

Preparing for the MBBS

**Approach to
and Resources for
the Exam**

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Preface

This book began in the lead up to MBBS, when we began writing as a tool for ourselves to consolidate what we have learnt. After MBBS, we decided to tidy up and compile these notes, in the hope that passing them down will make preparations slightly easier.

These notes offer a suggested approach to each clinical paper of the MBBS, with sample scripts and worked examples. Our focus is the thought process, not content – which you can get anywhere. We have divided the notes into five chapters: (1) surgical short cases, (2) medical short cases, (3) surgical long cases, (4) medical long cases, and (5) communications and task stations for both surgery and medicine. Each chapter begins with a discussion of strategy for that paper – we suggest that you read this before browsing the rest of the chapter.

Some suggestions:

- Don't study just for the MBBS, but more importantly, how to be a competent and safe HO. In fact, the MBBS exam has been tuned to emphasize the practical things you will encounter as a HO. Patients are your best teachers; please spend time in the wards!
- Do not simply cram information, but organize your thoughts as you go, make linkages between concepts, and – most importantly – apply to the patients you see.
- Take ownership of your own learning. Think about what you are learning, how you are learning, and how you can learn better.
- MBBS is daunting but do not be disheartened. Work consistently, and take heart – we too cannot remember everything we wrote!
- It is difficult to go through this alone. Practice with each other, help each other, look out for each other.

We wish to acknowledge our friends who have contributed cases: Eugene Gan, Vivien Lee, David Ng, May Na, Joyce Huang, Darius Pan, Michael Chee, Cheryl Lam, Teo Ling Li, Aaron Tang. Most importantly, we thank God for His grace, comfort, and peace through the entire MBBS, and for whom we work.

*Unless the LORD builds the house,
those who build it labor in vain.
Unless the LORD watches over the city,
the watchman stays awake in vain.
It is in vain that you rise up early
and go late to rest,
eating the bread of anxious toil;
for he gives to his beloved sleep.
(Psalm 127: 1-2)*

- Nigel Fong & Marianne Tsang

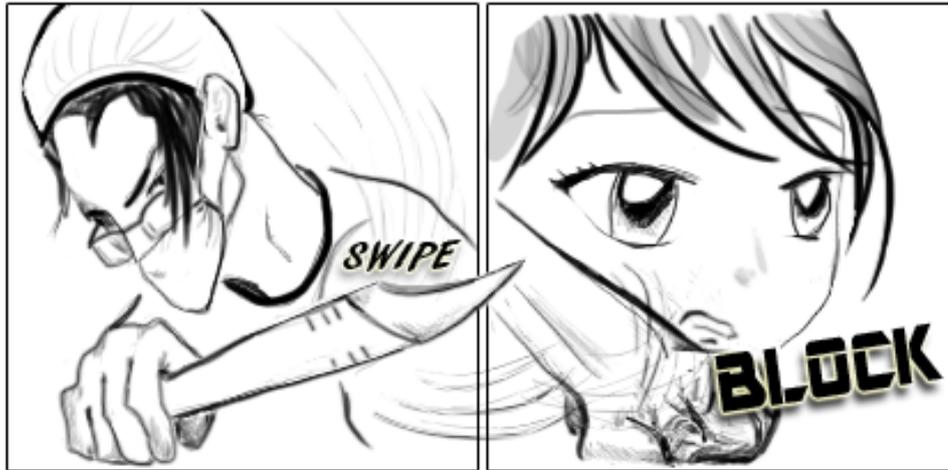
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N.B. Please help to flag any errors spotted to [nigelfong \[at\] gmail.com](mailto:nigelfong@gmail.com).

Surgery Short Cases

the song & dance



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Strategy

What are surgical short cases like? These are 8-minute stations focusing on clinical examination of a surgical complaint. Although physical examination is the focus, you will also be asked to take a brief history, and thereafter to discuss your assessment with the examiner (usually diagnosis & differentials, investigations and management).

Examiners want you to:

- Perform a technically competent (*steps*), accurate (*signs*), and smooth (*style*) physical examination.
- Offer running commentary, both to vocalize your thought process, and to describe your findings and assessment on-the-go (this is generally expected and also helps you to save time or be guided if you go off track).
- Be able to form a clinical impression and have a basic approach to management.

Your strategy: The repertoire of surgical short cases is quite limited and cases are very predictable. Very often, inspection alone will tell you what problem you are dealing with. However, it can be daunting to have to examine, think, give a running commentary at the same time, and *still* look smooth and confident. Therefore, it is hugely advantageous to have a war-chest of well-oiled songs and dances you can pull out to use. This chapter helps you to build that war-chest by providing some set-pieces (covers ~90% of all surgical short cases), plus common questions and answers. Practice with them and build on these scripts!

Troubleshooting:

- *I cannot find patients with signs.* Often desperate students outnumber grumpy patients. Don't panic. Try day surgery, clinic, and A&E. If there are too many students, don't do a full examination – focus on picking up the signs and rehearse the song and dance with each other separately (unlike medical short cases, you *can* rehearse with each other). Work consistently - look for patients throughout your posting, not in the last month before MBBS.
- *Running commentary is so difficult.* Start practicing early and get used to it. It will be difficult and choppy initially, but as with all performance arts, practice helps.
- *There is no time.* Be smooth and disciplined in your examination. Practice with stopwatch.
- *I am tongue-tied when the examiner asks questions.* Prepare answers to commonly asked questions – not just to learn content, but to be able to articulate what you know. Preparing answers will help you be smoother and faster, but remember not just to vomit a template, but tailor your answer to *this* patient you are seeing.

Abdomen: Scars & Stoma

This short case is provided in the form of a sample OSCE; a generic script is appended at the end.

SAMPLE OSCE

Candidate information

Mr Beng is a 50-year-old Chinese gentleman who just underwent abdominal surgery. Please take a brief history and examine his abdomen.



Patient / Scenario information

Mr Beng is a 50-year-old Chinese gentleman who presented with a 3-day history of abdominal pain, distension, nausea, vomiting, and constipation; just before presenting to A&E he had severe unrelenting abdominal pain, made worse by any movement. X-ray showed free air under the diaphragm and therefore Mr Beng was brought to emergency theatre. Laparotomy found perforated descending colon tumor. Tumor resection, end colostomy, and closure of rectal stump (Hartmann's procedure) was performed. *[The patient will however deny knowledge of the diagnosis]*

On examination, the stoma bag has been removed but (if asked) it was draining semisolid brown contents. Abdomen is soft non-tender but there is nodular hepatomegaly. Otherwise there is no ascites, other masses, peritoneal nodules, or lymph nodes. The anus is patent and it is empty. The patient is generally well, non-toxic, not jaundiced.

Suggested questions (see rubric for answers):

- What kind of stoma is this?
- What operation was done?
- What is the patient's underlying condition?
 - Follow-up question: even if obstructed, why Hartmann's instead of resection with defunctioning stoma? → not all obstruction requires Hartmann's; usually it's an emergency e.g. perforation, impending perforation (e.g. cecum >9cm), closed-loop obstruction.
- Why choose this operation instead of primary anastomosis
- What other types of stoma do you know

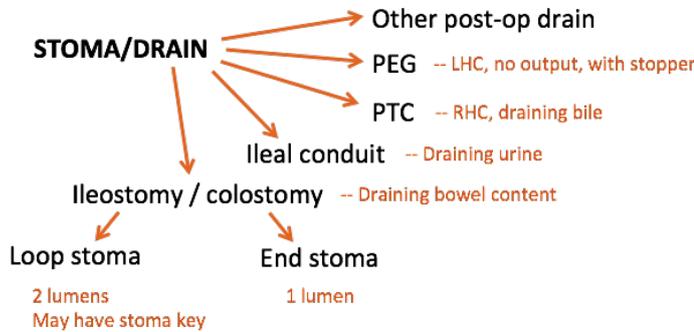
Marking Rubric

		Performed and competent	Performed but NOT fully competent	Not performed or incompetent
History & Examination				
1	Greet patient and introduction	2	1	0
2	Elicit presentation → IO	2	1	0
Examination				
3	Identify the end colostomy and justify why - single large lumen - flush with skin - draining semisolid content	2	1	0
4	Looking for stoma complications - Mucosa pink - No prolapse/retraction - No parastomal hernia - No skin excoriation - Offer I/O charts, per-stoma exam	2	1	0
5	Examining overall condition / abdomen - No cachexia / jaundice - No other organomegaly - Identifying hepatomegaly - No ascites	2	1	0
6	Requesting to look for an anus - present	2	1	0
Discussion				
7	Identify operation: Hartmann procedure	2	1	0
8	Identify cause: metastatic colon cancer complicated by perforation / impending perforation / closed loop obstruction /	2	1	0
9	Understands the choice of Hartmann procedure over primary anastomosis	2	1	0
10	Knows other types of stoma and their indications	2	1	0

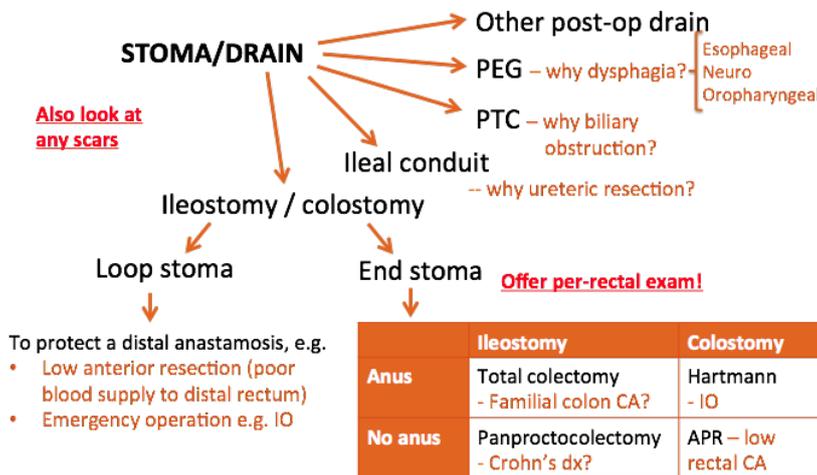
GENERAL APPROACH

To every stoma, ask several questions:

1. What kind of stoma is this?



2. What operation was performed and why?



3. Are there any complications?

- Look at the stoma
 - Is mucosa pink? – *necrosis (dusky), infection, bleeding*
 - Is it in situ? – *prolapse, retraction*
- Do cough impulse – *parastomal hernia*
- Offer to remove stoma base plate – *Skin excoriation*
- Offer per-stoma examination
 - Is it patent? – *fecal impaction, stenosis*
- Examine hydration, ask for I/O – *Stomal diarrhoea*

GENERIC SCRIPT

Brief history

- Age, past medical hx
- What made you have this operation? (Try to fish underlying condition and procedure. If colorectal cancer, try to localize)
- Has the stoma given you problems?

