

NEW PTS APPROVED 2A SYLLABUS 2022-2023

Dear Phase 2a's,

Below is a checklist of key topics covered in Phase 2a created on behalf of the Sheffield Medical School Peer Teaching Society (PTS).

We have read through the medical school's curriculum from the Phase 2a handbook and used it to make a list of the concepts you are expected to know for your exams. Although this list will not cover every topic that could come up in your exams, it is hopefully a useful resource for helping you with revision and note taking.

This is definitely not everything that could be on your exams but hopefully covers the most important topics. We have also included a guide at the end covering some resources and methods of studying in phase 2a.

We wish you the best of luck and are here to support you throughout the year.

Please use the Peer Teaching Website (<https://www.peerteaching.co.uk>) for useful resources and revision aids or for contact information. Good luck and don't forget to take well deserved breaks when you can!

Tom Grayson
2022-2023 PTS President

Paige Wilson
2022-2023 PTS Vice President

Haroon Tariq
2022-2023 PTS 2a Co-ordinator



Disclaimer - We have done our best to make sure there are as few mistakes as possible. Peer Teaching Society is not liable for any misleading information.

INTRODUCTORY CLINICAL SCIENCES & MISCELLANEOUS LIST

This list has been created by Haroon Tariq.

For these topics, I have provided a rough guide for your learning. Essential knowledge which should be prioritised has been highlighted in blue. Use the boxes to check off topics once they've been completed.

Your lecturers will also often stress key points they think are likely to come up in your exams and these should therefore be focused on.

Any inquiries on the syllabus, note making or any other concerns my email is htariq5@sheffield.ac.uk :)

PATHOLOGY

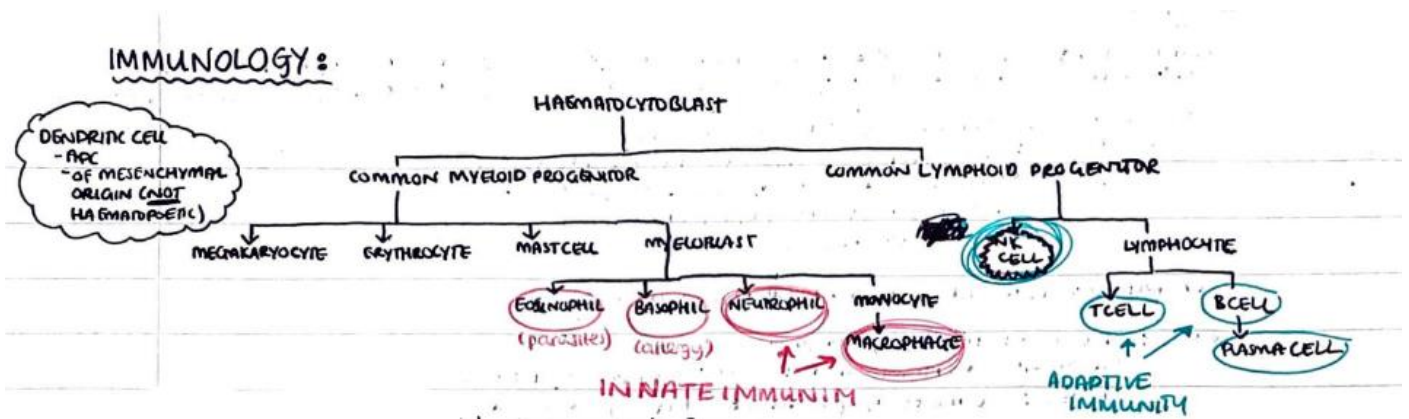
(note 2a pathology is based on Simon Cross' book *Underwood's Pathology: a Clinical Approach* – copies are available in the Med School Library)

- Inflammation
- Acute and chronic cells involved
- The '5 cardinal signs'
- Stages of inflammation - increased vessel permeability, fluid exudate, cellular exudate
- Neutrophil action - margination, adhesion, emigration and diapedesis
- Outcomes of inflammation - resolution, suppuration, organisation, progression
- Granulomas; definition, which conditions you may see them in (NB - granulomas all secrete ACE as a blood marker)
- Thrombi and Emboli
- Definition of each
- Example of each
- Venous vs arterial thrombi & emboli
- Virchow's triad!!! (AND factors affecting each component)
- Coagulation; vascular spasm, primary platelet plug (aggregation, adhesion, activation), secondary stable plug (coagulation cascade)
- Atherosclerosis
- Definition
- Risk factors
- Constituents of atherosclerotic plaque
- Formation process
- Apoptosis and Necrosis
- Apoptosis vs necrosis
- Mechanisms of apoptosis → Intrinsic, extrinsic, cytotoxic
- Cell Definitions
- Hypertrophy
- Hyperplasia
- Atrophy
- Metaplasia
- Dysplasia
- Ischemia
- Infarction
- Cancer Pathology
- Definition of a neoplasm (and of a tumour)
- Tumour classification - behaviour (benign vs malignant)
- Tumour classification - histogenesis (nomenclature of carcinomas and sarcomas)

Disclaimer - We have done our best to make sure there are as few mistakes as possible. Peer Teaching Society is not liable for any misleading information.

