

III. Investigations

- Skin prick tests (SPT) for specific allergens
- RAST blood tests if SPT not possible



IV. Treatment

- Conservative
  - Allergen avoidance
  - Nasal douching
- Medical
  - Antihistamines
  - Topical nasal steroids
- Immunotherapy

Peri-orbital cellulitis & Orbital abscess

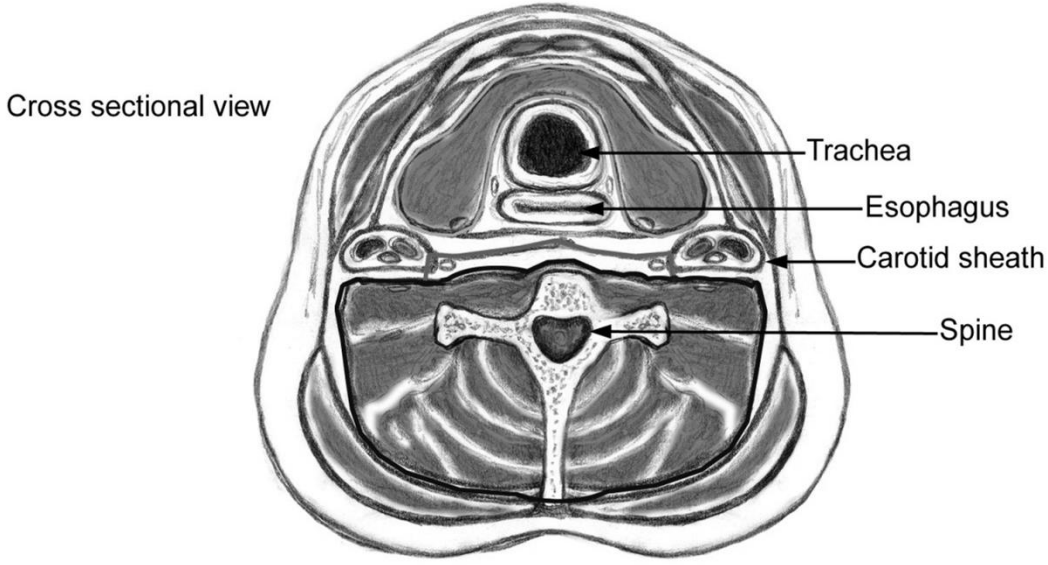
This is a site-threatening emergency which is more common in children and often results from direct spread of pus from the ethmoid sinus, or from thrombophlebitis of mucosal vessels in any of the sinuses. Patients develop pain followed by oedema of the eyelids and with orbital collection, the eye will become proptosed and eye movements will be reduced. There is a risk of blindness as a result of tension and septic necrosis of the optic nerve. Colour blindness is an early sign. A CT scan will confirm the collection and extent of the disease. Treatment with intravenous antibiotics, nasal decongestants, and urgent surgical drainage of any abscess is indicate.

The Neck

Learning Objectives:

- To revise the gross anatomy of the neck, including thyroid gland, salivary glands, pharynx and larynx.
- To understand common clinical conditions affecting the neck, thyroid gland, salivary glands, pharynx and larynx and their management.

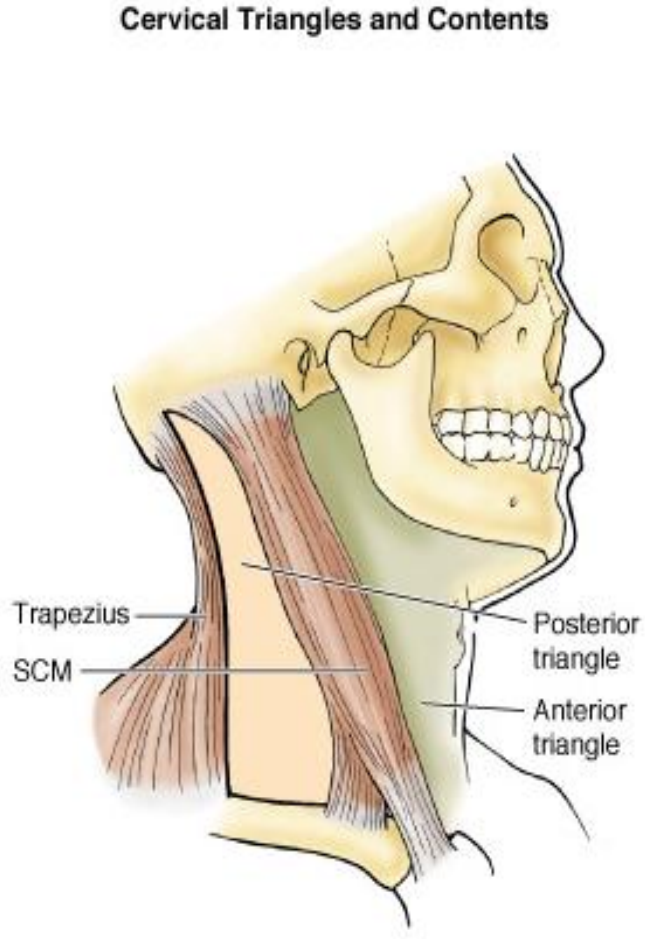
Anatomy of the Neck



Triangles of the Neck

There are a large amount of structures to consider within the neck.

The neck can be divided into anterior and posterior triangles on either side.

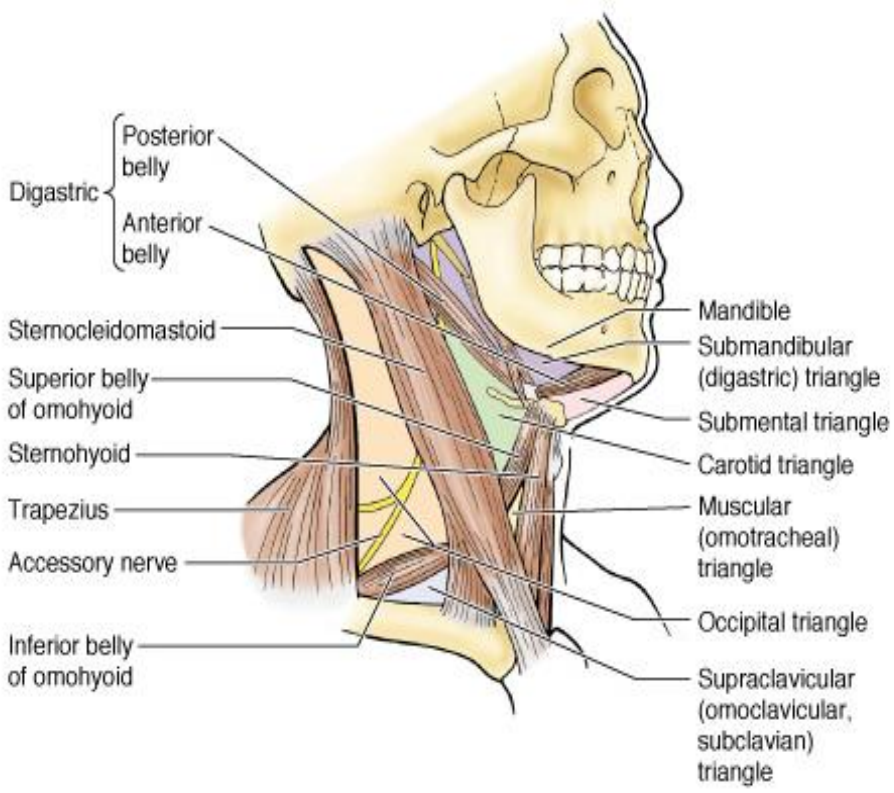


A. Anterior Triangle

- Boundaries
    - Medial – Midline of the neck
    - Lateral – Anterior border of sternocleidomastoid
    - Superior – Lower border of the mandible

B. Posterior Triangle

- Boundaries
    - Anterior – Posterior border of sternocleidomastoid
    - Posterior – Anterior edge of trapezius
    - Base – Middle third of clavicle



Neck Space Infections

Retropharyngeal abscess

Anterior to the prevertebral fascia, behind the pharynx, is a potential space, the retropharyngeal space, where an abscess may form. This space extends from the base of the skull to the mediastinum.

i. Clinical Features

- Commonly in young children, commonly after an URTI
- Neck held rigid and upright with reluctance to move
- Systemically unwell
- Airway compromise
- Dysphagia/Odynophagia
- Widening of the retropharyngeal space on lateral X-Ray
- Associated mortality due to airway problems & mediastinitis

ii. Investigations

- CT Neck

iii. Management

- Secure airway if any concerns
- IV antibiotics
- Surgery - Incision & drainage

Ludwig's Angina

Infection of the space between the floor of the mouth and mylohyoid, most commonly associated with dental infection.

i. **Clinical Features**

- Swelling of the floor of the mouth
- Painful mouth
- Protruding tongue
- Airway compromise
- Drooling

ii. **Investigations**

- CT neck
- OPG

iii. **Management**

- Secure airway if any concerns
- IV antibiotics
- Surgery to drain any collection

Parapharyngeal Abscesses

The parapharyngeal space is a potential space postero-lateral to the oropharynx and nasopharynx which is divided by the styloid process. Parapharyngeal abscesses may present in a similar manner to peritonsillar abscesses or "quinsy". There is typically a history of febrile illness, odynophagia, trismus, reduced neck movement and a swelling in the neck around the upper part of the sternocleidomastoid. The parapharyngeal space contains the carotid sheath, and there is therefore the risk of severe complications in this area.