Examination: Sir, my patient is an elderly Chinese gentleman who is alert and comfortable at rest. He does not appear cachectic.

On inspection of the abdomen I notice the presence of a midline laparotomy scar, one drain scar in the right iliac fossa, and a stoma in the right iliac fossa. **Uncle can you cough please?** There is no incisional hernia [every time you see a scar, cough!]

Uncle, may I touch the stoma?

- I note that the stoma is draining liquid fecal contents into a stoma bag. It has a small diameter and is spouted. There are two lumens. This is consistent with a loop ileostomy.
- I note that the stoma is draining liquid fecal contents into a stomal bag. It has a small diameter and is spouted. I only note one lumen. It may be an end ileostomy.
- I note that the stoma is draining semisolid fecal material into a stoma bag. It has a large diameter and is flush with skin. There are two lumens. This is a loop colostomy
- I note that the stoma is draining semisolid fecal material into a stoma bag. It has a large diameter and is flush with skin. There is only one obvious lumen. This is most likely an end colostomy
- I note that the stoma is draining urine coloured liquid into a stoma bag. It is small diameter and spouted. It is an ileal conduit.

The stoma mucosa is pink. There is no dehiscence, retraction, or prolapse. **Uncle can you cough please?** I do not note any parastomal hernia.

I will now examine the rest of the abdomen. My abdominal examination is otherwise unremarkable; the abdomen is soft non-tender, i do not note any organomegaly. The peripheries are also unremarkable with no cachexia, jaundice, or pallor. The patient is well hydrated.

I will like to complete my examination by

- Doing a digital rectal examination and (if it an end stoma) in particular I want to know if there is an anus
- Removing the stoma base plate to look for skin excoriation
- Doing a per-stoma examination to look for stenosis
- Looking at the i/o charts for stomal diarrhoea.

Summary: In summary my patient is an elderly Chinese gentleman with

- A midline laparotomy scar and a loop ileostomy / colostomy. Given his history which is suggestive of colorectal cancer, he probably had a colectomy with defunctioning ileostomy / colostomy. The stoma is healthy.
- A loop ileostomy but no other surgical scar. The patient gives a history suggestive of rectal cancer. I suspect that he has a defunctioning ileostomy in preparation for neoadjuvant chemoradiotherapy.
- A midline laparotomy scar and an end colostomy. If he has no anus this is probably an abdominal perineal resection and he does give a history suggestive of low rectal cancer / if he has an anus this could be a Hartmann's operation and he does give a history suggestive of emergency operation for intestinal obstruction. Unfortunately I also note hepatomegaly and cachexia which could suggest disease recurrence with metastasis.
- A midline laparotomy scar and an end ileostomy. If he has no anus he probably had a panproctocolectomy and he gives a history consistent with ulcerative colitis. (If he has anus - a total colectomy; perhaps less common as you would do an ileal pouch anal anastomosis in this case).
- A Pfannestiel scar and a ileal conduit. He most probably has had a radical cystectomy for bladder cancer.

QUESTIONS

What are the indications for a stoma?

Stomas may be used for input or output. An example of the former is the percutaneous endoscopic gastrostomy used for feeding. Output stomas may be defunctioning or end stomas. Defunctioning stomas rest distal bowel for example to protect an anastomosis post resection. End stomas are used when the distal bowel has been resected. Apart from colorectal stomas, other types of stomas include the ileal conduit and percutaneous transhepatic biliary drain.

What are the complications of a stoma?

Sir, the complications of a stoma can be divided into early and late. Early complications include stomal diarrhoea, necrosis, bleeding, and fecal impaction. Late complications include prolapse, retraction, parastomal hernia, skin excoriation, and psychological problems.

What are the principles of siting a stoma?

A stoma should be sited away from the surgical scar, belt line, bony prominences, and skin creases. It is preferable to go through the rectus sheath (lower risk of prolapse, retraction, or parastomal hernia) and to ensure no tension in the bowel loop (risk of ischemia).

Picture source: surgicalexam.com

Abdomen: Umbilical Hernia

contributions from Darius Pan

SAMPLE OSCE

Candidate information

You are the resident in the surgical ward. A M5 SIP student tells you that there is a patient with abnormal abdominal finding. Please examine this patient's abdomen and explain each step of the exam and your findings to the examiner. There is no need to take a history.

Findings

- Midline laparotomy scar
- Central abdominal mass with positive cough impulse:
- LIF stoma with parastomal hernia
- Ascites, splenomegaly



Presentation: paraumbilical hernia secondary to increased intra-abdominal pressure from ascites; concurrent LIF stoma with parastomal hernia

QUESTIONS

How to differentiate between paraumbilical and umbilical hernia?

- Umbilical hernia (the above): umbilicus everts as a round central lump, with skin of the center of the umbilicus attached to the center of the sac
- Paraumbilical hernia has the umbilicus is pushed to one side and stretched into a crescent shape pit. The umbilical skin is pushed to the side of the sac (and not center of the sac) If the umbilical pit is too deep to clean, it may produce foul-smelling discharge or dried sebaceous secretion (ompholith)

Which is more common

- Paraumbilical hernia – particularly develops in middle and old age, more common in women, obese, multiparity

What are the contents of a paraumbilical hernia?

- Contains extraperitoneal fat and omentum
- If hernia contents are strangulated, present with discomfort and tenderness around the umbilicus made worse by prolonged standing/strenuous exercise
- Does not cause bowel obstruction

When might a patient have umbilical hernia

- Acquired umbilical hernia: secondary to raised intra-abdominal pressure
- Congenital umbilical hernia: usually disappear spontaneously during the first few years of life
 - If there is still a defect at 4 years old (Norman Browse), the hernia is unlikely to seal itself and an operation is required
 - Does not cause any symptoms for patient (only parental anxiety)

How will you manage this hernia?

- First, manage underlying risk factors for increased intra-abdominal pressure
 - Weight loss, Change in job position, Avoid heavy lifting
 - Treat co-morbidities: Chronic cough, Ascites, BPH, Constipation
- Operative management – Hernia Repair
 - Mayo's "Vest Over Pants" operation
 - Upper and lower edges of defect is overlapped using large non-absorbable sutures, such that the lower edge (pants) is pulled under the upper edge (vest)
 - Unfortunately also commonly referred to locally as 没有 repair due to high recurrence rates
 - Tension free mesh repair
 - If done open, there will be an infraumbilical smiley faced scar
 - If done laparoscopic, there will be several port scars surrounding the umbilicus in the periphery of the abdomen

What are the risk factors for parastomal hernia?

- Patient factors
- Poor wound healing: advanced age, diabetes, cancer, steroids, immunosuppressants, smoking, malnutrition
- Increased intra-abdominal pressure: Chronic Cough, COPD, Obesity, Ascites, Constipation, BPH, Heavy lifting
- Early mobilization post-op
- Technical factors:
- Type of Surgery: Emergency surgery > Elective Surgery
- Type of stoma: Colostomy > Ileostomy, Loop > End
- Wound infection

What do you think is the cause of this patient's hernia?

- Increased intra-abdominal pressure from underlying ascites from portal hypertension

How do you manage parastomal hernia?

- Conservative: surgical repair is generally avoided due to the propensity for parastomal hernia to recur
 - Educate patient about symptoms of bowel obstruction and strangulation, instruct to seek medical attention if such symptoms occur
 - Stoma belt (ostomy binder) to provide stability around the stoma site to minimize bulging at the skin level
- Surgery: Revision of stoma with prosthetic mesh repair indicated if
 - Incarcerated hernia
 - Stoma appliance dysfunction and leakage
 - Peristomal skin breakdown

Breast Lump

GENERIC SCRIPT

Brief History:

- Age
- Where is the lump? When did you first notice it?
- Has it been increasing in size? If so, how fast?
- Any pain? Any nipple discharge? LOW LOA?
- Family history of breast cancer
- Any previous procedures, mammogram, scans

Examination: Sir, my patient is an elderly Chinese lady who is alert and comfortable. She does not look cachectic.

Ma'am can you sit at the edge of the bed and put your hands behind your head? Ma'am can you put your hands on your hips and press down? On inspection, the breasts are symmetrical with no obvious masses, scars, skin changes or nipple changes. There is no skin tethering.

Ma'am can you now lie back and put your hands behind your head again? On palpation, I note a lump in the 10 o'clock position, 2cm from the nipple. This measures 4cm by 4cm in diameter. It is hard and irregular. It is non-tender and not warm, not fixed to the overlying skin or underlying structures. There are no other masses felt in the right breast, including the retroareolar area and the axillary tail. The contralateral breast is normal. **Ma'am please rest your right arm on my arm. I am going to check your armpits.** There is a small non-tender mobile node about 1.5cm by 1.5cm. **Ok the other arm now.** The left axilla is normal. **Ma'am can you sit forward again please. I'm now going to feel your neck.** There is no cervical lymphadenopathy.

Ma'am, do you have any discharge from the breast? (If yes, demonstrate). My patient is not able to express any nipple discharge.

I would like to complete my examination by:

- Percussing the spine for tenderness
- Auscultation of the lungs
- Abdominal examination for hepatomegaly

Summary: In summary, this is an elderly Chinese lady with a painless progressively growing right breast lump. Examination reveals a hard lump with right axillary lymph node, worrisome for mitotic lesion

QUESTIONS

(also see breast lump case)

What are the differentials for a breast lump?

- Benign: breast cyst, fibroadenoma, breast abscess, fat necrosis
- Malignant: breast cancer (ductal, lobular, paget's)

How would you investigate this mass?

- I would complete the triple assessment. This will involve a mammogram for radiological assessment, and a core biopsy for histological assessment.

What is the BIRADS scoring system, and what would you do for each score?