- Modes of tumour spread
- Which tumours metastasise to bone ('BLT KP')
- Risk factors for cancers (carcinogens, inherited predispositions e.g. FAP and HNPCC in colorectal cancer)
- Which cancers are screened for in the UK and how they're screened (colorectal, breast, cervical) NB: Cancer screening is a form of secondary prevention

IMMUNOLOGY



- HEMATOPOIESIS FLOWCHART:
- Innate Immunity
- Definition and description (e.g. rapid, non specific)
- Cells involved and functions: neutrophil, basophil, eosinophil, macrophage
- Complement system - pathways and function
- TLRs (know function of a few subtypes; TLR2, 4, 5, 7, 9)
- The 'professional' antigen presenting cell (= dendritic cell). Note B cells and macrophages can also do this.
- Adaptive Immunity
- T cells; subtypes, cytokines involved, maturation + thymic tolerance
- B cells; maturation, cytokines, antibody production
- Immunoglobulins (GAMED)
- Major histocompatibility complex (MHC 1 and 2 in humans)
- Hypersensitivity reactions 1-4 - names, mechanisms, examples
- Other
- Autoimmunity
- Vaccinations

PHARMACOLOGY (have a look at drugs on 2a drug list too!)

- Routes of drug administration
- Definitions:
- Agonist
- Antagonist
- Inverse agonist
- Competitive/non-competitive inhibition
- Selectivity + specificity
- Bioavailability
- Drug targets - receptors, ion channels, enzymes, transporters
- Pharmacodynamics definition

- Pharmacokinetics - ADME
- Sympathetic vs parasympathetic nervous system (including neurotransmitters)
- Cholinergic pharmacology (Ach movement at the synaptic cleft, Cholinergic Crisis SLUDGE mnemonic)
- Adrenergic pharmacology (alpha and beta receptor action)
- Other receptors - CNS receptors, histamine receptors
- Pain - types, pathway, the pain ladder for drugs
- Adverse drug reactions - ABCDE, and the Yellow Card Scheme
- Opioid pharmacology
- Paracetamol overdosing
- Anti-bleeding drugs - Antiplatelets, anticoagulants (mechanisms, uses)
- Common drugs and side effects - NSAIDs, ACEIs, diuretics, Beta blockers, CCBs, PPIs

MICROBIOLOGY

- For ICS module:**
 - Gram staining - 'Come In And Stain'
 - Ziehl-Neelsen stain
 - Cultures; blood agar, XLD, MacConkey, CLED, Sabourard, Lowenstein-Jensen
 - Catalase test
 - Coagulase test
 - Alpha beta gamma haemolysis
 - Optochin test
 - Gram positive bacteria structure function examples - note mostly cocci, in chains or clusters
 - Gram negative bacteria structure function examples - note mostly rod shaped
 - Antibiotics; types, sites of action, organisms they are effective against, antibiotic resistance mechanisms
 - Protozoa - examples (MALARIA; learn condition and learn the life-cycle!)
 - Fungi - examples, key conditions, treatment
 - Helminth worms - examples, definition of pre-patent period
- By the end of 2a:**
 - Viral infection; diagnosis (PCR + serology), EBV, CMV, HIV, viral causes of meningitis + encephalitis (also need to know bacterial causes of meningitis), Hepatitis A-E
 - Lung disease: common organisms and Abx used (COPD, pneumonia, bronchiectasis, CF, PCP pneumonia in HIV, TB)
 - Cellulitis common organisms and Abx
 - UTI; KEEPS organisms, causes (obstruction), investigations (1st line and gold standard) + management
 - Infective endocarditis; organisms, immunological phenomena
 - Enteric infection: causes of diarrhoea, organisms in appendicitis and in ascending cholangitis

GP SESSIONS

(ALONGSIDE KNOWING THESE CONDITIONS; this list is the content they like to ask in exams) - based on prescription tasks and exam questions:

- Paediatrics:
 - baby checks (when + briefly what happens), 5 in 1 vaccination at 8 weeks, paracetamol routes of administration
- Myocardial infarction:

prophylactic medication for MI, chronic ECG changes after an MI, heart failure drugs, ACEi mode of action and side effect/s, Africans on CCB for hypertension due to low renin dependence

- **Chronic pain:**
MOA of NSAIDs including side effects, Pain ladder for pain escalation
- **Chronic bowel disorder:**
IBD investigations, UC vs Crohn's, IBD vs IBS, red flag Sx, MOA of antimotility agents and laxatives, cOCP use when woman has diarrhoea (and other contraception options to consider)
- **Diabetes:**
diabetes drugs especially metformin (mode of action, side effects, and that excretion is via kidneys)
- **Inflammatory arthritis:**
rheumatoid arthritis vs osteoarthritis, treatment for OA and RA, gout vs pseudogout, Mode of action for methotrexate and folate supps (including folate supplementation in pregnant women)
- **Depression:**
signs of depression (what you'd ask patient, what you'd ask witnesses), pharmacology (mode of action, side effects) - SSRI, SNRI, TCAs, St John's Wort (complementary)
- **Epilepsy:**
drugs - sodium valproate, carbamazepine (mode of action), side effects, contraindications), differentiating epilepsy and other causes of seizures (e.g. tongue biting)
- **Lower Urinary Tract Symptoms:**
know what these LUTs are. Tx for BPH (MOA, side effects), why PSA isn't reliable, urinary retention and incontinence causes
- **Breast Cancer:**
Physical examination findings, Ddx of a breast lump, risk factors for breast cancer, diagnosing breast cancer (incl. Mammography screening age).
NB that oestrogen is osteoprotective. Post-menopausal women have low oestrogen so higher chance of osteoporosis - they take bisphosphonates → know MOA, side effects, and instructions for use