Management

- Secure airway if any concerns
- IV Antibiotics
- Surgical drainage

Epiglottitis

This is mainly seen in children aged 2 - 6 and is an EMERGENCY.

i. **Cause**

- Normally Haemophylis influenza
- Incidence has reduced with introduction of HIB vaccine

ii. **Presentation** – rapidly progressive

- Stridor
- Drooling
- Pyrexia

iii. **Management**

- SECURE THE AIRWAY
  - The patient should not be examined as this may precipitate airway obstruction.
  - The child should be kept in a calm environment with their parents to avoid undue distress to the child that may also precipitate airway obstruction.

- If possible the child should be taken to theatre to be intubated by a paediatric anaesthetist with an ENT surgeon on standby to do a surgical
- airway if intubation is not possible. If transfer to theatre is not safe they should be intubated in Accident & Emergency where they normally present.
- iv antibiotics - Patients will normally respond after a couple of days of iv antibiotics after which they can be extubated

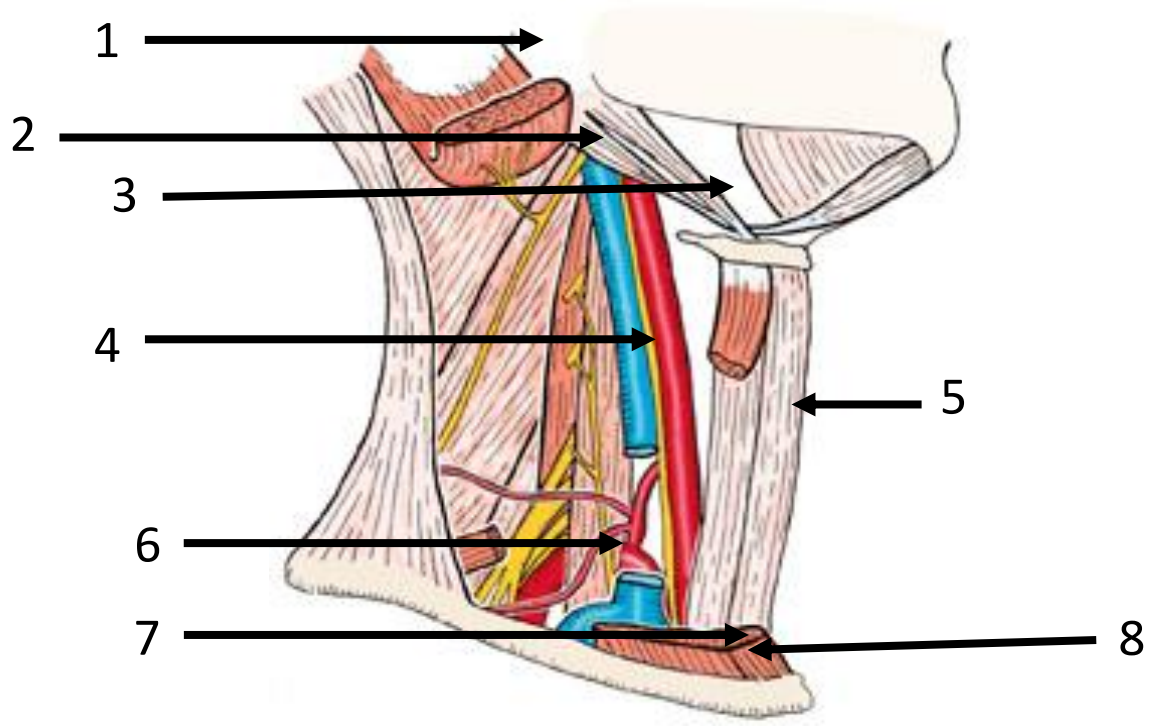
Neck Lumps

Despite the vast number of structures in the neck, the location of the lump can help significantly reduce the number of differentials

Typical lump locations and associated pathologies are shown below:

1. Parotid neoplasm - Anterior to the ear
2. Jugulodigastric – Angle of the mandible
3. Submandibular gland – Inferior border of mandible
4. Carotid body tumour or aneurysm – At bifurcation of common carotid artery
5. Thyroglossal cyst – In midline between hyoid bone and thyroid gland
6. Branchial cyst – Anterior border of sternocleidomastoid
7. Thyroid nodule – At level of thyroid
8. Virchow's node – Supraclavicular fossa node may represent metastasis

Neck masses should be investigated with an ultrasound guided fine needle aspiration (except for pulsatile masses).



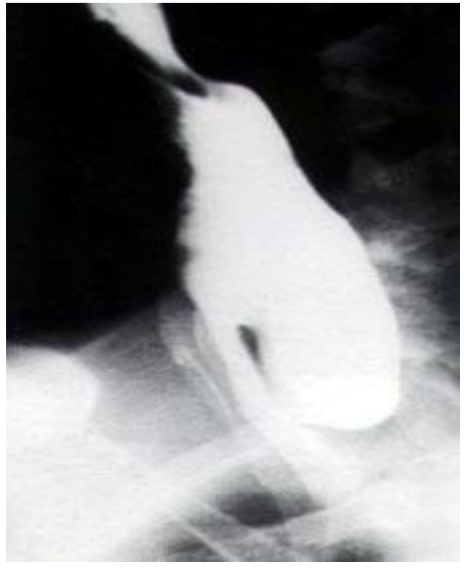
Anatomy of Pharynx and Oral Cavity

Dividing the pharynx and oral cavity into four separate entities allows surgeons to localise lesions and predict their behaviour depending on the surrounding anatomy.

The regions are divided as follows:

- Oral cavity – Extends from the lips to the posterior soft palate.
- Nasopharynx – Superiorly bound by the base of the skull, and inferiorly by an imaginary line at the level of the soft palate
    - Contents include the adenoids and eustachian tube opening
- Oropharynx – Extends from the level of the soft palate to the superior border of the epiglottis
    - Contents include the palatine tonsils, anterior and posterior tonsillar pillars
- Hypopharynx – Extends down from the superior border of the epiglottis to the inferior border of the cricoid cartilage. It is posterior to the larynx.

Unlike the rest of the GI tract where the muscular layers are arranged in circular and longitudinal layers, the pharynx has only a circular layer which is formed in principle by four muscles; Superior, middle, and inferior constrictors, and cricopharyngeus. Between inferior constrictor and cricopharyngeus, there is an area deficient of muscle at which herniation may occur. This is known as Killian's dehiscence, and is the site of pharyngeal pouch formation. Pharyngeal pouches typically present with dysphagia, delayed regurgitation of food and sometimes recurrent chest infections from aspirated food.



Barium swallow of pharyngeal pouch

In addition, three pairs of muscles cause elevation and depression of the pharynx - stylopharyngeus, salpingopharyngeus, and palatopharyngeus.

Obstructive Sleep Apnoea

Obstructive sleep apnoea and snoring can be considered as a spectrum of disease. Snoring is caused by partial obstruction to the upper airway during sleep from anywhere from the level of the nose to the larynx. In obstructive sleep apnoea there is a complete obstruction of the airway which requires the patient to wake up to alter their position in order to open up the airway again. Repeated apnoea in a night leads to a poor night's sleep and strain on the cardiovascular system. In children the commonest cause is adenotonsillar hypertrophy whereas in adults it is obesity.

Investigation

- BMI
- TFT – ?Hypothyroidism
- CXR – ?Signs of obstructive lung disease
- ECG – ?Signs of right ventricular failure
- Sleep study

Treatment

- Advice and lifestyle changes including weight loss
- CPAP – Continuous Positive Airway Pressure (mainstay of OSA treatment)
- Mandibular positioning devices in selected cases
- Surgery –adenotonsillectomy in children, rarely a treatment for adults

Tonsillitis

Tonsillitis is a common infection mainly seen in childhood and young adults.