- The BIRADS acronym stands for Breast Imaging - Reporting and Data System. It is a system for reporting risk assessment based on radiological imaging of the breast.
- Scores:
 - 0 : incomplete, further imaging or information is required.
 - 1 : negative – symmetrical, no masses, architectural disturbances, calcifications
 - 2 : benign findings, e.g. fibroadenomas, simple cyst, lipomas
 - 3 : probably benign; short interval follow-up needed
 - 4 : suspicious for malignancy
 - 5 : highly suspicious for malignancy
 - 6 : known biopsy-proven malignancy

How would you manage this patient? (remember to tailor to this patient)

- Confirmation of cancer
- Staging scans - CT TAP
- Then,
- Aim for local control of the disease with surgery - either breast conserving (+ compulsory adjuvant radiotherapy) or simple mastectomy
- Assess need for systemic therapy by assessing involvement of regional lymph nodes, either by sentinel lymph node biopsy or axillary clearance
- If there is regional lymph node involvement then systemic therapy is needed - either chemotherapy, hormonal therapy, targeted therapy

Groin Lump: Inguinal Hernia

GENERIC SCRIPT

Brief History

- Age, Occupation, PMHx (COPD, BPH, abdo mass, liver cirrhosis with gross ascites), SHx (previous hernia repairs?), drug allergy
- Where is the swelling, duration of swelling
- Any symptoms such as pain over swelling, generalized colicky abdominal pain, vomiting or no BO or pass flatus

Initial examination - First determine if scrotal or inguinal swelling (*please remember to glove*)

1. On inspection there is an inguinal swelling not descending into the scrotum. (Later: demonstrate testes is separately palpable)
2. On inspection there is a right inguinoscrotal swelling (Later: demonstrate testes is separately palpable).
3. On inspection there is a right scrotal swelling. I am now palpating the spermatic cord to determine if it is purely a scrotal swelling (feel normal spermatic cord) or an inguinal scrotal swelling (can feel a thicker structure - hernia contents) – *see next approach on scrotal swelling*

INDIRECT HERNIA

Examination: Sir, my patient is a middle aged indian gentleman who is alert and comfortable.

On inspection, I see a large 8cm by 9cm right inguino-scrotal swelling. There is a longitudinal scar along the left groin, likely from a previous hernia repair. There are no overlying skin changes or sinuses.

On palpation, the swelling is soft, 3cm by 3cm in diameter, not fixed to skin or underlying structures, not warm or tender. It has a positive cough impulse. There is no cough impulse over the contralateral groin. I am unable to get above the scrotal portion of the swelling. The right testes is separately palpable from the swelling in the scrotal portion. I was unable to palpate the spermatic cord on the right, as I did on the left.

I am now surface marking the inguinal ligament which is a line drawn between the pubic tubercle (not symphysis unlike femoral pulse) and anterior superior iliac spine, I note that the swelling starts from a point superior and medial to the inguinal ligament therefore it is an inguinal hernia.

[Lie patient down] The patient was able to reduce the swelling himself partially. I am able to help him reduce the swelling completely. I am now surface marking the deep inguinal ring, which is located 1cm superior to the midpoint of the inguinal ligament (not same as mid-inguinal point). I am now occluding the deep ring. **Sir can you please stand up together with me. Now cough one more time.** The hernia is controlled with occlusion of the deep inguinal ring, therefore this is likely to be an indirect inguinal hernia. I am now going to release my pressure on the deep inguinal ring. **Sir can you cough one more time.** The hernia reappears.

On auscultation of the swelling, there were active bowel sounds heard but no tinkling.

I would like to complete my examination by doing:

- Abdominal exam: scars, masses, ascites, ARU, constipation, IO
- DRE for BPH, impacted stools
- Respiratory exam for any cause of chronic cough.

Summary: In summary, this is a pleasant middle aged Eurasian gentleman with previous left inguinal hernia s/p repair, now presenting with a recurrent indirect right inguinal hernia. It is not incarcerated, strangulated, or complicated by intestinal obstruction. He does not have any underlying causes of increased intra-abdominal pressure.

DIRECT HERNIA

Examination: Sir, my patient is an elderly Malay gentleman who is alert and comfortable.

On inspection, I see a large 8cm by 9cm swelling over the right groin. I do not note any scars, overlying skin changes or sinuses over both groins.

On palpation, the swelling is not warm or tender, soft in consistency. It has a positive cough impulse. There is no cough impulse over the contralateral groin. The right testes is separately palpable from the swelling in the scrotal portion. I am able to palpate the spermatic cord on the right. After surface marking the inguinal ligament, found between the pubic tubercle and anterior superior iliac spine, I note that the swelling is located superior and medial to the inguinal ligament.

[Lie patient down] The patient was able to reduce the swelling himself completely. I am now re-surface marking and occluding the deep inguinal ring, found at the midpoint of the inguinal ligament. ***sir can you please stand up again*** The swelling reappears again even while I am still occluding the deep inguinal ring. I am now releasing the pressure over the deep inguinal ring. ***sir can you cough again*** There is no additional indirect component to the hernia (both sides - Pantaloon hernia).

On auscultation of the swelling, there were active bowel sounds heard but no tinkling.

I would like to complete my examination by doing:

- Abdominal exam: scars, masses, ascites, ARU, constipation, IO
- DRE for BPH, impacted stools
- Respiratory exam for any cause of chronic cough.

Summary: In summary, this is an elderly Chinese man with a right reducible direct inguinal hernia for 3 years, no complications of intestinal obstruction and no underlying causes of increased intra-abdominal pressure.

QUESTIONS

What are the differentials to a groin lump?

Sir, I would divide my differentials according to the structures present around the area

- Hernia: inguinal, femoral
- Vascular: femoral artery aneurysm, saphena varix
- Lymphatics: lymph node, lymphoma
- Soft tissue/bone: lipoma, sebaceous cyst, groin abscess, bone tumour
- Nerves: neuroma of femoral nerve
- Undescended testes, hydrocoele of spermatic cord

What is a hernia?

Protrusion of an organ through an opening in the wall of the cavity in which it is normally contained

What are the types of hernias you know?

- Inguinal hernia
- Direct - hernia through the weakness in the posterior wall of the canal, within the Hasselbach's triangle (medially rectus abdominis, inferiorly inguinal ligament, laterally inferior epigastric artery)
- Indirect - hernia through the deep ring of the inguinal canal
- Pantaloon - both direct and indirect components
- Femoral hernia
- Abdominal wall hernias
- Umbilical/paraumbilical hernia
- Incisional hernia
- Spigelian hernia - hernia through spigelian fascia, the aponeurotic layer between the rectus abdominis muscle medially, and the semilunar line laterally
- Richter's hernia - hernia involving only part of bowel (rather than entire circumference), knuckle of bowel is strangulated but lumen is patent
- Parastomal hernia

- Other special types
- Sliding hernia - contains retroperitoneal structures eg bladder, caecum, sigmoid
- Little's hernia - contains Meckel's diverticulum
- Amyhen hernia - contains appendix
- Intracranial hernias
- Diaphragmatic hernia

What are the common complications of hernias?

Incarceration → obstruction → strangulation (6 hours to bowel turning gangrenous)

What are the borders of the inguinal canal?

A useful mnemonic is MALT (2Ms, 2As, 2Ls, 2 Ts)

- Superior wall (roof): 2 **M**uscles
 - Internal oblique
 - Transverse abdominis
- Anterior wall: 2 **A**poneuroses
 - Aponeurosis of internal oblique
 - Aponeurosis of external oblique
- Inferior wall: 2 **L**igaments
 - Inguinal ligament
 - Lacunar ligament
- Posterior wall: 2 **T**s
 - Transversalis fascia
 - Conjoint **T**endon

Why do femoral hernias tend to strangulate much more than inguinal hernias?

- Femoral hernias have a narrower neck.
- The boundaries of the femoral ring are on 3 out of 4 sides made of rigid structures:
- Anteriorly is inguinal ligament
- Medially is lacunar ligament
- Posteriorly is the superior pubic rami
- Laterally is the only compressible structure - the femoral vein

What treatment would you offer this patient?

Tailor your answer to the patient for example

- Sir my patient is not bothered by his hernia and it is not complicated. I would not do anything / offer conservative options such as lifestyle modification (weight loss, change jobs, reduce heavy lifting), treatment of underlying medical conditions (COPD, BPH, constipation), and the option of abdominal binders.
- Sir my patient has bilateral hernias I want to do laparoscopic repair so the patient doesn't get two scars.
- Sir I want to do open repair to reduce the risk of recurrence for my patient

How do you choose between the surgical options?

- Generally open repair lower recurrence, laparoscopic repair faster recovery.
- Open repair includes tension free mesh (Lichtenstein) and non-mesh (Shouldice) techniques. Favour in -
 - Complicated hernia (e.g. obstruction, incarceration)
 - Prior abdominal/pelvic surgery
 - COPD or lung disease, may not tolerate pneumoperitoneum
- Laparoscopic repair includes transabdominal preperitoneal (TAPP) or totally extraperitoneal (TEP) options, favour in -
 - Bilateral hernias: no need two scars.
 - Recurrent hernias: do repair in a previously undissected tissue plane
 - Patient preference

What are the post-op complications to look out for?

- Post-op complications can be divided into those related to general anesthesia and those related to the surgery.
- Complications related to local anesthesia include allergy to lignocaine.
- Complications related to procedure itself can be further divided into
 - Immediate: acute urinary retention, bruising, scrotal hematoma, injury to vas deferens → infertility, injury to autonomic nerves → urinary incontinence, impotence
 - Early: infection of wound mesh, wound dehiscence, scrotal hematoma
 - Late: recurrence of hernia, testicular atrophy from testicular artery damage, ischemic orchitis from damage to pampiniform plexus draining the testes

What are the structures in the spermatic cord?

- Three arteries: testicular artery (from aorta), artery to vas deferens (from internal iliac), and cremasteric artery (from inferior epigastric)
- Three nerves: ilioinguinal nerve, nerve to cremaster, and autonomic nerves
- Three others: vas deferens, pampiniform plexus, lymphatics

Groin Lump: Scrotal Swelling

GENERIC SCRIPT

Brief History:

- Age
- Where is the swelling, duration of swelling
- Any symptoms such as pain, fever, LOW LOA bone pain SOB

Examination: Please refer to 'approaches to symptoms of disease' for a list of differentials and a suggested approach to the scrotal swelling. Some examples given below -

TESTICULAR TUMOR

History: painless progressive testicular enlargement

Examination: Sir, my patient is an elderly Chinese gentleman who is alert and comfortable. On inspection, I see that the right scrotum is grossly enlarged. There are no scars, nor overlying skin changes or sinuses.

On palpation, there is a mass within the scrotum. I am able to get over it. I am able to feel the right spermatic cord. However I am unable to feel the right testes separately from the mass. It measures about 5cm by 5cm, is hard and irregular, not warm or tender, not attached to overlying skin and still mobile within the scrotal sac. It is not transilluminable. Contralateral testes was normal.

I would like to complete my examination by palpating the abdomen for any organomegaly, percussing the spine for tenderness and auscultating the lungs.

Summary: In summary, this is a elderly Malay gentleman with a testicular mass, likely mitotic

How would you investigate?

- Tumour markers: Beta-HCG, alpha fetoprotein, lactate dehydrogenase
- US scrotum to characterize the mass further
- Staging scan: CT thorax abdomen pelvis, looking for lymph node involvement and distant metastasis

What surgery is the patient likely to require?

Right radical orchidectomy via the inguinal approach

VARICOCELE

History: swelling over the past 3 months, with LOW LOA

Examination: Sir, my patient is an elderly Chinese gentleman who is alert and comfortable. On inspection, there is a tortuous mass in the left scrotum with a 'bag of worms' appearance. There are no scars, nor overlying skin changes or sinuses.