ILAs

(again alongside main conditions - this is what you'd need to know for the exam)

- **Atherosclerosis (think ACS, PVD):**
 - Structure of normal arterial wall, arterioles, elastic vs muscular
 - Process of atherogenesis
 - Components of an atherosclerotic plaque
 - Risk factors for atherosclerosis (modifiable/non-modifiable) and prevention
- **Anaphylaxis:**
 - Definition and hypersensitivity type
 - Examples of different hypersensitivity (Gell and Coombs classification 1-4)
 - Pathophysiology of anaphylaxis and symptoms
 - Blood test specific to anaphylaxis
 - Treatment for anaphylactic shock
 - Risk factors for anaphylaxis
 - Prevalence vs incidence
- **Pharmacokinetics and Pharmacodynamics**
 - Definitions of both
 - Anaesthesia - protein binding and solubility
 - Bioavailability definition and application
 - Agonist vs antagonist in regards to affinity and efficacy
 - Definition of first and second pass hepatic metabolism
 - Excretion and bioavailability of morphine
- **Thrombosis and Coagulation**
 - Arterial vs venous thrombosis
 - 6 Ps of PVD

- Blood test for DVT and why it's sensitive but isn't specific
- DVT and PE treatment (and their mode of action)
- Recognise PE is complication of DVT and consider VTE risk factors - VIRCHOW'S TRIAD
- AKI
 - Diagnostic criterion for AKI stages 1, 2 and 3 (KDIGO)
 - Pre-, intra-, post- renal causes of AKI
 - Drug for renal colic (Diclofenac - an NSAID)
 - Drugs to stop in an AKI (DAMN mnemonic)
 - Main complication to monitor in AKI - hyperkalemia. Hyperkalemia investigations, the major risk factor associated with it (cardiac) and treatment
- Stroke
 - Definition of stroke and TIA
 - Definition of amaurosis fugax
 - Pathophysiology of TIA + strokes, and main risk factors
 - Recognise terminology for region of brain affected by strokes; TACS, PACS, LACS, POCS (including which are most common, and BVs responsible for supplying these areas)
 - Signs of raised intracranial pressure including Cushing's reflex triad

THE CONDITIONS LIST

This conditions list has been created by Tom Grayson.

Colour banding is the last line of defence. Avoid using the colour coding if possible!

I have created a green, yellow, red colour banding. First of all you should know every topic listed in this as any of them could come up and they are all important. If you have run out of time when taking notes/ revising that is when the banding is useful. I have banded the most important/ highest yield concepts in green and the more niche concepts in red with yellow somewhere in-between. If you have run out of time when revising, this banding system is the last line of defence. If you have time on your side or are aiming for the highest grades you should not start off using the banding system. It is only there for those that have ran out of time and are struggling to strategize their learning time close to the exam.

Second of all I have done my best to list every topic you should know at phase 2a but I cannot guarantee these are the only topics that will come up in your exam but I have done my absolute best to make a revision guide of what you need to know to make your year a little easier and more enjoyable.

Any inquiries on the syllabus, note making or any other concerns my email is tgrayson1@sheffield.ac.uk please feel free to email me :)

ENDOCRINE

- Diabetes mellitus (type I and II) including micro/macrovacular complications and
 - Ketoacidosis
 - Hyperosmolar hyperglycaemic state
- Hypoglycaemia
- Thyroid Disorders
 - Hyperthyroidism – mainly Graves disease, recognise other causes
 - Hypothyroidism – mainly Hashimoto’s thyroiditis, recognise other causes
 - Recognise other causes of thyroid disorders; De Quervain’s thyroiditis, post-partum thyroiditis, amiodarone, lithium toxicity
 - Thyroid cancer
- Pituitary adenomas – symptoms, examples of conditions which may be caused by them;
 - Cushing’s syndrome and disease
 - Acromegaly
 - Prolactinoma
- Carcinoid tumours and syndrome
- Conn’s Syndrome
- Adrenal insufficiency: primary (Addison’s) and secondary
- SIADH
- Diabetes insipidus (cranial and nephrogenic)
- Hyperparathyroidism (primary secondary tertiary)
- Hypoparathyroidism
- Pheochromocytoma
- Electrolyte imbalances
 - Hypercalcaemia

Disclaimer - We have done our best to make sure there are as few mistakes as possible. Peer Teaching Society is not liable for any misleading information.