Microbiology

i. **Bacterial**
   - Beta-haemolytic Streptococci
   - Staphylococci
   - Streptococcus pneumoniae
   - Haemophilus influenza
   - Escherischia coli

ii. **Viral**
   - Rhinovirus
   - Adenovirus
   - Enterovirus
   - Epstein-Barr virus (EBV)

iii. **Clinical Features**
   - Pyrexia
   - Dysphagia
   - Lymphadenopathy
   - Odynophagia
   - Trismus
   - Swollen tonsils with or without exudate
   - Otalgia (Referred pain)

iv. **Management**
   - Analgesia
   - Antibiotics
   - Drainage of any peritonsillar abscess
   - Tonsillectomy for recurrent tonsillitis

Treatment with amoxicillin should be avoided as this will cause a maculopapular rash in the presence of EBV (glandular fever). Furthermore, patients with EBV should be advised to avoid contact sports for 2-3 months as the virus commonly causes hepatosplenomegaly.

Infection may also be complicated by a peri-tonsillar abscess (quinsy).

These normally present with a sore throat that is lateralised to one side and a "hot potato" voice. They may be amenable to drainage under local anaesthetic.

Head & Neck Cancer (excluding thyroid and salivary gland)

Clinical Features/Presentation

- Dysphonia (esp laryngeal malignancy which may cause hoarseness)
- Dysphagia/odynaphagia
- Dyspnoea –stridor from narrowing of airway, especially laryngeal tumours
- Neck Mass
- Pain from site of pathology or referred e.g. to ear
- Bleeding from nose or mouth depending on site of primary(rare presentation)
- Nasal blockage –normally unilateral progressive for nasal/nasopharyngeal pathology

Histopathology

The majority (90%) are squamous cell carcinomas.

Epidemiology

Men are affected twice as much as females.

Risk Factors

Alcohol

Tobacco

Beetle nut chewing for oral cavity malignancies

Chinese ethnic origin for nasopharyngeal malignancy

Investigations/Staging

The purpose of the investigations in head and neck cancer is to get a histological diagnosis and to stage the disease. From this information a management plan can be formulated and a decision made as to whether any treatment given as part of this management plan will be with a curative intent or for palliation only.

The most commonly used staging system in Head & Neck cancer in the UK is the TNM staging system which is also commonly used in other parts of the body.

T= Tumour size, 1 (small) – 4 (big)

N= Nodal Metastasis, 0 (no nodes) - N3 (big/multiple nodes)

M= Distant metastasis 0 (none) – 1 (present)

The higher the number of each of these components for the TNM system, the worse the prognosis. Patients with distant metastases are not curable and should therefore only be treated with palliation in mind. Patients without distant metastases can potentially be cured but as mentioned the higher the T&N number the worse the prognosis and also associated morbidity from treatment so all head and neck cancer patients should have their management discussed at a multidisciplinary meeting with the patient's co-morbidity and personal views in mind.

i. **Investigations of primary tumour site**
   - Examination under anaesthetic
     - Also called Panendoscopy or laryngopharyngo-oesophagoscopy in H&N
     - Purpose – Biopsy for histological diagnosis, assess size of tumour, look for second primary
   - CT Neck
     - Purpose – assess size of tumour and neck node metastasis

ii. Investigation of neck metastasis

If a patient has a biopsy proven primary squamous cell carcinoma in the head and neck it can be assumed that any palpable cervical lymphadenopathy is secondary to this primary and the CT scan of the neck to assess the primary will give all the information required for staging. If this is not the case any palpable neck masses should be investigated with an ultrasound guided fine needle aspiration (FNA). While blind (i.e. no US) FNA can be done the ultrasound will increase the accuracy of the specimen taken and will give extra information on the mass being sampled.

Sometimes patients with head and neck squamous cell carcinoma may present with neck metastases with no obviously primary even after examination under anaesthetic and imaging. These should be diagnosed with a FNA of the neck mass.

While it is possible to do an open biopsy on these lymph nodes this can lead to seeding of the tumour through the wound onto the skin and end up in a worse prognosis for the patient. Although FNA is very good at diagnosing squamous cell carcinoma it is not so good for other causes of cervical lymphadenopathy such as tuberculosis and lymphoma which often require an open biopsy so in these cases an FNA should be carried out to exclude SCC before an open biopsy is done.

iii. Investigation of distant metastasis

CT chest are routinely done to check for metastasis in the chest, the commonest site of distant metastasis.

Other investigations are not routinely done unless there is suspicion of other distant metastasis.

Management

(i) Palliation

Treatment should be aimed at reducing suffering and in some cases prolonging life. This may involve chemotherapy and radiotherapy and occasionally surgery.

iv. Curative

Treatment needs to be aimed at the primary site and also any neck metastases. In some head and neck cancers there is a high rate of metastasis to the neck so even if no neck metastasis are detected treatment will still be given to the neck. The possible treatment options are

- Radiotherapy – to the primary site +/- to the neck, +/- chemotherapy
- Surgery
    - Endoscopic – e.g. laser resection
    - Open surgery
        - To primary site – e.g. laryngectomy
        - To neck – i.e. neck dissection

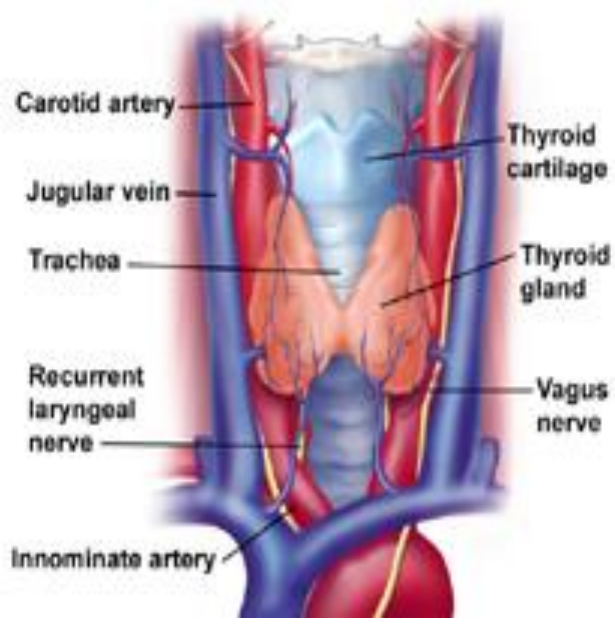
The Thyroid Gland

A goitre is an enlarged thyroid gland. A thyroid nodule is an abnormal mass in the thyroid. Most nodules are not true neoplasms. Thyroid nodules need to be investigated to check that they are not hyperfunctioning nodules causing hyperthyroidism and that they are not neoplastic. Large goitres may also cause compressive symptoms such as airway obstruction and dysphagia.

Anatomy

The thyroid gland is located at the base of the neck. It is a highly vascular structure responsible for controlling the basal metabolic rate of the body through the production of thyroxine and triiodothyronine hormones.

The arterial supply to the thyroid is from the superior and inferior thyroid arteries. Venous drainage of the thyroid is via the superior, middle, and inferior thyroid veins. The recurrent laryngeal nerves are intimately associated with the thyroid gland running in the tracheo-oesophageal groove and are at risk during surgery. They supply the muscles of the larynx (except cricothyroid) and sensation below the vocal cords. Injury causes vocal cord palsy with associated hoarseness and can also give problems with airway obstruction if the nerves on both sides are injured.



The parathyroid glands are also closely related to the thyroid gland. They are involved in calcium haemostasis and can be accidentally removed or damaged during surgery leading to hypocalcaemia. This is normally only a problem when both sides of the thyroid have been operated on. i.e. total or completion thyroidectomy.

Investigations

i. Thyroid function tests
ii. Ultrasound guided fine needle aspiration

Fine needle aspiration is a very good way of assessing thyroid nodules but like most tests is not 100% accurate so the information has to be taken in conjunction with the history and examination. If there is any diagnostic doubt a hemithyroidectomy should be carried out to give definitive histology. Lumpectomies are not done on the thyroid because if it is a malignant nodule this will not give adequate margins on the mass and it would also make further surgery very difficult due to scarring and put the recurrent laryngeal nerve at an unacceptably high risk. Fine needle aspirations are not reliable at differentiating a follicular carcinoma from a follicular adenoma and in these cases a hemithyroidectomy should be carried out for definitive histology.

Histopathology

i. **Non – neoplastic nodules**
- Single nodule – colloid, cystic
- Multinodular goitre
    - These are common and if they show typical features on ultrasound do not necessary require fine needle aspiration. Any dominant nodule should have an FNA.

ii. **Thyroid Neoplasms**
- Benign
    - Adenoma – Mainly follicular
- Malignant
    - Papillary adenocarcinoma – 70% - Often seen in younger patients, or where there is a history of irradiation of the neck.
    - Follicular carcinoma – 20% - It has a preponderance to metastasis to the bones and lungs.
    - Medullary carcinoma – 5% - This is a neoplasm of the calcitonin regulating C-cells. It is typically seen in multiple endocrine neoplasia, and therefore screening of other organs involved in MEN syndromes is required. There is a genetic component.
    - Anaplastic carcinoma - ~5% - Typically seen in older patients. This has a poor prognosis and by the time of diagnosis the prognosis if often a matter of weeks to months.