On palpation, the mass is soft and compressible and feels like a bag of worms. It extends upwards towards the groin and measures 4cm by 8cm. It is not attached to overlying skin and still mobile within the scrotal sac. It is not warm or tender. **Sir can you please hold your breath, pinch your nose and blow out?** The mass is accentuated with valsalva maneuver. I am able to feel the right spermatic cord, as well as the testes separately from the mass. Contralateral scrotum was normal.

I am now going to examine the abdomen looking for any organomegaly, most especially any ballotable masses. There is some left flank fullness. **Sir have you also had any flank pain or blood in your urine recently?**

I would like to complete my examination by percussing the spine for tenderness and auscultating the lungs.

Summary: In summary this is an elderly chinese gentleman with new onset left sided varicocele, associated with flank fullness, hematuria and loss of weight. I would like to rule out a mitotic lesion in the left kidney.

SCROTAL HEMATOMA

History: mass appeared suddenly a few days after a hernia operation. It was tense and tender at first, but now not tender anymore. Size of the mass is stable.

Examination: Sir, my patient is young Bangladeshi gentleman who is alert and comfortable. On inspection, I see that the right scrotum is grossly enlarged. There is a newly healed scar over the right groin, but no overlying skin changes or sinuses.

On palpation, there is a mass within the scrotum. I am able to get over it. I am able to feel the right spermatic cord and right testes separately from the mass. It measures about 3cm by 3cm, is smooth and round and hard, not warm or tender, not attached to the scrotum and is still mobile within the scrotal sac. It is not transilluminable, and is not accentuated on valsalva maneuver. Contralateral testes was normal.

Summary: In summary, this is a pleasant young bangladeshi gentleman with recent inguinal hernia repair, presenting now with a scrotal mass that is likely a scrotal hematoma.

Neck Lump: Lymph Node

GENERIC SCRIPT

Brief History:

- Age, point to lump
- Time course: when did it start, has it been growing
- Pain
- Any lumps elsewhere
- Suggestions of etiology
- Malignant: any loss of weight / blood stained nasal discharge / hearing loss
- Infective: URTI, night sweats, chronic cough, FHx TB.
- Inflammatory:

Examination: Sir, my patient is an elderly Chinese gentleman who is alert and comfortable at rest. On inspection I note a small round swelling in the right posterior triangle of his neck measuring 2cm by 2cm in diameter. There are no overlying skin changes, no sinuses, and no scars. I do not note any other visible lumps. My patient does not appear cachectic.

On palpation, the lump is firm, not erythematous, non-tender, and non-pulsatile. It is mobile in all directions and not attached to underlying structures or overlying skin. Examining the entire neck, I do not note any other lumps.

The lump appears to be a lymph node and I will proceed to examine the areas that drain into this lymph node. Beginning with the skin of the head and neck region, I do not note any growths or infective lesions. **Sir can you open your mouth please?** I do not note any lesions in the oral cavity. The thyroid and salivary glands are not enlarged. Examining the oral cavity, I do not note any abnormality. Performing a quick screen of the cranial nerves, I do not note any cranial nerve palsy which can suggest nasopharyngeal mitotic lesion. The patient's voice is not hoarse.

I will like to complete my examination by

- Perform otoscopy looking for any mitotic lesion and otitis media with effusion that may suggest nasopharyngeal mitotic lesion.
- Auscultating the lung for any consolidation or unilateral effusion
- Palpating the abdomen for hepatosplenomegaly or gastric mass.
- Examine other lymph node groups which could suggest lymphoma.

Summary: In summary my patient is an elderly Chinese gentleman with a painless progressively enlarging lump in the the posterior triangle of his neck which is most likely a lymph node. My differentials are that of a chronic infective lesion such as tuberculous disease or a mitotic lesion. I do not note any obvious infective or mitotic lesion in the draining areas to this node.

QUESTIONS

Apart from lymph nodes, what are some other causes of a neck lump?

Sir, I will like to divide the causes of a neck lump by anatomical region.

- Midline lumps include thyroid and thyroglossal cyst.
- Other anterior triangle lumps include submandibular swellings, schwannoma (mobile side to side but not up and down), carotid body tumor (as for schwannoma but also pulsatile), and branchial cleft cyst.
- Posterior triangle lumps include cystic hygroma, cervical rib, and pharyngeal pouch.

What are some etiologies of this gentleman's enlarged lymph node?

- Sir, the etiologies of lymphadenopathy can be divided into infective, neoplastic, and inflammatory.
- In this gentleman with a worrying history I am particularly concerned about a chronic infection such as tuberculosis, as well as mitotic lesions especially in the head and neck region such as NPC.
- If there are other areas of lymphadenopathy I am also worried about lymphoma.

See approaches to symptoms of disease for an approach to lymphadenopathy. Think in terms of pathologic category and location.

Assume his lung and abdomen is normal and there are no other lymph nodes. How will you investigate this gentleman?

- Sir, I need to refer this gentleman to the ENT surgeon to do flexible nasoendoscopy, and to the surgeon for OGD.
- He may need a CT of the neck looking for other enlarged lymph nodes, and chest X ray looking for lung nodules or TB.
- The enlarged lymph node may also be biopsied (FNAC vs excision biopsy)

What are the lymph node levels you know of?

Level	Location	Drainage area
Level 1	Submandibular	Oral cavity, submandibular gland
Level 2	Internal jugular from skull base to carotid bifurcation	Nasal pharynx, oral pharynx, parotid, superglottic larynx
Level 3	Internal jugular below carotid bifurcation to omohyoid	Oral pharynx, hypopharynx, superglottic larynx
Level 4	Internal jugular below omohyoid	Subglottic larynx, hypopharynx, esophagus, thyroid
Level 5	Posterior triangle	Nasal pharynx, oral pharynx
Level 6, 7	Adjacent to thyroid; Tracheal esophageal groove and superior mediastinum	Thyroid, larynx, lung

Note: Bilateral nodes can occur with cancers of soft palate, tongue, epiglottis, and nasal pharynx.

Neck Lump: Parotid

GENERIC SCRIPT

Brief History

- Age
- How long have you had this swelling?
- Has it been increasing in size? If so, how fast?
- Is it painful?
- Any other swellings?

Examination: Sir, my patient is a middle aged Chinese gentleman who is alert and comfortable. On inspection I note a swelling over the right angle of the jaw that is lifting the earlobe. There are no overlying skin changes, no scars or sinuses. I also do not note any obvious facial droop although I will examine the facial nerve again later.

On palpation, the mass is not warm or tender. It measures 5cm by 6cm in size, is hard and irregular. The skin is mobile over it, and it is not fixed down to the underlying structures. I will now palpate the neck looking for cervical lymphadenopathy. There is no cervical lymphadenopathy.

I will now examine the branches of the facial nerve. First the temporal branch - sir can you raise your eyebrows. Then the zygomatic branch - can you close your eye as hard as you can. Then the buccal branch - can you smile for me. And finally the marginal mandibular branch - sir say eeeeeee.

I will now like to look inside the oral cavity and palpate the swelling bimanually (don gloves, take pen torch and tongue depressor). **Sir I'm going to use this stick to press down your tongue and look into your mouth.** I am able to visualize the posterior arches and note that they are not pushed medially to suggest involvement of the deep lobe of the parotid. **Sir can you lift up your tongue.** I note that the submandibular glands are normal and symmetrical in appearance, secreting saliva. **Ok put down your tongue.** I am now visualizing the opening of the parotid duct, located opposite the second upper molar. I do not see any discharge or stones coming out of the opening. **Sir I'm going to put 2nd and 3rd finger into the right side of your mouth and feel the lump from both sides.** The swelling is palpable bimanually.

Summary: In summary, this is a middle aged Chinese gentleman with a painless right parotid mass that has been slowly increasing in size over the past 6 months. There is no skin or facial nerve involvement.

QUESTIONS

What are the causes of parotid swelling?

- Sir, I will divide the causes of parotid swelling into painful and painless causes.
- Painful causes include sialolithiasis, sialadenitis, and mumps virus infection.
- Painless causes include neoplasms, sjogren's syndrome, and alcoholic liver disease.
- The salivary gland neoplasms can be divided into benign and malignant types.
- Benign neoplasms are pleomorphic adenoma and warthin's tumor, which can be bilateral.
- Malignant tumors include adenoid cystic carcinoma, mucoepidermoid carcinoma, carcinoma ex pleomorphic, and squamous cell carcinoma.

How would you investigate this mass?

Sir, I would want to do a fine needle aspiration cytology as well as MRI Head and Neck, keep in view thorax, abdomen, pelvis.

What is the 80% rule?

80% of salivary gland neoplasms occur in the parotid, of which 80% are benign. 80% of the benign tumours are pleomorphic adenomas.

What are types of parotid cancers that you know of?

- Epithelial: mucoepidermoid, adenoid cystic, carcinoma-ex-pleomorphic, SCC, adenocarcinoma, undifferentiated
- Non-epithelial: lymphoma, hemangiopericytomas, rhabdomyosarcomas

This patient has a longstanding parotid lump which suddenly grew bigger in the last 3 months. What is it?

Sir, this is likely a carcinoma ex pleomorphic

What operation is this patient likely to require?

- If tumour only involves the superficial lobe → superficial parotidectomy
- If tumour involves the facial nerve or deep lobe → total parotidectomy, with radical lymph node dissection if nodes are positive, and adjuvant radiotherapy.

What are the common post-op complications?

- Early: bleeding, infection, damage to facial nerve
- Late: Gustatory sweating (Freye's syndrome), facial synkinesis, parotid fistula
- If operation for pleomorphic adenoma → can recur due to incomplete excision as the tumor is not encapsulated

Neck Lump: Thyroid

GENERIC SCRIPT

Brief History

- Age
- Where is the lump? When did you first notice it?
- Has it been increasing in size? If so, how fast?
- Is it painful?
- Symptoms of local invasion: change in voice, difficulty breathing, difficulty swallowing
- Family history of thyroid cancer

Examination: Sir, my patient is an elderly lady who is alert and comfortable at rest, not in respiratory distress, not cachectic looking.

On inspection, there is a visible central neck mass, more prominent on left than right. There are no overlying skin changes or sinuses. There are no scars over the neck. **Ma'am can you take a sip of water and keep it inside your mouth. Only swallow it when I tell you to.** Neck mass moves upwards on swallowing. **Ma'am can you open your mouth. Now, stick out your tongue. Now, keep your mouth open and put your tongue back in.** The mass does not move up on tongue protrusion. Therefore this is a thyroid mass.