- Hypocalcaemia
- Hyperkalaemia
- Hypokalaemia
- Hyponatremia
- Hypernatremia

CARDIOVASCULAR

- Angina
 - Stable
 - Unstable
 - Myocardial Infarction; NSTEMI & STEMI (note 'Acute Coronary Syndromes' are: unstable angina, NSTEMI & STEMI)
 - Prinzmetal's angina
- Heart Failure
 - Right sided
 - Left sided
 - Ischaemic
 - Myopathic
 - Hypertensive
 - Cor pulmonale
- Abdominal aortic aneurysm
- Aortic dissection
- Arrhythmias (including ID on an ECG!)
 - Supraventricular tachycardias
 - Atrial fibrillation
 - Atrial flutter
 - AVRT (Wolff-Parkinson White Syndrome)
 - AVNRT
 - Ventricular tachycardias
 - Ventricular ectopic
 - Prolonged QT syndrome
 - Torsades de Pointes
- Conduction blocks
 - Heart block; 1st, 2nd (Mobitz I and Mobitz II subtypes), 3rd (Complete)
 - Bundle Branch Block (WILLIAM MaRRoW)
- Hypertension
- Venous Thromboembolism
 - Deep vein thrombosis
 - Pulmonary embolism
- Peripheral vascular disease
- Pericarditis
- Pericardial Effusion (complication ☒ Cardiac Tamponade)
- Infective Endocarditis
- Valve diseases
 - Aortic stenosis
 - Aortic regurgitation
 - Mitral stenosis

- Mitral regurgitation
- Shock
 - Hypovolaemic
 - Anaphylactic
 - Septic
 - Cardiogenic
 - Neurogenic
- Cardiomyopathy
 - Hypertrophic
 - Dilated
 - Restrictive
- Rheumatic Fever
- Structural heart defects
 - Tetralogy of Fallot
 - Coarctation of aorta
 - Ventricular septal defect
 - Atrial septal defect
 - Patent ductus arteriosus

HAEMATOLOGY

- Anaemia
 - Microcytic (MCV < 80); Fe deficiency, Alpha & Beta Thalassemia, Sideroblastic
 - Normocytic (MCV 80-95);
 - Haemolytic – Sickle cell, Hereditary spherocytosis, G6PDH deficiency, Malaria, Autoimmune Haemolytic
 - Non-Haemolytic – CKD, Aplastic
 - Macrocytic (MCV 95 <);
 - Megaloblastic – B12 deficiency, Folate deficiency
 - Non-megaloblastic – Hypothyroidism, Alcohol excess, Liver disease
- HIV
- Glandular fever
- Leukaemia
 - Acute myeloid leukaemia
 - Acute lymphoid leukaemia
 - Chronic myeloid leukaemia
 - Chronic lymphoid leukaemia
- Lymphoma
 - Hodgkin
 - Non-Hodgkin
- Multiple Myeloma
- Polycythaemia – primary (PCV) and secondary
- Thrombocytopenia
 - ITP
 - TTP
- Bleeding disorders
 - Von Willebrand
 - Haemophilia A

- Haemophilia B
- Disseminated Intravascular Coagulopathy
- Chemotherapy complication: Tumour lysis syndrome
- Blood cells and clinical values
 - Neutropenia/philialia
 - Lymphopenia/philialia
 - Thrombocytopenia/philialia
 - PT/INR
 - APTT

GASTROINTESTINAL

- Inflammatory bowel disease
 - Crohn's
 - Ulcerative colitis
- Irritable bowel syndrome
- Coeliac disease
- Gastritis
- GORD (complication ☐ Barrett's oesophagus)
- Peptic Ulcer Disease
 - Gastric
 - Duodenal
- Mallory Weiss tear vs Oesophageal Varices (will cover oesophageal varices in Liver module)
- Achalasia
- Bowel ischaemia
 - Ischaemic colitis
 - Mesenteric ischaemia
- Appendicitis
- Diverticular diseases – diverticula, diverticulosis, diverticular disease, diverticulitis
- Gastritis
- Intestinal obstruction
 - Small bowel obstruction
 - Large bowel obstruction
 - Pseudo-obstruction
- Diarrhoea (Causes, types, main bacteria involved – C.difficile, E.coli, Shigella, Salmonella, C.jejuni, treatments)
- Helicobacter Pylori (investigations and treatment)
- GI cancer (small bowel cancer is very rare)
 - Oesophageal cancer
 - Gastric cancer
 - Large bowel cancer including hereditary predispositions (FAP, Lynch syndrome)
- Meckel's diverticulum (aka 'pharyngeal pouch')
- Pseudomembranous colitis (CMV 'owl eye inclusion bodies')
- Perianal disorders
 - Haemorrhoids (internal and external)
 - Fistulae
 - Fissure

- Perianal abscess
- Pilonidal sinus / abscess

LIVER & FRIENDS

- Acute and Chronic liver Failure
- Alcoholic liver disease
- Non-alcoholic fatty liver disease
- Hepatitis – A/B/C/D/E/ autoimmune
- Liver Cirrhosis
- Jaundice; a symptom. Note pathophysiology and causes (pre-, intra-, post-hepatic)
- Wernicke's encephalopathy and Korsakoff syndrome
- Hepatic encephalopathy
- Hepatobiliary cancers
 - Pancreatic cancer (including Courvoisier's sign)
 - Hepatocellular carcinoma
 - Cholangiocarcinoma
- Paracetamol overdose
- Gilbert's syndrome
- Hernias
 - Inguinal
 - Femoral
 - Umbilical
 - Incisional
 - Epigastric
 - Hiatal
- Biliary tract disease
 - Biliary colic
 - Cholecystitis
 - Ascending cholangitis
- Primary biliary cholangitis
- Primary sclerosing cholangitis
- Acute and chronic pancreatitis
- Ascites; a symptom. Pathophysiology, SAAG, causes and diagnosis
- Portal hypertension and oesophageal varices
- Spontaneous Bacterial Peritonitis
- Metabolic liver disease
 - Haemochromatosis
 - Wilson's disease
 - Alpha 1 antitrypsin deficiency