Management

i. **Non –neoplastic nodules**
- Conservative –these can be managed conservatively unless there is diagnostic uncertainty
- Surgery
  - For compressive symptoms, cosmesis or patient preference.
  - Should aim to restrict surgery to hemithyroidectomy due to the increased morbidity from a total thyroidectomy and the need for lifelong thyroxine replacement.

ii. **Neoplastic nodules**
- Adenomas – require no further treatment after diagnostic hemithyroidectomy
- Carcinoma
  - Surgery
    - Total Thyroidectomy for papillary, follicular and medullary carcinoma
    - Anaplastic disease is normally too far advanced for curative surgery
  - Radio-iodine therapy
    - For papillary and follicular carcinoma after surgery

Complications of thyroid surgery include

- Post-operative haemorrhage
- Airway obstruction
  - Secondary to haemorrhage
  - Secondary to bilateral vocal cord palsy
- Vocal cord palsy
- Hypocalcaemia

Salivary Glands

Parotid Gland

i. **Anatomy**

The parotid gland is a serious salivary gland located anterior to the pinna and lateral to the ramus of the mandible. The gland is split into deep and superficial lobes by the facial nerve which passes through the gland. The majority of the gland lies superficial to the facial nerve. Facial palsy is one of the most serious risks from parotid surgery and can also be caused by a parotid malignancy.

ii. **Clinical Features**
- The parotid duct opens opposite the second upper molar tooth after piercing the buccinator muscle and the buccal mucosa.
- 80% of salivary gland neoplasms occur in the parotid gland.
- 80% of parotid neoplasms are benign in nature.
- 80% of benign parotid neoplasms are pleomorphic adenomas.
- Compared to the submandibular glands, neoplasms are 9 times more common in the parotid gland.
- Infections however, are 9 times less common in the parotid gland than the submandibular gland.

Submandibular and Sublingual Glands

i. **Anatomy**

The submandibular gland is located inferior to the body of the mandible and superior to the digastric muscle. It's duct opens into the mouth close to the frenulum of the tongue. The hypoglossal and lingual nerves run medial to the gland and are at risk during submandibular gland surgery. Unlike the parotid gland, the submandibular gland has mixed mucous and serous secretions. The sublingual glands are entirely mucous and are located in the floor of the mouth.

ii. Clinical Features

- 50% of neoplasms occurring in the submandibular gland are malignant.
- Infections in the submandibular gland are 9 times more common than those in the parotid gland.
- Submandibular abscesses may develop following sialolithiasis or sialadenitis
- 80% of sublingual gland neoplasms are malignant

Salivary Gland Pathology

i. Acute Sialadenitis

Acute sialadenitis may be viral or bacterial. Bacterial disease is typically due to staphylococcal infection and is typically seen in dehydrated or immunocompromised individuals. Viral disease is caused by a range of viruses including:

1. Paramyxovirus – Mumps
2. Coxsackievirus
3. Echovirus
4. HIV

Chronic sialadenitis is rare, and sometimes seen in TB, sarcoidosis, HIV, and syphilis.

ii. Sialolithiasis

Stones in the salivary duct cause obstruction and subsequently lead to pain and swelling which is worse during meals. Stones are 9 times more common in the submandibular gland than the parotid.

Investigations

- Ultrasound
- or sialogram

Management

- Conservative
  - most will settle conservatively
  - analgesia
  - hydration
  - sialogogues
- Endoscopy
- Radiological removal
- Surgery
  - Intraoral removal of palpable stones
  - Removal of salivary gland

Complications

- Sialadenitis
- Abscess formation

iii. Sjögren's Syndrome

This is an autoimmune disease causing lymphocytic infiltration into the ductal tissue of secretary glands. It classically presents with dry eyes and dry mouth and patients may have enlarged salivary glands. Patients are at increased risk of developing lymphoma.

The disease may be primary:

Xerostomia and xerophthalmia without connective tissue abnormality.

Or secondary:

As primary disease, with connective tissue disease, most commonly rheumatoid arthritis.

Diagnosis is made from the history, examination, some specific autoantibodies and a biopsy of minor salivary glands on the inner lip.

ENT Case Study Answers

	Q1	Q2	Q3	Q4	Q5	Q6
1	Otitis Externa	Pseudomonas aeruginosa, staphylococcus aureus	Avoid water which may be unsanitary. Flying is unlikely to be a problem unless she has a cold at the time or has had previous pain on flying	Ear canal probably narrowed due to inflammation of the lining skin. Tympanic membrane may be inflamed or difficult to see due to debris	Use of cotton buds. Swimming, humid conditions. Weakened immune system.	
2	Trauma to the ear canal. Impacted wax Trauma to the tympanic membrane	Can lead to the above conditions	Otoscopy. Pure tone audiogram. Keep the ear dry. No more cleaning. No eardrops. Review in 4 weeks.			
3	Acute otitis media	Chronic otitis media with effusion	Bulging and red.	Acute mastoiditis	Staphylococcus aureus, Strep pnuemoniae, Haemophilus influenza	Facial nerve Inner ear Sigmoid sinus Brain
4	Cholesteatoma	Debris causing a conductive hearing loss or erosion by cholesteatoma of the ossicles.	Conductive hearing loss with Weber towards the right ear and Rinne negative on that side	Disease recurrence with breach of the otic capsule bone causing dizziness on pressure change transmitted from the middle ear to the vestibular system		
5	Benign Paroxysmal Positional Vertigo	How long has he had it How often are episodes How long do the episodes last What are the specific triggers Has he had it before and if so did it resolve spontaneously or require treatment Any hearing loss or tinnitus associated Any loss of consciousness with episodes	Dix Hallpike test Expect to see (when the affected ear is undermost) Latency Torsional geotropic nystagmus Fatigue Habituation on repeating the test	Epley manoeuvre		
6	Septal haematoma followed by erosion of the septal cartilage	Anterior rhinoscopy and palpation of the bulging septum	Cocaine. It causes loss of blood supply to the cartilage of the septum			

7	Nasal steroid sprays, possible nasal steroids if polyps are present. Long course of antibiotics – at least 6-8 weeks	Bleeding, infection, CSF leak, visual loss or disturbance.				
8	Arrest epistaxis. Evaluate for shock. Take measures to restore circulating blood volume.	Look at the posterior wall of the oropharynx by shining a light in the mouth and using a tongue depressor. If there is fresh blood running down the wall he will require removal of the anterior packing and either a post nasal pack or a nasopharyngeal balloon followed by further anterior nasal packing	He would require a general anaesthetic either to pack more effectively or to cauterise the bleeding point or ligation of the sphenopalatine artery and possibly the anterior ethmoidal artery.			
9	It is in two parts – superficial and deep to the posterior border of the mylohyoid muscle in a U fashion. The larger superficial part is in the digastric triangle and partly deep to the mandible. Is is also covered superficially by the platysma and skin. The deep part is between mylohyoid and hyoglossus. The duct runs from the deep part and is crossed by the lingual nerve. The hypoglossal nerve lies deep to the gland. The cervical branch of the facial nerve runs superficial to the gland and can be damaged in a high skin/platysma incision.	Bleeding infection, damage to the lingual or hypoglossal nerves or cervical branch of the facial nerve.				
10	Parotid	Facial nerve paresis suggests malignancy. Reinforced by the lymph nodes	Freys syndrome	Accessory nerve Shoulder drop		
11	Thyrotoxic with a thyroid nodule	On clinical examination a thyroglossal cyst will move upwards on tongue protrusion. A thyroid nodule would move upwards on swallowing.	Thyrotoxic storm and a higher risk of bleeding.	Bleeding, infection, injury to the recurrent laryngeal nerves, hypothyroidism, hypoparathyroidism	The nerves branch off the vagus nerves in the neck. The eft side loops under the aortic arch, the right loops under the right subclavian artery. Both then pass upwards to supply the laryngeal muscles passing deep to the thyroid and entering the larynx posterior to the cricothyroid joint.	Calcium. Tingling around mouth and fingertips. If severely decreased muscle spasm occurs.