Ok Ma'am I'm going to feel your neck from behind now. On palpation, I note a lump in the left lobe of the thyroid measuring 8cm x 5cm. **Ma'am can you please swallow again.** It moves superiorly with swallowing. It is firm, non-tender, not warm. Its surface is smooth, edges well defined, and it is not fixed to overlying skin or underlying structure. It is fluctuant and not transilluminable.

I note that there is another lump in the right lobe of the thyroid measuring 6cm x 5cm. The surface is irregular, slightly nodular, firm in consistency, not fixed to overlying skin or underlying structures. It is not warm or tender. I cannot feel lower border of mass even when it moves upwards during swallowing.

There is no cervical lymphadenopathy

Ma'am I'm going to feel your neck, this may be slightly uncomfortable. I am unable to palpate trachea. There is no voice change to suggest recurrent laryngeal nerve involvement.

Ma'am I'm going to tap lightly across your chest. There is no retrosternal dullness.

I am now going to assess the patient's thyroid status

- There are no fine tremors, acropachy, palmar erythema, sweating palms, AF, hyper-reflexia, proximal myopathy
- There is no ophthalmoplegia, proptosis, exophthalmos, lid lag, chemosis
- There is no pre-tibial myxedema

Summary: In summary, this is an elderly Chinese lady with what is likely to be a large left thyroid cyst on background of multinodular goiter. Another differential could be a large left thyroid cyst with right adenoma. I would like to investigate further to rule out carcinoma.

QUESTIONS

Please refer to chapter on thyroid long case

Peripheral Arterial Disease

Note: Be especially careful of the stem 'please examine the legs' because it may well be arterial, venous, or neuropathic foot! Look hard to ensure you are using the right approach before proceeding. Inspection pays off.

GENERIC SCRIPT

Brief History:

- Age, PMHx, Occupation, drug allergy.
- What's the problem with your leg? > Identify if the patient is a claudicant or critical limb ischaemia (rest pain, tissue loss)
- How long has the problem been for?
- Risk factors: DM HTN HLD smoking, ask about control if there is time.
- Complications: infection?
- How has this been affecting your function?

Examination: Sir, my patient is an elderly Chinese lady who is alert and comfortable.

On inspection of her lower limbs, I notice that her left LL is mottled and blue below the ankle. There is a 4cm x 4cm round ulcer over the dorsum of her left foot, with punched out and gangrenous edges. The tendons of her extensor digitorum were visible at the base of the ulcer, but otherwise it was clean with no discharge and no surrounding erythema. Surrounding the ulcer are arterial skin changes over both lower limbs, namely: hyperpigmentation, shiny and hairless skin. ***Make a show to look between the toes and lift legs to look at back of heels*** There were no other ulcers or gangrenous patches.

On palpation, the left LL is colder than the right LL. Capillary refill time is prolonged at 4s on the left and normal at 2s on the right. **Ma'am I'm going to pull down your pants slightly so I can feel the pulse over your groins. I am palpating the femoral artery at the mid-inguinal point, which is the the midpoint between the anterior superior iliac spine and pubic symphysis (not tubercle unlike hernia).** Femoral pulses are well felt bilaterally. **Ma'am please bend your knees up, I'm going to press into the back of your knees.** Popliteal pulses are well felt bilaterally. I am now palpating the dorsalis pedis on the right which is located one third way down a line drawn from the midpoint between the two malleoli to the first webspace. The right dorsalis pedis pulse is well felt, I will not palpate the left due to the ulcer. I am now palpating the posterior tibial pulse which is one third down a line from from the medial malleolus to the heel. This is not palpable bilaterally.

I would now like to do the Buerger's test. **Ma'am I'm going to lift your leg up, then bring it down to to the side of the bed. As I bring your leg down please sit up at the same time - I will support you ok?** [Negative] There is no elevation pallor, therefore Buerger's is negative and I will not go on to look for dependant rubor [Positive] There is elevation pallor at 30 degrees with dependant rubor.

Sir I would now like to palpate the abdomen for a pulsatile expansile mass.

I would like to complete my examination by

- Palpating all pulses and looking for RR, RF delay
- 4 limb blood pressure
- Performing a full cardiovascular examination
- Listening for carotid bruit, renal bruit and femoral bruit
- Ankle brachial pressure index

Summary: In summary, this is an elderly Chinese lady, a vasculopath with left LL critical ischemia as evidenced by rest pain and tissue loss. Examination confirms peripheral arterial disease which is mainly below knee.

QUESTIONS

What is the definition of critical limb ischemia?

Rest pain of >2 weeks not relieved with opioid analgesia, tissue loss, resting LL arterial pressure of <55mmHg, or ankle brachial pressure index of <0.5.

What investigations do you want to do?

- I will start with basic inx
 - FBC for infection, low platelets, Hb looking for anemia that can also contribute to limb ischemia
 - UECr as she may need contrast study
 - GXM PT PTT as she is likely to require an invasive intervention
- I would also like to do an Ankle brachial pressure index as a very basic assessment to confirm critical limb ischaemia and also a reference point to compare post-op and quantify improvement.
- I would also do an ultrasound arterial duplex as noninvasive test to plan for the surgical procedure.
- With regards to the ulcer, I would do a wound swab and also an Xray foot looking for osteomyelitic changes.

What else do you plan to do now?

- Ensure best medical therapy – aspirin plavix statin taken?
- Analgesia for pain - opiates.
- Start Abx KIV stop/change when wound swab return.
- Revascularize the left LL

What are the methods to revascularize?

- Sir, based on my clinical examination, it is likely distal vessel disease below the popliteal.
- Therefore the options are endovascular distal angioplasty or open bypass if the anatomy allows for adequate takeoff and landing site (evidence shows better long term results with open bypass).

In general, options are

- *Acute limb ischaemia: thrombolysis vs embolectomy*
- *Chronic / critical limb ischaemia: endovascular (angioplasty +/- stenting if proximal) vs open (bypass)*

How would you manage a patient with acute limb ischemia?

- Anti-coagulation: IV heparin bolus 5000 units, followed by infusion 1000 units/hour
- Improve perfusion
 - Correct any hypotension
 - Put leg in dependant position
 - Supplemental oxygen
- Emergent revascularization: either thrombolysis or embolectomy
- Watch for rhabdomyolysis or reperfusion injury → AKI, hyperkalemia
 - Hydrate aggressively, treat hyperkalemia
- Watch for compartment syndrome
 - Pain out of proportion to signs, pain on passive stretch

Peripheral Venous Disease

contributions from Eugene Gan

SAMPLE OSCE

Candidate Information:

Mr Chao Kar is a 68-year-old gentleman who has been having pain in his right leg. Take a quick history and perform a target physical examination.

Scenario & SP instructions:

History: You are a 68 year old coffeeshop waiter with longstanding (>10 year) bilateral varicose veins and a sensation of leg heaviness especially at the end of each day; there is no pain on walking. You have had two ulcers on his right leg for 2-3 years. In the last 2-3 weeks the ulcer on the gaiter region of your right leg has become painful, and it is discharging a foul-smelling liquid; however you are afebrile. The ulcer on the lateral aspect of the right leg is much less problematic, it has never been painful and you are not interested in it. You have had no weight loss. You have never seen a doctor but are coming now because it has been difficult to work in the last 2-3 weeks due to the pain.

Examination: show the candidate the following pictures -

(a) You are bothered by these ugly worms



(b) You are also bothered by this painful ulcer with yellow discharge.



(c) You are absolutely not interested in this ulcer – it doesn't trouble you at all.



Photo sources: drbcshah.com, pcds.org.uk

SAMPLE SCRIPT

Brief History:

- Age, occupation
- Diagnose venous disease and rule out ddx: leg discomfort and swelling especially at the end of the day, better on leg elevation
 - Severity of venous disease: unilateral vs bilateral, duration and progression
 - If presentation is leg pain → any claudication? (pain worse on walking)
 - If presentation is leg pain or swelling → any DVT? (acutely red, swollen, also see PMHx)
- If ulcers present: duration, healing/non-healing, painful/painless
 - Infection: discharge, fever, pain
 - Malignant change: if painless and non-healing, beware marjolin & take quick hx for distant spread
- Any treatment received so far? E.g. has patient tried compression stockings
- Past medical history
 - Red flags for secondary causes of CVI: previous DVT, abdominal masses
 - What may affect management: vasculopath, smoking, allergies
- What is the impact on patient's function?

Examination:

Sir, this is a middle aged Malay lady who is alert and comfortable at rest.

Ma'am could you please stand up? *kneel in front of her for inspection*

- On inspection I see a large ulcer about 8cm by 10cm over the gaiter area of the right leg. It is shallow with sloping edges and a granulating base. There is some yellow discharge and surrounding erythema that suggests infection.
- I note another ulcer higher up on the medial aspect of the calf, measuring 7cm by 8cm. It has irregular, everted edges; and looks fleshy. I am concerned about the possibility of this being a marjolin's ulcer, especially as the patient says this has been painless and non-healing for almost 2 years.
- There are venous skin changes over both lower limbs, namely: hyperpigmentation, atrophic blanche, lipodermatosclerosis.
- There are varicose veins over the both LLs, noted over the greater saphenous vein distributions, but none over the lesser saphenous vein distribution.
- Overall she is has CEAP grade C6 disease.

[subsequent tests are time consuming, may have to do selectively or offer]

On palpation, i note pitting edema in both LLs up to mid shin. There is warmth and tenderness in addition to the erythema surrounding the gaiter area ulcer. The other ulcer is notably non-tender on palpation. Tap test shows transmission of impulse from distal to proximal indicating patency and from proximal to distal indicating incompetence.

I am now auscultating over the varicosities. There is no bruit to suggest arteriovenous malformation.

I am now surface-making the saphenofemoral junction which is located 2.5cm inferior and 2.5cm lateral to the pubic tubercle. **Ma'am can you cough please?** There is no palpable saphena varix and no cough impulse.

I will now like to demonstrate the tourniquet test at the saphenofemoral junction. **Ma'am could you please lie down?. Ma'am I am now going to lift up your left leg, empty the veins and tie a tourniquet around your thigh. Sir (examiner) may I please request for your assistance to support the leg while I apply the tourniquet. Ok ma'am now please stand up.**

When tourniquet is applied to SFJ, there is refilling of the varicosities

- Therefore there is incompetence below level of SFJ
- But I cannot make any conclusions about SFJ itself

When tourniquet is applied to SPJ, there is no refilling of the varicosities

- Therefore the SPJ is incompetent and there is no incompetence below the SPJ

I would like to do Perthe's test to assess the deep venous system (if deep venous system is obstructed, stripping of superficial veins is not an option) - *examiner will probably disallow*

I will also like to palpate the abdomen to ensure there is no mass compressing on the iliac veins.