MUSCULOSKELETAL & RHEUMATOLOGY

- Osteoarthritis
- Rheumatoid arthritis
- Crystal arthritis
 - Gout
 - Pseudogout

- Osteoporosis (+ osteopenia)
- Fibromyalgia
- Sjogren's syndrome
- Vasculitis
 - Giant cell arteritis
 - Wegener's granulomatosis (granulomatosis with polyangiitis)
 - Polyarteritis nodosa
- Paget's disease of bone
- Spondyloarthropathies (SPINEACHE)
 - Ankylosing spondylitis
 - Psoriatic arthritis
 - Reactive arthritis
 - Enteric arthritis
- Infective arthritis
 - Septic arthritis
 - Osteomyelitis
- Systemic lupus erythematosus
- Antiphospholipid syndrome
- Dermatomyositis/polymyositis
- Scleroderma (CREST)
- Polymyalgia rheumatica
- Mechanical lower back pain, including vertebral disc degeneration
- Osteomalacia
- Primary and secondary bone tumours (KNOW WHICH TUMOURS METASTASISE TO BONE)
- Connective tissue disorders
 - Marfan's
 - Ehlers Danlos

RENAL & GENITOURINARY

- Renal Colic (nephrolithiasis)
- Acute kidney injury
- Chronic kidney disease
- Urinary tract infection
 - UPPER - Pyelonephritis
 - LOWER – Cystitis, Urethritis, Epididymo-Orchitis, Prostatitis
 - Note complicated vs non complicated UTI
- Nephritic syndrome
 - IgA nephropathy (Berger disease)
 - Post-strep glomerulonephritis
 - Goodpasture's syndrome
 - SLE nephropathy
- Nephrotic syndrome
 - Minimal change disease
 - Focal segmental glomerulosclerosis
 - Membranous nephropathy
- Nephritic and nephrotic

- Diffuse proliferative glomerulonephritis
- Membranoproliferative glomerulonephritis
- Benign prostate hyperplasia
- GU Cancer
 - Prostate cancer
 - Testicular cancer
 - Bladder cancer
 - Kidney cancer
- Polycystic kidney disease – dominant and recessive
- STIs
 - Chlamydia
 - Gonorrhoea
 - Syphilis
- Scrotal disease
 - Varicocele
 - Testicular torsion (and NB difference to testicular appendage torsion)
 - Epididymal cyst
 - Hydrocele
- LUT symptoms including causes of incontinence and of retention

NEUROLOGY

- Stroke
 - Ischaemic stroke
 - Haemorrhagic stroke
 - Transient ischemic attack
- Haemorrhagic stroke
 - Intracerebral haemorrhage
 - Subarachnoid haemorrhage
 - Extradural (epidural) haemorrhage
 - Subdural haemorrhage
- Amaurosis fugax; a symptom. Note when it can occur.
- CNS infection;
 - Meningitis (bacterial, viral and fungal)
 - Encephalitis
- Multiple Sclerosis
- Guillain-Barre syndrome
- Parkinson's disease
- Huntington's disease
- Dementia
 - Alzheimer's disease
 - Frontotemporal
 - Lewy body
 - Vascular
- Headaches (primary)
 - Migraine
 - Tension

- Cluster
- Trigeminal neuralgia
- Epilepsy
 - Other causes of seizures and differentiating epilepsy from these
 - Generalised seizures
 - Focal seizures (simple and complex)
- Spinal cord compression (including sciatica)
- Cauda equina
- Cranial nerve lesions
- Motor Neurone Disease
- Upper and lower motor neuron lesions
- Myasthenia Gravis
- Lambert Eaton Syndrome
- Syncope
- Limb neuropathies;
 - Carpal tunnel syndrome
 - 'Wrist drop'
 - 'Claw hand'
 - 'Foot drop'
- Peripheral neuropathy (including mononeuritis multiplex)
- Brown-Sequard syndrome
- Charcot-Marie-Tooth Syndrome
- Duchenne Muscular Dystrophy
- Depression
- Primary and secondary brain tumours (note Glioblastoma multiforme)

RESPIRATORY

- COPD
 - Chronic bronchitis
 - Emphysema
- Asthma
- Lung Infection
 - Tuberculosis
 - Pneumonia (Community acquired and Hospital acquired). NOTE: Pneumocystis pneumonia is most common in HIV patients
- Cystic fibrosis
- Bronchiectasis
- Pleural space pathology
 - Pleural Effusion
 - Pneumothorax
 - Empyema
- Interstitial lung diseases
- Pulmonary fibrosis
- Sarcoidosis
- Pulmonary hypertension

- Lung cancer
 - Small cell
 - Non-small cell
 - Mesothelioma
- Hypersensitivity pneumonitis
- Dyspnoea; a symptom. Recognise the MRC dyspnoea scale, and causes of dyspnoea.
- Type 1 and 2 respiratory failure – understand differences, name conditions which are type 1 + 2
- Occupational lung disorders
- Goodpasture's syndrome
- Upper respiratory tract infections
- Pharyngitis
- Otitis media
- Sinusitis
- Acute epiglottitis
- Croup
- Whooping cough

APPROACHES TO NOTE-MAKING:

Below are some great tips for your revision from the Phase 2a PTS Co-ordinator (Haroon) and the PTS President (Tom).