Dermatology: Student Workbook

Learning Objectives and Aims

By the end of the course, you should be able to

- Take a Dermatological history
- Explore a patient's concerns and expectations
- Examine skin, hair, nails and mucous membranes systematically showing respect for the patient
- Describe cutaneous physical signs in skin, hair, nails and mucosae
- Record their findings accurately in the patient's records
- Interact sensitively with people with skin diseases

Important Learning Objectives in Dermatology

These are detailed in the following document BAD's Undergraduate Dermatology Curriculum:
http://www.bad.org.uk/shared/get-file.ashx?itemtype=document&id=4168

They are mostly covered in this workbook, and can be summarised under the following headings:

- Background knowledge
- Skin Failure and Emergency Dermatology
- Skin Infections and Infestations
- Common and Important Problems
- Skin Tumours
- Skin Signs of Systemic Disease
- Preventative medicine
- Management and Therapeutics
- Clinical Skills

The above learning objectives will be achieved by:

- ✓ Interacting with patients attending clinics, minor ops lists and phototherapy
- ✓ Discussing patients with Dermatology doctors and nurses
- ✓ Attending Seminars
- ✓ Bedside teaching
- ✓ Self-study and completion of workbook

A warm welcome to Dermatology! We are very proud to have secured a week of Dermatology for all medical students in Leicester since September 2018. We have a passion for teaching, so please be proactive and help us look after our patients so that you can learn as you do so. If you would like to do a different activity to the one you have been assigned to, please ask one of our doctors and nurses in clinic to see if they can accommodate your request. If you have any questions do not hesitate to ask. We hope you enjoy your time in Dermatology.

Meet the Dermatology Team

Burton	**Consultants** Dr Georgina Elston Dr Sheena Goyal Dr Naomi Hutchinson Dr Mahmood Afsari Dr Rabi Nambi Dr Harriet O'Neill	
Kettering	**Consultants** Dr Olivia Stevenson Dr Taseer Bhatt	
Northampton	**Consultants** Dr. Pick Woo, Dr. Kazeem Salako Dr. Dina Ismail Dr. Rajesh Goel Dr. Christine Soon Dr. Shruthi Dhir	**Specialist Trainees** Dr. Mohammad Elnaggar Dr. Andrew Hart Dr. Elizabeth James Dr. Amar Lally

Leicester	**Consultants** Dr Karen Harman, Head of service Dr Elizabeth Roberts, Head of service Dr Ingrid Helbling, Dermatology UG Lead Dr Graham Johnston Dr Manisha Panchal Dr Matthew Scorer Dr Akhtar Rasool Dr Alex Gan Dr Rushqia Mukhtar Dr Mo Aldiwani Dr Andrew Sharpe	**Specialist Trainees** Dr Daryl Teo Dr Mariam Abu Jubain Dr Fiza Ahmed Dr Linda Svesden Dr Maryam Barfei Dr Nikolaos Georgiou Dr Luca Borg Dr Khansa Ashfaq Dr Neema Azeez Dr Chirag Patel Other Dermatology Clinical Teachers: Dr Asma Farooq Dr Abdullahi Mukhtar

You will meet experienced dermatology nurses and HCAs at all hospitals who can further support your learning.

Attendance at Clinics and Teaching Sessions

Clinical experience in the Dermatology Departments is the best way of learning dermatology. You will only achieve the essential learning objectives in Dermatology if you talk to and examine patients with skin problems.

You will meet people with the common skin conditions that you may have to manage as a Foundation doctor. Whenever we have space, we will let you see your own patients, so please ask us!

Please set your own learning objectives for each clinic and discuss these with a doctor or nurse at the start of the session.

You should also spend time with the nurses in the Phototherapy Unit.

Teaching Sessions

You are expected to attend and add to MKM all timetabled teaching unless you have discussed reasons for absence with the Undergraduate Coordinator.

You will be assessed on the following

- Attendance and professional behaviour throughout the week
- Interaction with patients
- Satisfactory description of rashes/lesions
- Satisfactory diagnostic and management skills of common skin conditions
- Log of each clinic or activity you attend

Taking a Dermatological history

Using the standard structure of history taking, below are the important points to consider when taking a history from a patient with a skin problem:

For any pigmented lesion, pay attention to questions marked with an asterisk (*).

Main Headings	Key questions
Presenting complaint	Nature, site and duration of problem
History of presenting complaint	Initial appearance and evolution of lesion* Symptoms (e.g. itch and pain)* Aggravating and relieving factors Previous and current treatments (effective or not) Recent contact, stressful events, illness and travel History of sunburn and use of sunbeds* Fitzpatrick skin type
Past medical history	History of atopy i.e. asthma, allergic rhinitis, eczema History of skin cancer and suspicious skin lesions
Family history	Family history of skin disease*
Social history	Occupation (including skin contacts at work) Improvement of rashes when away from work
Medication and allergies	Regular, recent and over-the-counter medications
Impact on quality of life	Impact of skin condition and concerns

Examining the skin

1. Adequate exposure and good lighting are essential – Dermatology is a visual specialty!
2. Examination should include hair/scalp, nails and mucous membranes
3. Comment on morphology / how individual lesions look:
   - small lump (<5mm) → **papule**
   - larger lump (5-10mm) → **nodule** (solid raised lesion with a deeper component)
   - redness → **erythema**
   - scaling → **scaling**
   - small water blister → **vesicle**
   - larger water blister → **bulla**
   - pus-filled vesicle → **pustule**
   - thread vein → **telangiectasia**
   - hair loss/ thinning → **alopecia**
   - hairiness → **hirsutism**
   - scratch marks → **excoriations**
   - stretch marks → **striae**
   - itching → **pruritus** (not pruritis)

➢ thinning → **atrophy** ➢ non palpable area of discoloration → **macule** ➢ macule > 2cm → **patch** ➢ palpable, flat topped area > 1-2cm → **plaque** ➢ loss of epidermis (superficial) → **erosion** ➢ loss of epidermis and dermis (deep) → **ulcer** ➢ thickening of the skin with exaggerated skin markings → **lichenification**
4. Comment on distribution and sites involved. Distribution (the pattern of spread of lesions): ➢ Generalised → All over the body ➢ Widespread → Extensive ➢ Localised → Restricted to one area of skin only ➢ Flexural → Body folds i.e. groin, neck, behind ears, popliteal and antecubital fossa ➢ Extensor → Knees, elbows, shins ➢ Pressure areas → Sacrum, buttocks, ankles, heels ➢ Dermatome → An area of skin supplied by a single spinal nerve ➢ Photosensitive → Affects sun-exposed areas such as face, neck and back of hands
5. Palpate!
6. Examine other systems if appropriate e.g. Joints, lymph nodes

Dermatology Case Studies

Case Study 1

Mr Robert Andrews is a 72-year-old man who presents with his wife to the dermatology clinic today because the GP has referred him for a lesion on his face under the 2 Week Wait pathway.

He explains the lesion on his forehead has been present for the last six months or so, but it has grown a lot in size since then. Sometimes it bleeds and he also feels it can be painful. He has “a lot” of moles. In the past, he has had a couple of skin lesions from his face and ear removed but was told they were not cancerous and not anything to worry about. His wife tries to check his skin regularly but finds it difficult because he has so many moles. His other past medical history includes hypertension and Type 2 DM. He has no known drug allergies. His regular medication consists of Ramipril and Metformin.

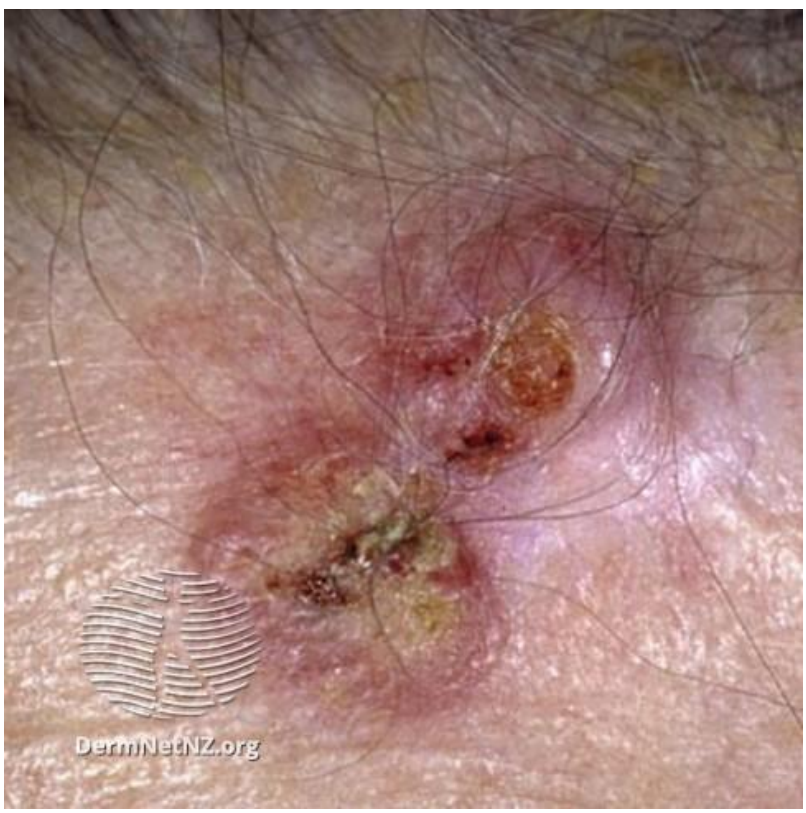
He has no family history that he can recall. He is a retired accountant but explains he is originally from New Zealand and reports getting sunburnt a lot as a child and young adult. He currently enjoys gardening in his spare time but always remembers to wear Factor 50 and a hat – his wife is very good at reminding him to do so. He used to smoke quite a lot but quit smoking five years ago. He does not drink any alcohol currently as he is on a diet (BMI 28). He has managed to lose three stones successfully over the last four months.