Palpating the LL pulses, I note that DP, PT pulses are well felt bilaterally. I will also like to do an ankle-brachial pressure index, knowing that one third of venous patients have concomitant arterial disease and that affects management

Summary: In summary, this is a middle aged Malay lady, ex-nurse, who has chronic venous insufficiency with an active ulcer -- this makes her CEAP grade C6. There is also a chronic non-healing ulcer with suspicion for malignant transformation into Marjolin's ulcer. Her symptoms impair her ability to work.

QUESTIONS

What are the differentials of an ulcer in the shin/gaiter's region?

- Venous ulcer
- Squamous cell carcinoma (Marjolin ulcer)
- Medical causes: Pyoderma gangrenosum, vasculitis

Explain the pathophysiology of lipodermatosclerosis.

- Venous hypertension → Edema → Hemosiderin deposition and breakdown → Hyperpigmentation, Inflammation and Fibrosis
- In 2 words: Fibrosing panniculitis

Tell me about CEAP classification. What grade is this patient?

- Sir it is a classification system for venous disease including clinical, etiologic, anatomic, and pathophysiologic components
- C0 no dx, C1 reticular vein, C2 varicose vein, C3 edema, C4 lipodermatosclerosis, C5 healed ulcer, C6 open ulcer
- This patient is C6 in the right leg and C3 in the left leg.

Is it common to have different classes on each leg?

No. Two possibilities:

- One side has been treated
- (Red flag!) Secondary causes of CVI:
 - Post thrombotic e.g. DVT, thrombophlebitis
 - Pelvic mass causing iliac vein compression

How would you investigate?

Sir, my investigations would be directed at evaluating the ulcers, the venous system and the arterial system.

- **Ulcers:** For the active ulcer, I would do a wound swab and right LL XR to rule out OM. For the chronic non-healing ulcer, I would do a punch biopsy of its edge because I am worried about Marjolin's ulcer
- **Venous disease:** I would evaluate with a venous U/S duplex, looking for superficial vein incompetence as well as any deep vein thrombosis which would make stripping contraindicated.
- **Arterial system:** I perform an ankle brachial pressure index and an arterial US duplex. In the presence of peripheral vascular disease, I would be much more cautious in applying compression bandaging as that may tip the patient over into critical limb ischemia! Furthermore, poor vascular supply precludes poor surgical wound healing.

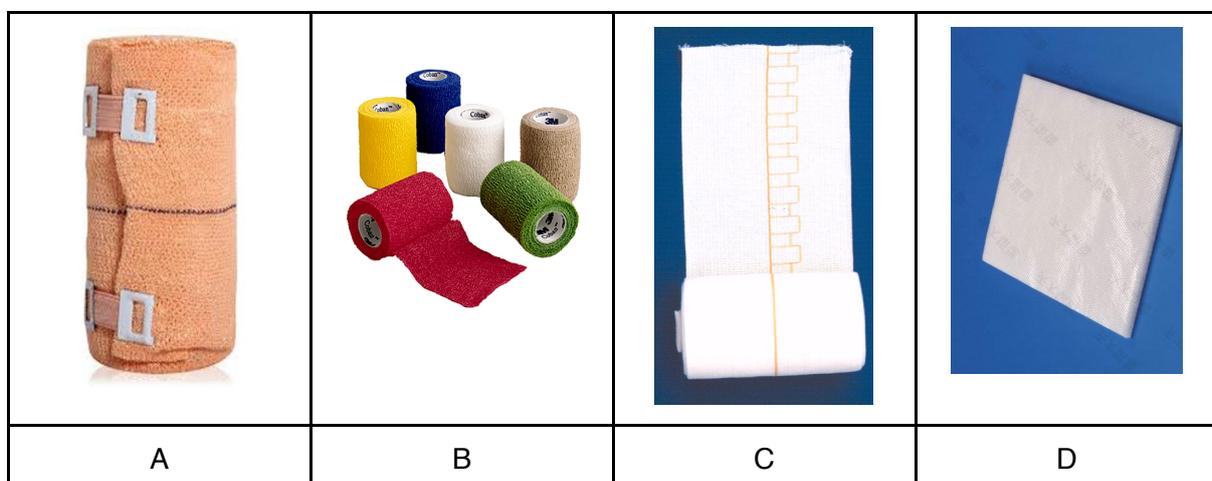
What are the treatment options?

Treatment of the active ulcer:

- Antibiotics, KIV wound debridement
- 4 layer compression bandaging has been shown to be beneficial for ulcer healing

Treatment of varicose veins can be divided into:

- Non-surgical: Lifestyle modification, weight loss
- Symptomatic: Compression stockings, daflon (poor evidence) – not recommended in NICE guideline but commonly practiced
- Surgical:
 - Traditional open surgery: high tie and stripping down to knee and avulsion. In past used to strip down to ankle but high risk of saphenous nerve injury.
 - Endovenous: laser (burn) + avulsion, foam sclerotherapy + avulsion (CLASS study: EVLT and surgery superior to foam), endovascular radiofrequency ablation.

Can you tell me in what order do you apply 4 layer compression stocking?

Apply:

1. Base - non adherent absorptive (e.g. Menolin) dressing followed by wool bandage [D]
2. 2nd layer - Crepe bandage [A]
3. 1st compression layer - e.g. Blue line compression (Elset) bandage [C - has a line]
4. Top compression layer - Adhesive compression bandage (Coban) [B]

What is the difference between 4-layer compression stocking and graduated compression stocking?

- 4 layer compression stocking is for active ulcer → change once a week, applied by nurse at clinic
- Graduated (tighter at bottom) compression stocking is for varicose veins → wear daily, put on yourself

Note: treatment of varicose veins and treatment of venous ulcers are separate things! NO need compression bandage if varicose veins but no ulcer or significant edema

What exactly is high tie with GSV stripping and stab avulsion

High tie and GSV stripping (above knee)

- Ligate the GSV at the SFJ
- Thread a catheter through the GSV to around the level of the knee
- Make an incision in the supero-medial calf
- Strip the vein, guided by the catheter, through the superio-medial calf incision

Stab avulsion (below knee)

- Incision next to any visible varicosity
- Tie the varicosity on either side
- Blunt removal of varicosity

OTHER TIPS

Framework of venous station: think of these 3 components -

1. The ulcer
 - Infected?
 - Malignant? (non healing, recent growth, red flag signs on investigation)
2. The veins
 - The usual scenario: Idiopathic cause
 - Red flags (DVT, Pelvic masses)
3. The underlying arterial system and other co-morbidities (e.g. DM, smoking) that may affect surgical management.

Know how to talk through the special tests

- Where is the SFJ: 2.5cm below and lateral to pubic tubercle (read Browse)
- What is the purpose of the tourniquet: Demonstrate perforator incompetence below the level of occlusion.
- Trendelenburg test: aims to specifically occlude to SFJ instead of placing a tourniquet around the area, but the concept is no different from Tourniquet test.
- Perthes' test: Demonstrate deep venous insufficiency which would contraindicate superficial vein stripping

How to be more efficient with time:

- Make use of key words early on in the examination (e.g. C6 disease) and describe the landmarks as you are palpating (e.g. I am feeling for the SFJ which is at) so that examiners will not need to ask you for them in the Q&A
- Prioritize the issues and verbalize your thought process early in the PE e.g. Sir I am most concerned about the current active ulcer, and then the chronic non healing ulcer. It makes it easier for the examiner to guide you.
- Choose to offer certain tests e.g. Tourniquet, Perthe and wait for examiner to decide if he wants you to do it. Those are time consuming tests.

Skin Lumps & Bumps

GENERAL APPROACH

A general approach is provided in the approaches notes. Broadly, first decide if the epidermis is involved - are there skin changes or is the lump deep to the skin? The commonest lumps are -

- Epidermal growths relevant to the general surgeon: SCC, BCC, melanoma
- Dermal growths relevant to the surgeon: lipoma, sebaceous cyst, neurofibroma, dermoid cyst.
- In the hand there are also ganglion, PVNS, and implantation dermoid [*see approach on hand*]

Describe each lump according to look - feel - move!

LIPOMA

Brief history:

- Name, PMHx, allergies (especially to lignocaine)
- How long has the lump been there, growth
- Any other lumps
- What is the patient concerned about - does it bother him

Examination: Sir, my patient is a middle aged Chinese lady who has a lump on his upper back measuring 3cm by 3cm and spherical in shape. There are no scars, sinuses, and its surface is skin-coloured. On palpation, the lump is soft, not tender or warm. There are distinct / lobulated edges and a positive *slip sign*. The lump is mobile over underlying structures and the skin is mobile over it. It is not pulsatile. **Sir can you please help me to turn off the lights?** It is not transilluminable. I think this is a lipoma. I will like to complete my examination by examining the regional lymph nodes.

What is a condition with multiple lipomas?

Familial multiple lipomatosis

Decum's disease - multiple painful lipomas.

What is the risk of malignancy?

A lipoma does not turn malignant, liposarcomas arise de novo

How do you operate on a lipoma?

- Clean and drape
- Local anaesthesia - lignocaine max 3mg/kg
- Transverse incision
- Dissection in subcutaneous plane around the lipoma using scalpel under direct vision
- Once lipoma freed from surrounding tissue, use forceps to deliver the growth
- Hemostasis
- Palpate to ensure complete removal of the lump
- Buried sutures to close dead space
- Skin closure
- Send for histology
- STO POD7-10

Can you counsel the patient about surgery?

Uncle, if this lump is bothering you, it can be removed using a simple surgery under local anaesthesia - you will be awake the whole time. I just need to make a small cut here and remove the lump. This is generally a very safe procedure, some small risks include allergy to the anaesthetic agent, bleeding, infection. The surgery will leave a small scar and sometimes the scar doesn't heal so nicely. Sometimes the lump can grow again at the same place or elsewhere. If you don't want to take the lump out it is also OK, no harm.

What are Langer lines?

Lines of skin tension corresponding to the natural orientation of collagen fibres in the skin. Incision along these lines result in the most cosmetically pleasing scar.

SEBACEOUS CYST**Brief history**

- Name, PMHx, allergies (especially to lignocaine)
- How long has the lump been there, growth
- Any other lumps
- Cx - infection
- What is the patient concerned about - does it bother him

Examination: Sir, my patient is a middle aged Malay gentleman who has a lump on his upper back measuring 3cm by 3cm and spherical in shape. There are no scars, sinuses, and its surface is skin-coloured. There is a punctum on the skin surface (may or may not be present). On palpation, the lump is firm, not tender or warm. Its edges are well defined and the surface is smooth. The lump is mobile over underlying structures however the skin is not mobile over it (as it is part of the skin layer). It is not pulsatile. **Sir can you please help me to turn off the lights?** It is not transilluminable. I think this is a sebaceous cyst. I will like to complete my examination by examining the regional lymph nodes.