- Haroon's Tips; BEST Resources, 2a Pointers, My Teaching Drive Link and Notes Template

My approach to 2a note-making is very much simplicity and concision is key; should you go into massive detail, though your notes may look great they're hard to commit to memory. If you haven't noticed already, 2a is a huge step up from Phase 1 with there being so much more to learn in a shorter time-frame. The best thing to do with masses of information is to write it in as few words as possible while retaining its meaning, so using abbreviations and mnemonics were super useful for me.

Also have an idea of the resources you're going to use from the onset - it's tough to change your strategies midway through. Therefore, a good approach early on is crucial.

I'd recommend PTS powerpoints and recordings being an ideal starting point, accompanied with Drive Notes (such as 'KP'), past exam question reports/Medsoc mocks, and PTS papers as the best strategy for Sheffield-specific knowledge. Some of the God-Gifted resources I have to mention because they were so good were Quesmed (the question-bank, textbook and the SBA videos on specialties) and BiteMedicine (*religiously* used - seriously don't sleep on this). While BMJ best practice and NICE guidelines are great resources in themselves, they should be used mainly to check you have the right sort of information rather than using them primarily as a source for notes – personally they're too waffly and confusing to rely on alone. The end of this document details more resources I found useful.

Content-wise, Tom's list was what I referred to for my exam and it really does cover almost everything you need for 2a. As 2a Co-ordinator, in addition to the new ICS and Miscellaneous list, another development I've made is editing his list's layout and wording. Compared to previous years' lists I've added prompts, key information to be aware of & focus on, and more direct instructions to help streamline your revision and make it more user-friendly, which I hope you find useful.

Remember not to neglect microbiological and pharmacological aspects of condition-based learning; know the common organisms and common drugs/classes required as this frequently comes up in papers from experience.

You all have access to my teaching drive which has all my notes, teaching videos and any PTS teaching 2022/23, which can be found here: https://drive.google.com/drive/folders/1uyINaCx44avUz-cErjKyq0LrEM0qA1J7?usp=share_link

Below is the 'template' I used for each condition, and honestly I think this is more than enough if learnt well. Remember that the clearer your notes, the better. Furthermore, don't be afraid to customise your own headings - the notes I make are different to many, so you should tailor what is important for each condition to you.

In the meantime, if you have any questions feel free to drop me an email here: htariq5@sheffield.ac.uk :)

Disclaimer - We have done our best to make sure there are as few mistakes as possible. Peer Teaching Society is not liable for any misleading information.

TEMPLATE

- **Condition name and definition.**
Once you're in a position to revise – I found it useful to write a basic 'typical patient'
- **Epidemiology** (if necessary)
- **Causes, risk factors and associations**
Direct and indirect causes. Modifiable and Non-Modifiable risk factors. Associated with... (e.g. Polymyalgia rheumatica is heavily associated with Giant Cell Arteritis)
- **Pathophysiology**
I often summarised this in condition name and definition, but some cases it's worth the pathophysiology in more detail
- **Signs and symptoms (Sx)**
Signs are *clinical findings* by doctors. **Symptoms** are *what the patient presents* to doctors with. NB; it is useful to make a note of 'differential diagnoses' (i.e. based on the signs/symptoms, what diseases it may be.
E.g. causes of epigastric abdominal pain may be a ruptured AAA or pancreatitis (these are your differentials – overlapping symptoms for which you then need to consider all the other factors)
- **Investigations (Dx and Ix)**
The 1st line and **gold standard** are most important. Others - Bedside (e.g. ECG, BP), Bloods (FBC, U+E), Imaging (X-Rays, CT scans, MRIs, USS)
- **Treatment (Tx)**
Conservative (non-medicine), medication, surgical
- **Complications** (if applicable)

Here's an example from my own notes, using Cushing's disease & syndrome (Endocrine module):

CUSHING DISEASE + SYNDROME - hypercortisolemia

Cushing DISEASE = pit adenoma secreting excess ACTH
Cushing Syndrome = hypercortisolemia of any cause

Causes

- ACTH DEPENDENT → CUSHING DISEASE (MC) + ectopic ACTH (e.g. SCLC)
- ACTH INDEPENDENT → IATROGENESIS (STEROIDS) (MC) + adrenal adenoma

iatrogenesis is MOST COMMON OVERALL CAUSE

Path CRH → ACTH → cortisol.
 * CRH typically released w/ circadian rhythm (↑ in MORNING; ↓ at night)
 * Here, this rhythm is lost; excessive unregulated CRH, ACTH + cortisol.