He is already aware that this lesion will probably require removal like his previous lesions. Last time he had a routine operation booked that was cancelled once, so he ended up waiting three months for his surgery. He is

planning to go away with his wife for their 50th wedding anniversary in one month's time and would like to know if this operation can be done afterwards.

1. What questions would you like to ask him?

2. Describe the lesion on the below picture?



3. What other investigations would you like to carry out as part of your examination?

4. What would be your clinical diagnosis? Please provide your reasoning behind this.

5. What would be your management plan?

6. What do you think you should explain to the patient at this stage?

Case Study 2

Mrs Eileen Rogers is an 82-year-old woman who presents to the dermatology clinic today because the GP has referred her for a lesion on the skin above her left upper lip on a routine basis.

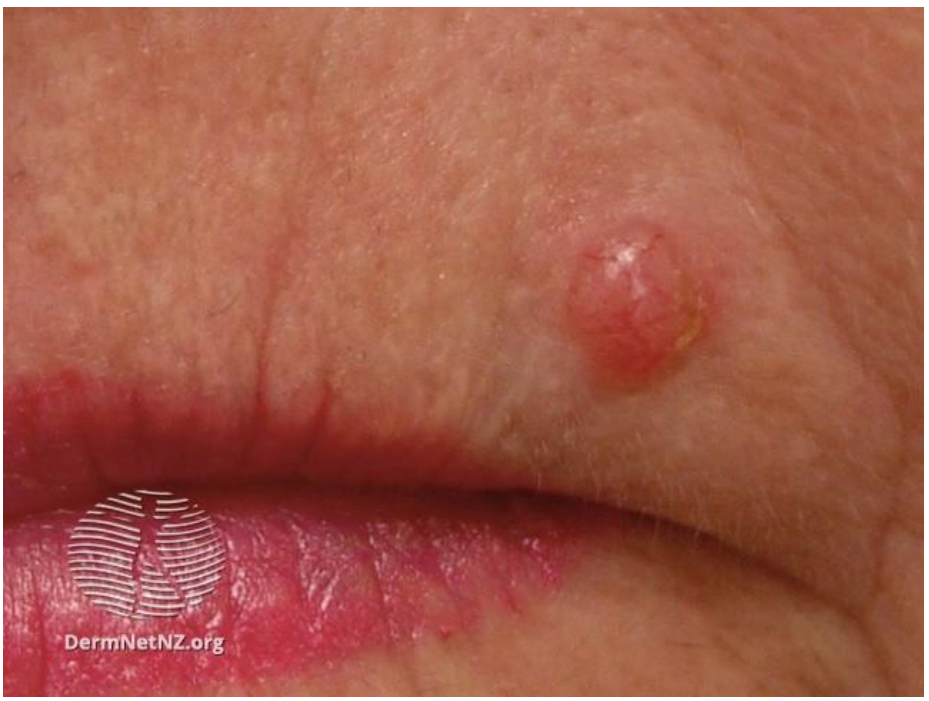
She has explained that she has had the lesion for a number of years. She does not think it's changed in size recently but thinks it may have grown a little over the last few years. It usually does not give her any issues apart from when it occasionally bleeds. She does not have any other skin problems. Her past medical history includes Congestive Cardiac Failure with reduced Ejection Fraction, COPD, Type 2 Diabetes, Osteoarthritis and Hypertension. She has forgotten to bring her list of regular medications to clinic today but informs you the list is very long and she relies on her dossette box to remember everything. She has no known drug allergies.

She is not too sure of her family history but thinks her mother had skin cancer because she had to get a large lump removed out of her leg many years ago. She is a retired schoolteacher and was born in the UK. She does not think she spends a lot of time outdoors but enjoys taking her grandchildren to the park multiple times a week. She cannot remember the last time she got sunburn because she always remembers to wear sunscreen. However she thinks she got sunburn a lot as a child because no one used sunscreen back then. She used to smoke a packet of cigarettes every day but quit ten years ago. She spends her days looking after her grandchildren and more recently, her frail husband who has Alzheimer's dementia.

Her oldest grandchild is a medical student and advised her to see her GP about the lesion on her face to make sure it wasn't cancer, especially since she has a positive family history. The GP explained she was referring her urgently and less than two weeks later, she is now here in clinic. As a result, she is very anxious about the fact that she may have skin cancer as she is worried about having to undergo surgery, having previously been told she is a high-risk patient for surgery. She is also worried about the amount of time this will take, especially as she has a lot of carer responsibilities.

1. What questions would you like to ask her?

2. Can you describe the lesion on the below picture?



3. What other investigations would you like to carry out as part of your examination?

1.

4. What would be your clinical diagnosis? Please provide your reasoning behind this.

5. What would be your management plan?

6. What do you think you should explain to the patient at this stage?

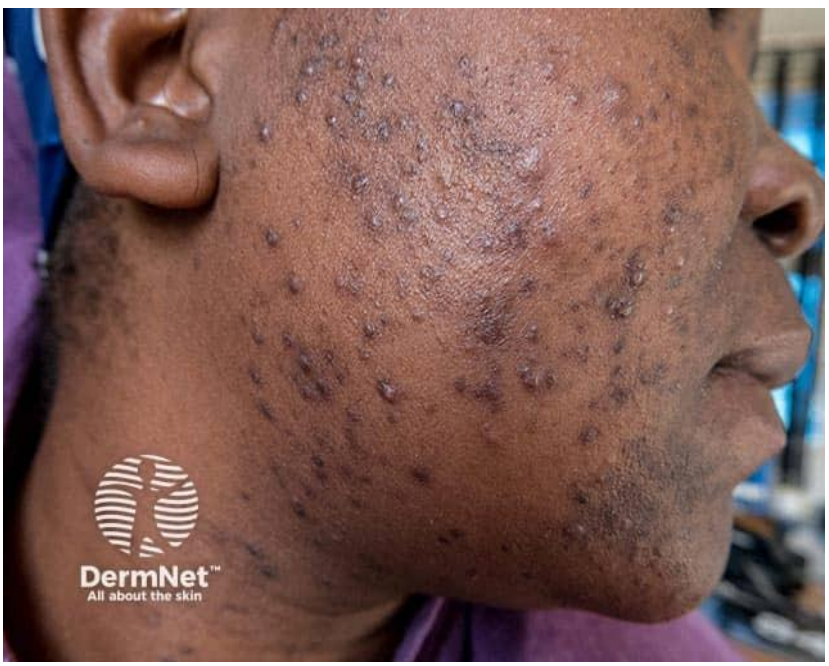
Case Study 3

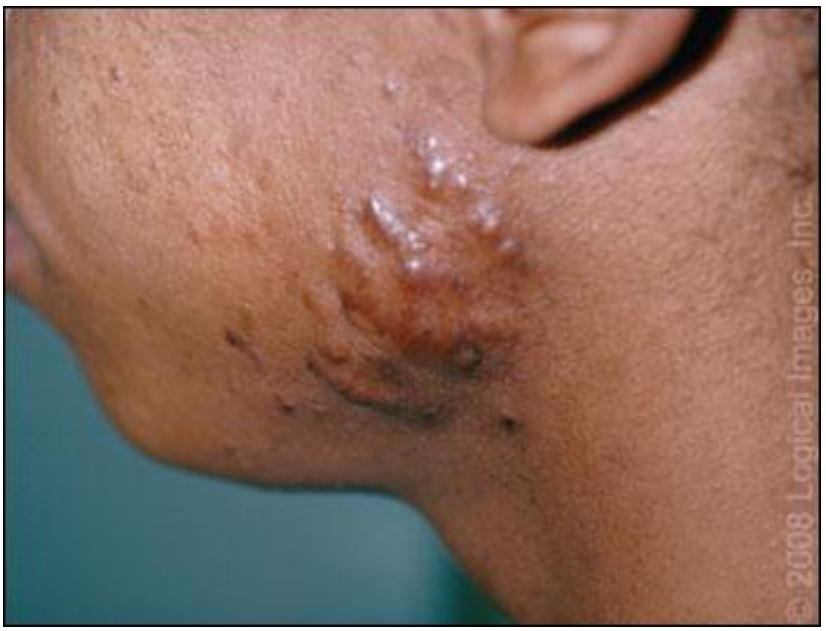
Jamile Clarke is a 15-year-old boy who presents to his GP with his mother. He reports that his acne is getting out of control and would like something to be done about it.