How does a sebaceous cyst form / what is the pathophysiology of a sebaceous cyst?

A sebaceous cyst arises when a sebaceous gland becomes blocked and distends with its own secretion. Histologically, the cyst wall is formed by stratified squamous epithelium derived from the sebaceous gland.

What are the complications of a sebaceous cyst?

- Infection
- Enlargement
- Sebaceous horn formation

What is Gardner's syndrome?

This is an autosomal dominant variant of the familial polyposis coli syndrome. It is characterized by adenomas of the colon, desmoid tumors, skull osteomas, and sebaceous cysts. There is risk of colorectal malignancy.

How do you operate on a sebaceous cyst?

- Clean and drape
- Local anaesthesia - lignocaine max 3mg/kg
- Elliptical incision
- Dissect in subcutaneous plane around the sebaceous cyst using scalpel under direct vision
- Remove in its entirety including the punctum overlying it.
- Hemostasis
- Palpate to ensure complete removal
- Buried sutures to close dead space
- Skin closure
- Send for histology
- STO POD7-10

If infected - usual operation may result in incomplete removal and recurrence. Better to give Abx and operate after no longer infected. Or saucerization.

Please counsel for operation

As above. Mention risk of infection if left alone.

Foot: Diabetic Foot

GENERIC SCRIPT

History as for long case (*truncate for short case focusing on **):

- * Background: Age, PMHx, Drug Allergy, Baseline function (ADL, Occupation)
- * Presenting complaint: ulcer → Explore duration, symptoms, what is bothering
- Presenting complaint: joint deformity → Likely Charcots. Confirm that it is painless *
- Etiology of foot disease
 - Background DM: control*, compliance, admissions for DM emergencies, other end-organ complications
 - Vascular disease*: PVD → Ask for claudication pain, known vascular hx, gangrene
 - Other cardiac risk factors: HTN, HLD, smoking *
- Complications & Management:
 - Any episodes of infection *
 - Is the ulcer healing? *
 - Has the patient required amputation?
- Function *

Examination: Sir, my patient is an elderly Indian gentleman who is alert and comfortable at rest. I note that he walks with a quad-stick and has a large body habitus.

On inspection (**open toewebs and look under sole**) he has a right Charcot's foot with a grossly deformed ankle joint and loss of the arch of the foot. On the sole of the foot under the first metatarsal head, there is a 3cm by 3cm ulcer. It appears to be deep with some slough at its base, but the underlying bone is not visible; its edge is clean with no discharge visible at present. There is some surrounding erythema of the skin. I do not note any skin changes suggestive of arterial disease (shiny skin, loss of hair) or venous insufficiency (lipodermatosclerosis, hyperpigmentation).

I will now like to walk the patient. He does not have an antalgic gait and is walking on the lateral border of his foot which is grossly abnormal.

I will now like to palpate the feet (**wear gloves**). The ulcer is not warm or tender to the touch. The deformed ankle joint is surprisingly non-tender. I will now like to move the joint. There is hypermobility and abnormal movement at this joint which is nontender.

I will now like to test pinprick sensation. Mr _____, This is a satay stick / toothpick, it should feel sharp but not painful. I want to test how well your hands can feel. This (test forehead) is 100%. When I touch your foot can you tell me how many % you can feel? Please close your eyes... (Go proximally until there is sensation) Sir I note that there is loss of pinprick sensation bilaterally in a glove and stocking distribution up to the mid shin. I will now like to test proprioception... proprioception is lost bilaterally.

I will now like to palpate for the peripheral pulses (see arterial examination). The dorsalis pedis and posterior tibial pulses are palpable and the capillary refill time is <2 seconds.

I would also like to take a look at the patient's footwear.

Summary: My patient is an elderly Indian gentleman with a right Charcot's foot and neuropathic ulcer, with bilateral glove and stocking sensory loss. The most likely underlying etiology is diabetes mellitus; corresponding to which this gentleman gives a history of poorly controlled DM. In terms of complications, I note that he is ambulant with a quad stick but lacks appropriate footwear (wears slippers which gives me cause for concern), and I do not note any signs of active infection. I will like to complete my examination by doing a full neurological examination of the lower and upper limbs, look at his temperature chart, examine his heart and do fundoscopy for other end-organ damage of diabetes.

QUESTIONS

What investigations would you like to do for this patient?

Sir I will like to send off some blood tests such as a FBC looking for raised TW, CRP, HbA1c to gauge state of diabetes control. I will like to do a foot Xray to look for OM and do the ankle brachial pressure index. I will also like to do ECG and chest X ray to gauge other cardiovascular dysfunction from diabetes, as well as UECr to look for renal impairment.

If infected - also do blood cultures, wound swab and culture, bone biopsy if OM.

How will you manage this patient?

Sir this ulcer is relatively clean, I will dress the ulcer e.g. with iodine dressing. I will like to send the patient to the podiatry for foot care, and ideally to get an orthosis to relieve the pressure point and prevent further ulceration. If the ankle brachial pressure index is abnormal I will also refer him to the vascular surgeon KIV revascularization. I need to optimise his glycaemic control (KIV endocrine referral) and also manage his comorbidities. I will also like to educate the patient and send him to the diabetic nurse educator.

If infected - debridement, Abx

What are the causes of a foot ulcer?

- Vascular causes include critical limb ischaemia, vasculitis, and venous insufficiency
- Neuropathic causes include diabetes mellitus
- Mitotic causes include squamous cell carcinoma
- Other causes are tuberculous ulcers, pyoderma gangrenosum.
- In many patients the ulcer is multifactorial in etiology.

Foot: Hallux Valgus & Pes Planus

contributions from Joyce Huang

GENERIC SCRIPT

Brief History:

- Age, occupation, PMHx, drug hx & drug allergy
- Presenting symptoms: pain, deformity, ulcers, swelling (bursitis)
 - Where (unilateral or bilateral)?
 - Duration and course
 - What bothers the patient most?
- Differentials:
 - Trauma: any injuries
 - Gout: any acute swelling
 - Referred pain: history of knee or hip problems
- Etiology:
 - Family history
 - RA or ligamentous laxity
 - Footwear: pointed toe, high heels,
- Function - able to walk? Daily activities?

Examination: Sir, my patient is a 60+ Chinese lady who is alert and comfortable. **Maam can you please stay seated for now.**

[Look] On inspection, I see bilateral hallux valgus deformities, with a hallux valgus angle of 40 degrees on the left and 20 degrees on the right. Both big toes are pronated. There is an overriding 2nd toe on the left but not right. The 3rd through 5th toes are clawed. I also note a bunion on the left foot but no overlying erythema to suggest bursitis. There are also calluses over the plantar aspects of both feet and over the dorsal aspect of the PIPJ of the left second toe.

[Feel] On palpation, the left bunion is slightly tender and feels knobby (bursitis). The bases of the metatarsal heads are not tender (metatarsalgia). There is tenderness below the medial malleolus to suggest posterior tibial tendonitis.

[Move] **Ma'am can you move this joint for me?** Dorsiflexion and plantarflexion is restricted and painful in the 1st MTPJ of the left foot, but full and not painful in the right 1st MTPJ. There is no hypermobility of the tarsometatarsal joint to suggest ligamentous laxity.

Ma'am can you stand up please. I note the pes planus in both feet (medial foot arches collapsed) which can be associated with hallux valgus secondary to ligamentous laxity. From behind, there is the too many toes sign and both heels are in valgus. **Ma'am can you tiptoe?** The arches of both feet re-appear on tip-toeing and the valgus of the heels corrects. **Ma'am can you raise your big toe?** Jack's test shows that the pes planus is correctable. **Ma'am can you tiptoe on one foot only?** The patient is unable to do a single foot heel raise, suggesting posterior tibialis tendon insufficiency.

Ma'am, can you walk there and back? The patient has an antalgic gait on the left but is still able to walk steadily unaided.

Ma'am can you show me your footwear? She has pointed heels/shoes with very narrow toe box. There is uneven wear.

Ma'am, let me have a look at your hands. There are no deformities of the small joints of the hands or other deformities of the toes that may suggest rheumatoid arthritis.

I would like to complete my examination by assessing the neurovascular status of the lower limbs (affects surgical plan), examining the ankle and knee.

Summary: In summary, this is a middle aged Chinese lady with bilateral hallux valgus, left worse than right. The left hallux valgus has been complicated by secondary osteoarthritis. Likely etiology is a combination of inappropriate footwear and ligamentous laxity. She also has bilateral pes planus secondary to posterior tibial tendon insufficiency. Functionally her mobility is limited by pain.

Notes:

An alternative sequence is to start inspection standing up.

Whatever is done be sure to describe in terms of forefoot (hallux valgus, bunion, lesser toe deformities), midfoot (pes planus), and hindfoot (valgus). And look - feel - move.

QUESTIONS

You mentioned pes planus. Tell me more about what you would look for.

- Look for symptoms of foot ache esp after prolonged standing
- Loss of medial foot arches, pronation of foot. Heel in valgus with "too many toes" sign.
- I would also determine whether the pes planus is flexible or rigid by making the patient tiptoe
 - Flexible: arch regained on tiptoe
 - Rigid: still no arch on tiptoe, heels fail to invert on tiptoe
- Palpate for tenderness at posterior tibialis, posterior to medial malleolus
- Look for posterior tibial dysfunction - single leg heel raise
- Look for other evidence of ligamentous laxity, neuromuscular disease

What are the causes of pes planus?

- Young children may have physiological pes planus
- In an adolescent, pes planus may be rigid or flexible
 - Rigid: due to bony deformity e.g. tarsal coalition
 - Flexible: idiopathic.
- Developing in older age - posterior tibialis dysfunction.

How would you investigate this patient?

Weight bearing XR of both feet, looking to measure hallux valgus angle (normal is <15 degrees) and intermetatarsal angle (normal is <9 degrees). Also looking for OA changes of the 1st MTPJs.

What are the treatment options for hallux valgus?

- My patient is symptomatic but has yet to have a trial of conservative management, hence I will like to give a trial of conservative management first such as analgesia, proper footwear or orthotics, and physiotherapy.
- If that fails, I could refer for surgery. Surgical options are bunionectomy, realignment osteotomy with soft tissue rebalancing, excision arthroplasty (resection of medial eminence + resection of base of proximal phalanx) or arthrodesis esp since the joint is osteoarthritic.

How do you manage pes planus?

- Conservative - insole, orthotics, analgesia, physiotherapy.
- Surgical - posterior tibialis reconstruction

Where might the other sources of pain be from in hallux valgus?