Sx MOON FACE, CENTRAL OBESITY, PURPLE ABDO STRIAE, OSTEOPOROSIS, THIN EASY-BRUISING SKIN, PLETHORIC COMPLEXION. + easy infections, muscle atrophy.

Dx **RULE OUT ORAL STEROIDS**; if on steroids, stop.
 Random serum cortisol ↑ than 1st wave test, measure at 12am
 * cortisol is normally at its LOWEST here; if ↑ then v. abnormal

★ DEXAMETHASONE SUPPRESSION TEST (OVERNIGHT)
 Dexamethasone = essentially cortisol ∴ in healthy patient should -ve feedback HPA axis and cortisol

- ① give Dexamethasone and measure cortisol (before giving dex). 00:00
- ② measure cortisol 8h later 08:00.
 - non Cushing's → suppression >50nmol/L
 - Cushing's → little/no suppression

If 1st line +ve, then measure PLASMA ACTH
 • ↑ = ACTH dependent cause → LOOK for Cushing disease on pituitary MR
 • ↓ = ACTH independent cause → consider adrenal adenoma.

Tx

- Cushing disease = transphenoidal resection or bilateral adrenalectomy - complication = NEURONAL SYNDROME; Pituitary tumour will continue to enlarge with no -ve feedback from adrenals; ↑↑ ACTH + skin hyperpigmentation
- Adrenal adenoma = unilateral adrenalectomy
- Ectopic ACTH = surgical removal e.g. SCLC.

Comp - OSTEOPOROSIS, 2° DM

Ab - alcohol makes PSEUDO-CUSHING's - resolves in 1-3 weeks

Disclaimer - We have done our best to make sure there are as few mistakes as possible. Peer Teaching Society is not liable for any misleading information.

- **Tom's Top Tips**

You've probably realised by now that the lectures in 2a are very different compared to phase 1 and it's hard to know where to begin for making notes as most of the lectures do not highlight key information which you need to know and are more a general overview of a disease.

The template below is a good layout on how to take notes for phase 2a. Obviously, this is just a template and you may find a way of note taking that suits you best, but this gives you an idea of how you might start. It is important to highlight the difference between 1st line and gold standard investigations as this can be a common exam question. Common exam questions are also which bacteria is the most common cause of disease x and you need to be able to tell between 2 similar diseases based off signs, symptoms and investigations. Similarly, with management, there is often a first line and main treatment that is given (this is the most important to learn) but also a number of additional treatments are often given to improve outcomes.

Disclaimer - I have done my best to make sure there are as few mistakes as possible but if there are any mistakes or misleading information I am not liable

Template

Disease name	
What is the disease	Definition and brief explanation of disease
Epidemiology if applicable	Does it affect more males or females. Is it a disease of elderly or young. This section isn't always applicable but when it is its obvious. For example lupus is 90% female, acute lymphoid leukaemia is mainly children, chronic lymphoid leukaemia is mainly over 70s ect
Aetiology	Cause of disease. E.g trauma, autoimmune. Infection (and if infection what bacteria or virus specifically !!!)
Pathophysiology	Pathology of disease, what goes wrong and in what order
Symptoms	What the patient complains of e.g. fatigue dizziness aches and pains
Signs	What you can see with a clinical examination e.g heart murmur on auscultation, shifting dullness on abdomen ect
Investigation	1 st line = what is the first test you would run Gold standard = the most accurate test to confirm a diagnosis Other useful tests
Differential diagnosis	Can ignore this bit but sometimes its useful. What diseases are similar and how can you tell a patient has this disease as opposed to another
Management	1 st line = drug you give first 2 nd line = drug you give second Other ways the disease can be managed
Complications	What happens if left untreated and side effects of management
Risk factors	What makes them more at risk of this disease e.g diabetic, alcohol abuse, hormone replacement therapy
Other	Other relevant stuff

Disclaimer - We have done our best to make sure there are as few mistakes as possible. Peer Teaching Society is not liable for any misleading information.

Example

Acromegaly	
What is the disease	Hormonal disorder resulting from too much Growth hormone release
Epidemiology if applicable	Not applicable
Aetiology	<ul style="list-style-type: none">• Most common cause = benign anterior pituitary adenoma• Other causes = small cell carcinoma + medication that increases growth hormone
Pathophysiology	Anterior pituitary secretes a plethora of hormones one of them being growth hormone. Growth hormone causes: increase in glucose release by the liver, muscles retain nitrogen causing them to grow, osteoblast stimulation making bones thicker, cells resistance to insulin increases. This is why when the cells responsible for secreting growth hormone randomly undergo hypertrophy (benign anterior pituitary adenoma) or the mechanism regulating these cells release of growth hormone is disrupted we get the signs and symptoms detailed below. The treatment is centred on removing these excess growth hormone releasing cells but if not we can instead increase inhibition of these cells to reduce the growth hormone release and manage the condition.
Symptoms	<ul style="list-style-type: none">• Bigger hands and feet• Excessive sweating• Headache• Tiredness• Weight gain• Deep voice• Amenorrhoea• Change in appearance
Signs	<ul style="list-style-type: none">• Bi-temporal hemianopia• spade like hands and feet• large tongue (macroglossia)• Jaw protrusion (prognathism)• Interdental separation• Prominent forehead (forehead bossing)
Investigation	<p>1st line</p> <ul style="list-style-type: none">• Raised insulin like growth factor 1 in acromegaly. <p>Gold standard</p> <ul style="list-style-type: none">• Oral glucose tolerance test – give glucose and in normal people serum growth hormone thus Insulin like growth factor 1 would reduce. In patients with acromegaly glucose has no effect on serum growth hormone or insulin like growth hormone 1 so they remain high <p>Other useful tests</p> <ul style="list-style-type: none">• Serum growth hormone would be high in acromegaly. Problem is the levels change depending on time of day, stress, sleep, diet, exercise ect so not really that useful but can be of some use non the less• MRI of pituitary usually caused by benign pituitary adenoma so this can be diagnostic but some are caused by small cell carcinomas (cancer in the lung thus wouldn't show up in MRI of head) or faulty