He has had acne since he started secondary school. His face has never been completely clear since the age of eleven but over the last two years, he has noticed that he gets spots on his chest and back too. Some of the spots last for months. More recently he has noticed "little holes" in his face which he thinks is as a result of scarring from his acne. In the past, he has endured some bullying from other pupils at school because of his acne. Therefore, if he has a really bad day, he refuses to leave the house and has missed a few days of school as a result.

He has no past medical history of note and no known drug allergies. He is not on any regular medication. He reports that his mother also had very severe acne which lasted up until her mid-twenties. It only went away after his mother was given a course of Roaccutane. His older sister is currently on Roaccutane for acne, which has helped immensely.

He has come today because she has had enough of his acne and would therefore like to be started on the drug that is Roaccutane so that he can get rid of her acne for once and for all.

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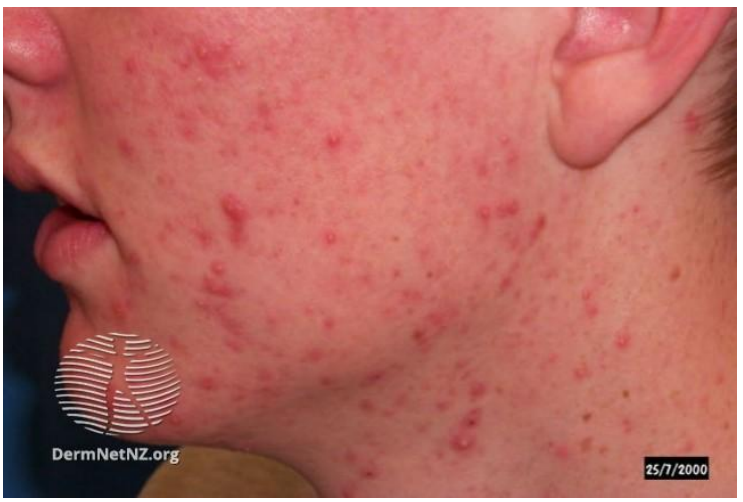
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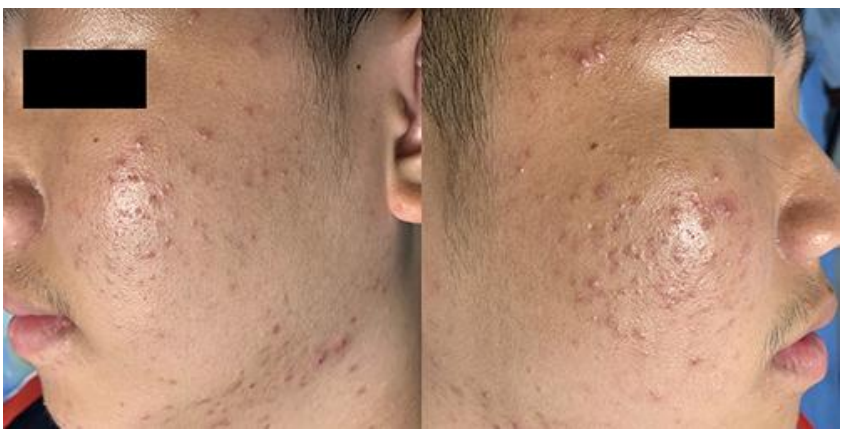


1. What questions would you like to ask?

2. What initial treatment would you like to start the patient on at the moment? (Think about what treatments you can offer as a GP)

3. What further treatments are there available for acne? (Think about what can be offered in secondary care)

4. What is oral isotretinoin (Roaccutane) and what factors must we consider when starting a patient on this medication?

5. What do you think you should explain to the patient at this stage?

Case Study 4

Timothy Charles is a two-year-old boy brought into the GP surgery by his mother who is worried about the rash on his legs.

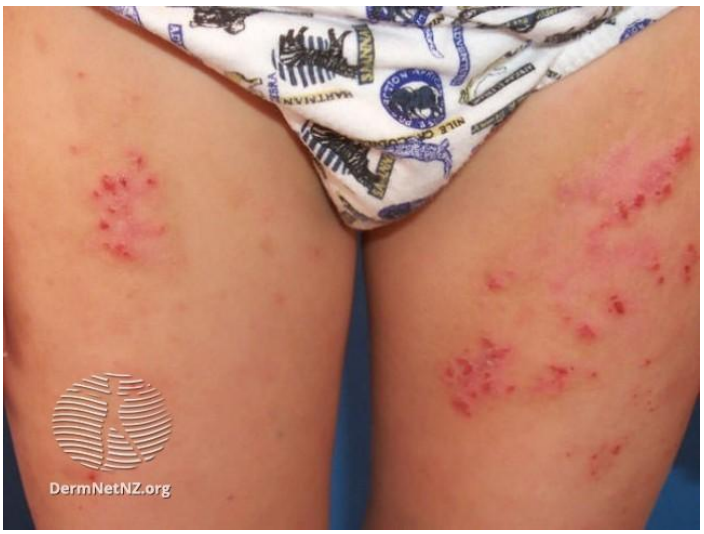
She noticed it started a few weeks ago but has gradually gotten worse and she is worried about how Timothy won't stop scratching as sometimes the rash bleeds. She has also noticed that he has become more irritable than usual and worries that he is not getting enough sleep at night because he is scratching. She recently tried a new baby bath oil and was worried he had had an allergic reaction to that. However she stopped using it last week and the rash has not improved. Timothy has no past medical history of note and no known drug allergies. He was born at term via Caesarean section and there were no complications during the pregnancy or at birth. There are no concerns about Timothy's development.

Mother has a history of hay fever herself and Timothy's older sister has recently been diagnosed with asthma. Timothy lives with his sister and both his parents at home. They have no pets due to the father's multiple allergies. Neither parent smokes in the household.

She is worried about how severe the rash has gotten and how much it is affecting Timothy. She would like to know if anything can be done for it and whether he needs special investigations such as allergy testing. Finally she wants to know if this will get better or not as she is worried that Timothy will get teased by other children when he starts nursery in a few months' time.

1. What questions would you like to ask?

2. Can you describe the rash on the below pictures?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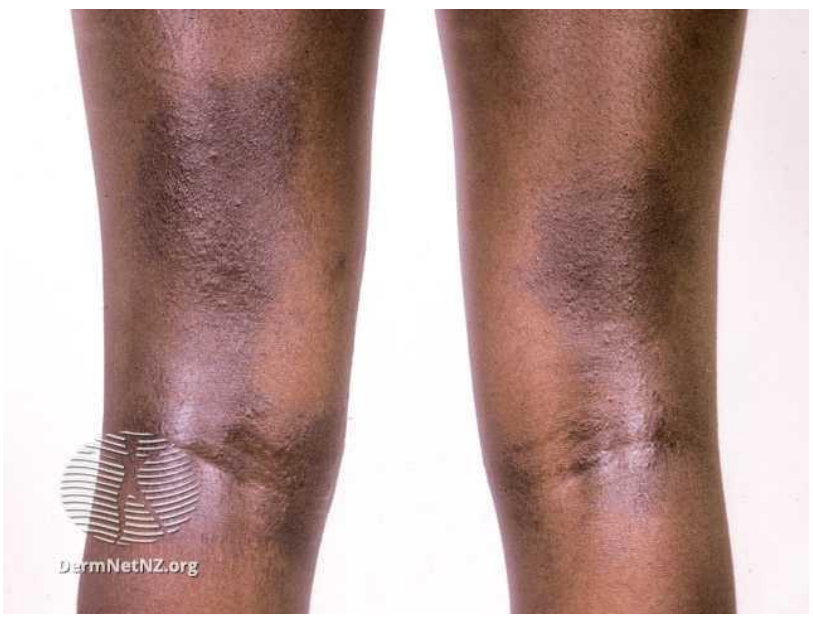
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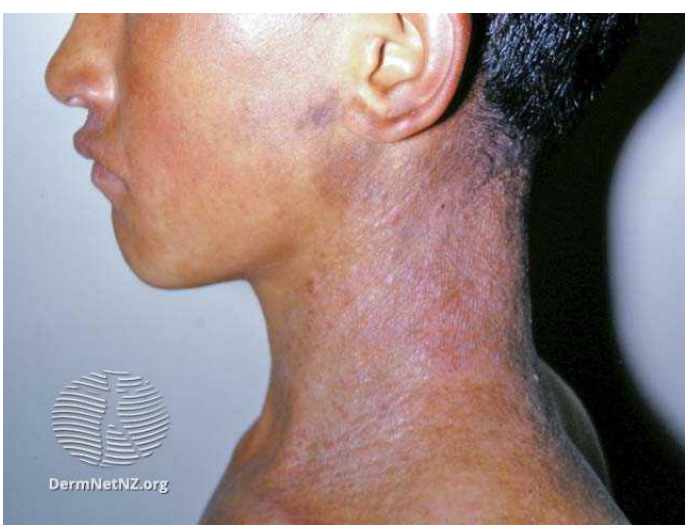

DermNetNZ.org

3. What would be your clinical diagnosis and why?

4. What would be your management plan?

5. What are the potential complications associated with this condition?

6. What do you think you should explain to the mother at this stage?

Case Study 5

Martin Jones is a 37-year-old who presents to the GP because he was worried about a new rash on his elbows and knees.