	feedback mechanism regulating growth hormone release e.g faulty somatomedin release thus again wouldn't show on MRI
Differential diagnosis	I usually miss out these as its very much down to your opinion on what diseases you think are similar an how well you can differentiate between them but ill fill it out anyway <ul style="list-style-type: none"> • Prolactinoma • Marfans syndrome • Precocious puberty
Management	1 st line <ul style="list-style-type: none"> • Trans-sphenoidal surgical resection (if it is a benign pituitary adenoma, which it probably is, this is first line. If the cause is something else like small cell carcinoma or medication abuse we wont start an opertation on his brain will we, so this wouldn't be first line for other causes but as the vast vast majority of causes are benign adenoma on the anterior pituitary this is the first line) 2 nd line <ul style="list-style-type: none"> • Cabergoline (dopamine agonist) and (octreotide) somatostatin analogue Other ways the disease can be managed <ul style="list-style-type: none"> • Radiotherapy • Pegvisomant – a growth hormone antagonist given subcutaneously and daily
Complications	Type 2 diabetes - due to constant high glucose levels Arthritis - due to overgrowth of cartilage Carpal tunnel - as muscle growth compresses nerves Heart failure – due to cardiomegaly GI cancer – due to colon polyps Sleep apnoea - as upper respiratory tract becomes obstructed especially when muscles relax Cerebrovascular disease Hypertension
Risk factors	Not applicable really here but history of pituitary adenomas, hormone secreting tumours or medication that increase growth hormone levels
Other	Not applicable here

SOURCES OF INFORMATION:

Here are some of the resources we have collated for you to use for revision:

- Lectures – these “should” go through the main diseases but they will miss lots out and don’t always have a clear layout to follow so they are useful but can’t rely solely on this like you could phase 1
- NICE guidelines – very good and up to date management of diseases
<https://www.nice.org.uk/guidance> and <https://cks.nice.org.uk/>
- Mayo clinic – not the most reliable but generally pretty accurate good for an overview the disease how its investigated treated etc
- Patient info – make of it what you will <https://patient.info/>
- Zero to finals – very pathology heavy which is useful for understanding but you don’t need much pathology for the exam mainly recognising signs symptoms saying patient likely has disease x how would I investigate disease x or treat it. has lots of nice tips.
- BMJ best practise - This is what I used mostly, has sections similar to the ones in the table which makes it quick to transfer the information across. You can get complete access through searching on StarPlus for BMJ Best Practice and following the links to the website from there. If you make an account, you can login through their app as well.
- Osmosis (YouTube) – you can pay for osmosis, I wouldn’t because a lot of their videos are free and some people have just recorded the videos you pay for and upload them to their youtube channel (although its copyright and probably illegal)
- Armando hasudungan (YouTube) – run the risk of information overload but good for getting a true understanding of a disease and as always YouTube videos are nicer to watch rather that read for hours
- Medicosis perfectionalis (YouTube) – a bit too pathology heavy but I used him a lot he has a lot of in depth explanations which all come together to make good sense and paint a clear image of what stuff you should be looking for with signs and symptoms
- Drive Notes – obviously these will have some errors in but I used these and found them very useful because you don’t need to spend ages googling separate aspects of the disease, the information is right in front of you. There is some info missing e.g. gold standard and first line treatments, but is a very good place to start from
- Geeky Medics (more 3rd year but some useful stuff) – nice layout and have a question bank where you can select topics I banged these out the week before my exam
- Oxford Clinical Handbook – good for the emergency stuff like anaphylaxis and the red stuff (because generally the red diseases have little clear information available online)
- Peer Teaching – going through powerpoints and mock papers available on the website is very useful for exams. Powerpoints give a good indication of a starting point for revision.
[Phase 2a - SHEFFIELD PEER TEACHING SOCIETY](#)
- Ninjanerd (YouTube) – very detailed videos on lots of conditions, a personal favourite
- Qesmed – a great question bank and lots of clinical flashcards on conditions. Paid subscription but highly recommend
- Passmed – a free question bank with lots of relevant (but lots of ‘extra’) questions. Worth using for exam practice.
- BiteMedicine – a great paid resource (can get limited access for free). Up to date notes with NICE guidance and webinars on conditions detailing almost everything you need to know, and is updated regularly by the team.

Disclaimer - We have done our best to make sure there are as few mistakes as possible. Peer Teaching Society is not liable for any misleading information.