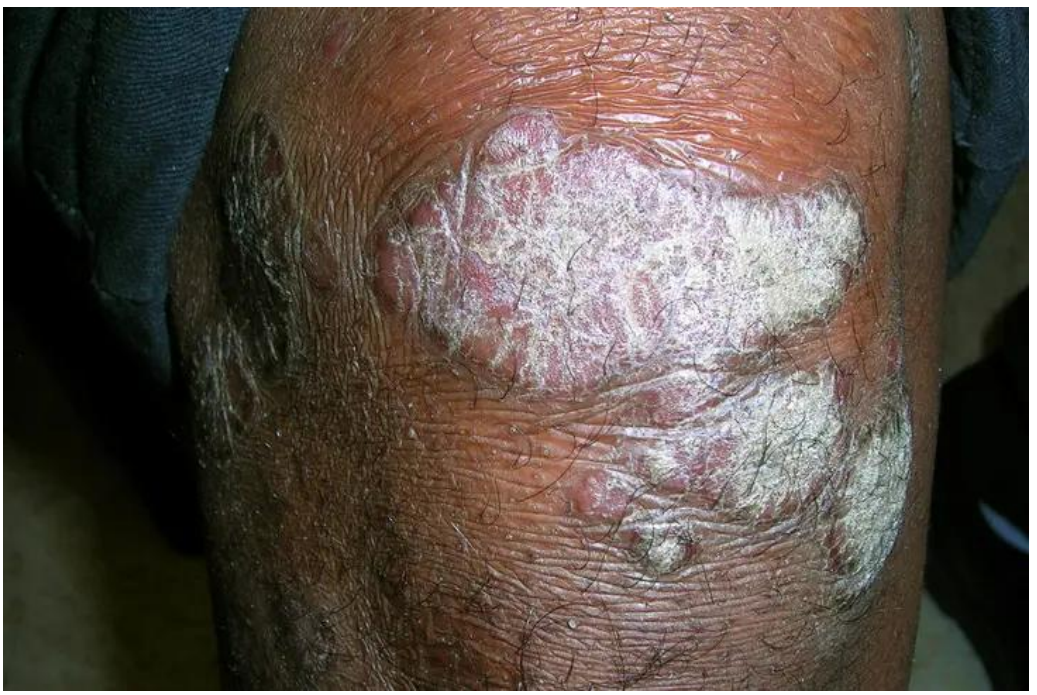
He first noticed he had one or two lesions a few months ago. They have gradually increased in number and size to the point that he has to always wear long sleeved shirts and cannot wear shorts anymore because he feels embarrassed. They are not painful. He has been trying lots of different creams that his wife buys him, but nothing has made a difference. His rash briefly improved over the summer but is now worsening again. His past medical history includes a recent diagnosis of Type 2 Diabetes Mellitus, Hypercholesterolemia and Hypertension. He has been advised to lose weight by his GP because his BMI is in the obese category (>30). He has no known drug allergies and is currently on Simvastatin, Ramipril and Metformin.

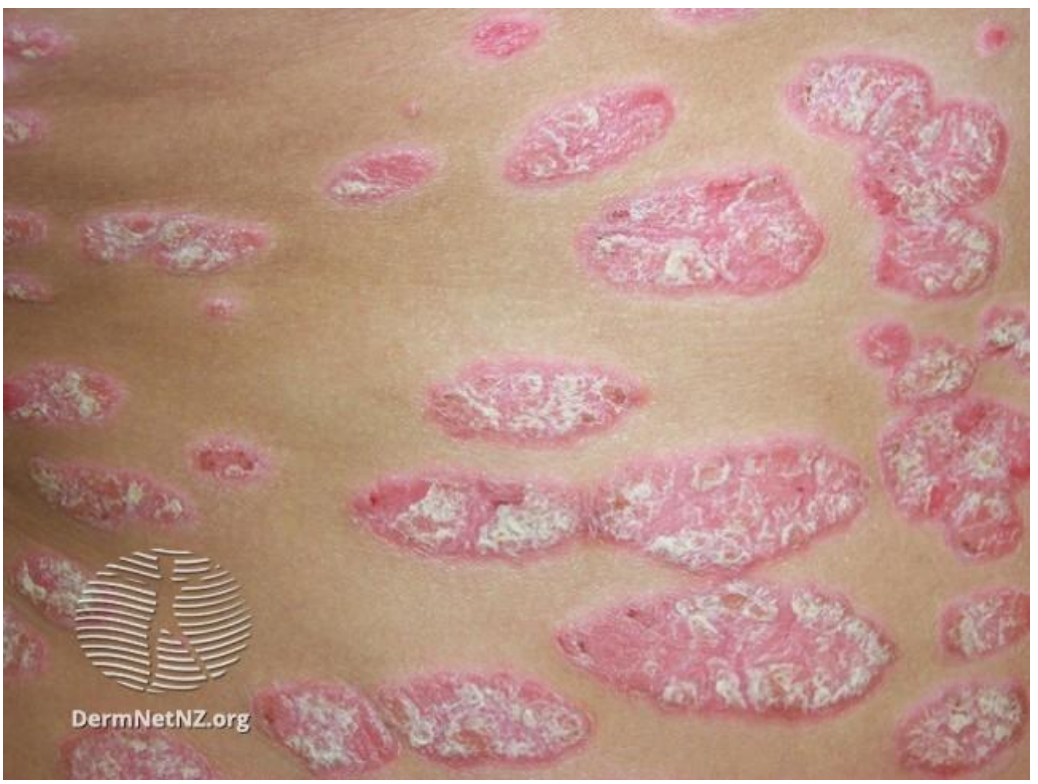
His mother had psoriasis and a heart attack at 67. He smokes around five cigarettes a day and admits he is a social drinker as this helps with the pressures of his job. He works long hours as a lorry driver and this is one of the reasons why it has taken him a while to see the GP as he struggles to make the time to get an appointment as he is often travelling around the country. He lives with his wife and three young daughters.

He feels embarrassed about the rash and would like to know if there is anything that can be done about it.

1. What questions would you like to ask him?

2. Can you describe the rash on the below picture?





3. What would be your clinical diagnosis and why?

4. Where else would you like to examine?

5. What would be your management plan?

6. What complications are associated with this condition?

7. What would you explain to the patient at this stage?

Resources

British Association of Dermatologists (BAD) Handbook for Medical Students and Junior doctors

https://cdn.bad.org.uk/uploads/2024/04/15113327/Derm_Handbook_3rd-Edition-_Nov_2020.pdf (please copy and paste this link in your navigator if you cannot open this page).

Medical Student Dermatology application, now web based (we are asking the British Association of Dermatologists to make it more clearly available on their webpage under British College of Dermatology/ Medical Students and Dermatology Trainees)

WE STRONGLY RECOMMEND YOU GO THROUGH THE WHOLE APP DURING YOUR DERMATOLOGY WEEK

Lecture Notes: Dermatology 11th Edition (available at the University Library, the most useful/comprehensive resource, so make the most of it!)

By Robin Graham-Brown, Karen Harman and Graham Johnston

Differential Diagnosis in Dermatology. 4th Ed Richard Ashton, Barbara Leppard, Hywel Cooper

Available at the University Library and at Odames Library. There is a Kindle Edition.

Other on-line Resources

NZ Derm Net http://www.dermnetnz.org/

For those of you who are already thinking of making your CV look great:

- British Association of Dermatologists Undergraduate awards
  https://www.bad.org.uk/education-training/scholarships-and-awards

- Leicester James Overton Dermatology Essay Award £150 - Enquiries to nab32@leicester.ac.uk
- DermSchool
  http://www.bad.org.uk/healthcare-professionals/education/medical-students/dermschool-event
- Leicester DermSoc
  Link to Facebook site: https://en-gb.facebook.com/UOLdermsoc/

Dermsoc Leicester is dedicated to increasing awareness of Dermatology as a medical specialty through such events as careers lectures, revision events for Phase 2 students. We always welcome new members and ideas for events, and are happy to pass on any questions about applications or research in Dermatology