- Transverse metatarsalgia : due to improper weight-bearing on 1st MTPJ
- Bursitis
- Clawed toe deformity (if present): Pain over metatarsal head, tip of toes, callosities at dorsum of PIPJ

Hand: Ganglion

A note on “Please examine the hands”: In an orthopaedic station, this stem means one of three stations: (1) a nerve lesion, (2) a lumps and bumps station, or (3) deformities e.g. OA or RA. The first task is to decide which of these 3 the station is about. Don't do a nerve exam when there is an obvious ganglion!

GENERIC SCRIPT

Brief history:

- Name, PMHx, allergies
- How long has the lump been there, growth
- Any other lumps
- dDx any penetrating hand injury (implantation dermoid cyst)
- Any problem with the underlying joint - hand OA?
- Any other hand symptoms (neuro deficits, pain, etc)
- Underlying function, occupation, handedness.

Examination: Sir, my patient is an middle aged gentleman. who presents with a hand lump.
Perform hand screening - open fully, close tightly, OK sign.

On inspection I note a 2cm by 2cm lump over the DIPJ which appears spherical in shape. There are no scars, sinuses, and the overlying skin appears normal. There is no punctum. I do not otherwise note any deformities of her hand and there does not appear to be an obvious nerve lesion.

I will now palpate the lump. It is firm, mildly tender on deep palpation, with smooth edges and well defined margins. It is mobile side to side but not in the plane of the flexor tendon, and is not fixed to overlying skin. It appears to be less apparent when the joint is extended. The lump is not pulsatile and Tinel's is negative. It is transilluminable (if big enough to transilluminate).

I note that the movements of the hand are full. There are no Herberden nodes and the patient does not have crepitus in the DIPJs. Functionally the patient is able to write and button his clothes with little difficulty.

Summary: my patient is a middle aged gentleman with a lump over her DIPJ. This is most likely a mucocyst (ganglion of DIPJ) however I would also like to consider a pigmented vilonodular tenosynovitis as my differential.

QUESTIONS

What is a ganglion and where is it usually found?

A ganglion is a fluid-filled outpouching of the synovial membrane of a joint or tendon sheath. It is usually found over the dorsal or radial palmar side of the wrist, over the flexor sheaths, and over the DIPJ.

If it is found on the DIPJ what must you think of?

A ganglion of the DIPJ is called a mucocyst and is associated with OA of the underlying joint.

Can you tell me the difference between a ganglion and PVNS?

A pigmented vilonodular tenosynovitis (tenosynovial giant cell tumor) is a benign growth usually arising from the volar aspect of the fingers. It tends to be firmer than a ganglion, is usually fixed to underlying structure, and does not transilluminate. However it can be difficult to distinguish from a ganglion.

What other lesions can arise on the fingers?

Sebaceous cyst, lipoma, PVNS and implantation dermoid can also occur.

What are the features of OA hands?

OA hands can result in a symmetrical deforming polyarthropathy with bony swellings over the DIPJ called Heberden's nodes. There can also be squaring of the first carpometacarpal joint. The patient may suffer pain and limitation of fine hand movements which can impair hand function.

OK so for this patient, how do you investigate?

- I will like to do ultrasound. A ganglion has well defined margins, thick walls, and appears anechoic.
- I will also like to X ray the underlying joint to look for osteoarthritis.

What is your treatment?

- My patient is not bothered by the ganglion and therefore I am inclined to observe, knowing that a ganglion may spontaneously resolve. Other options which I can discuss with her include aspiration (50% risk recurrence) and excision (risk decreased range of motion, tendon or neurovascular injury, lower risk recurrence).
- Historically the treatment was to hit the ganglion with a Bible which I don't really recommend!
- Other than that I will also like to manage her underlying osteoarthritis such as with activity modification, analgesia, and physiotherapy.

Hand: Nerve Palsy

OVERVIEW

Begin with inspection and a quick screen to identify which nerve is affected- full extension (radial), full flexion (high median), OK sign (high median), and crossing of fingers (ulnar). Then proceed to do the nerve-specific examination (below). End by looking for etiology and examining function.

	Median nerve lesion		Ulnar nerve lesion		Radial nerve lesion	
	Low	High	Low	High	Low	High
Screening findings	Thenar wasting	Thenar wasting Benediction Cannot 'OK'	Claw	Claw Hypothenar wasting	Finger drop	Finger drop
Sensory exam	Loss of radial 3.5 fingers.	Loss of radial 3.5 fingers + thenar eminence	Loss of ulnar 1.5 fingers	Loss of ulnar 1.5 fingers + hypothenar eminence	Loss over 1st dorsal webspace	Loss over 1st dorsal webspace
Motor exam	Weak APB	Weak APB Weak FDS + FDP of radial 2 digits.	Froment sign Weak abduction	Froment sign Weak abduction + FDP of ulnar 2 digits	Finger drop	Wrist drop Weak elbow flexion & triceps jerk
Other features	Tinel's Phalen's	Scar over cubital fossa	?Wrist scar	Elbow scar Cubital valgus		? Humeral scar
Causes & example	Carpal tunnel	Cubital fossa	Wrist trauma	Tardy ulnar nerve palsy after lat epicondyle #		Crutch palsy Humeral #

Mimics to beware of:

- The T1 lesion – e.g. pancoast tumor, cervical spine
- Other LMN pathology: e.g. peripheral nerve disease (DM neuropathy, charcot-marie-tooth) – see medical short case neurology

MEDIAN NERVE: GENERIC SCRIPT**Brief history:**

- Symptoms - numbness, weakness, tingling. One or both hands. Any neck pain (TRO cervical myelopathy)
- Onset and course
- Occupation, handedness and function
- Past medical history
- What treatment have you tried so far

Examination: My patient is a middle-aged Chinese lady. On inspection I note that she has wasting of the thenar eminence of both hands. There are otherwise no scars or swellings or skin changes of the hand.

Mdm ____, This is a satay stick / toothpick, it should feel sharp but not painful. I want to test how well your hands can feel. This (test forehead) is 100%. When I touch your hand can you tell me how many % you can feel? Please close your eyes... On sensory examination I note that there is decreased sensation over the radial 3 and a half digits on the palmar side bilaterally with a split ring finger. Sensation over the thenar eminence is preserved bilaterally.

Examining the motor function, I note that there is weakness of the right abductor pollicis brevis of MRC scale 3 over 5, and of the left abductor pollicis brevis which is 4 over 5. Testing the flexor digitorum profundus (isolate distal phalanx), I note that there is full power of distal phalanx flexion of the index and middle fingers bilaterally. Testing the flexor digitorum superficialis (isolate individual finger) I note full power of all fingers bilaterally. The patient is able to make an "OK" (Oschner clasping test) and has no benediction sign.

Doing a quick screen for the other nerves I note that the finger abduction is intact and there is no finger drop.

Maam, I will now press on your wrist, can you tell me if what you feel when I do this? (asking this way is better than giving a leading question - i.e. do you feel shooting sensation). Tinel's sign is positive. Phalen's test is also positive (must hold for 30sec minimum).

I will now like to assess the patient's function. Maam can you please show me how you write / unbutton your shirt / open this bottle.

Summary: In summary my patient is a middle-aged Chinese lady who works as a typist, complaining of numbness and tingling of hands that affects her function. Examination findings are that of a median nerve palsy at the level of the wrist and my diagnosis is carpal tunnel syndrome. In terms of etiology I do not note any evidence of RA, thyroid disease, or acromegaly.

MEDIAN NERVE: QUESTIONS

Tell me the course and supply of the median nerve

The median nerve is formed by the lateral and medial cords of the brachial plexus containing innervation from C5 to T1 nerve roots. It enters the arm in close relation to the brachial artery and does not make any branches above the elbow. In the cubital fossa it is located lateral to the brachialis tendon; there it gives off the anterior interosseous nerve which supplies the deep flexor muscles of the anterior forearm. Above the wrist it gives off the palmar cutaneous branch which supplies sensation over the thenar eminence. It enters the carpal tunnel and in the hand supplies the flexor pollicis brevis, abductor pollicis brevis, opponens pollicis, and lateral 2 lumbricals (LOAF), as well as the sensory supply of the radial 3 and a half digits.

How will the findings of a high median nerve lesion differ?

Involvement of the median nerve above the palmar cutaneous branch will also cause numbness over the thenar eminence. A lesion at or above the cubital fossa will also result in weakness of flexor digitorum superficialis, weakness of flexor digitorum profundus of the lateral 2 digits, a benediction sign and the patient will not be able to make an "OK" sign.

What is anterior interosseous nerve syndrome?

The anterior interosseous nerve is a motor branch of the median nerve that leaves the median nerve about 5cm distal to the elbow. It supplies the deep anterior forearm muscles which are the FDP of radial 2 fingers, FPL and pronator quadratus (FDP of ulnar 2 digits are supplied by the ulnar nerve). If there is a AIN lesion, patient cannot make the OK sign and there is no sensory loss. The most common cause of AIN syndrome is entrapment by the pronator teres.

A high median nerve lesion will, in addition to AIN syndrome, have:

- Motor: weakness of the FDS, flexor carpi radialis, LOAF muscles (lateral 2 lumbricals, opponens pollicis, abductor pollicis, flexor pollicis brevis)
- Sensory: sensory loss over the radial 3.5 fingers and thenar eminence

What are the causes of carpal tunnel syndrome?

Carpal tunnel syndrome is most commonly idiopathic in which case it is associated with repetitive strain e.g. in a typist. It may also be due to secondary pathology such as pregnancy, hypothyroidism, rheumatoid arthritis, and renal failure. Local anatomical lesions may compress on the median nerve e.g. previous wrist fractures, ganglion.

What investigations will you like to do for this lady?

I will like to do a nerve conduction study

How do you treat her?

Management can be divided into conservative and surgical options. My patient has attempted conservative measures such as night splinting and steroid injection without success, and as a typist it is difficult for her to modify her lifestyle to minimise stress on the carpal tunnel. Therefore I will like to offer her surgical options such as carpal tunnel decompression.

ULNAR NERVE: GENERIC SCRIPT**Brief history:**

- Symptoms - numbness, weakness, tingling. One or both hands.
- Onset and course
- Occupation, handedness and function
- Past medical history
- What have you tried so far

Examination: Sir, my patient is a young Malay gentleman who is alert and comfortable at rest. On inspection of his hands I note that there is clawing of the ulnar 2 digits of the right hand, associated with guttering of the fingers and wasting of the hypothenar eminence. There is also a right cubitus valgus deformity with a surgical scar at the right elbow over the medial epicondyle.

Mister ____, this is a satay stick / toothpick, it should feel sharp but not painful. I want to test how well your hands can feel. This (test forehead) is 100%. When I touch your hand can you tell me how many % you can feel? Please close your eyes... On sensory examination I note that there is decreased sensation over the ulnar one and a half digit on the right with a split ring finger. There is also decreased sensation over the hypothenar eminence.

Mister ____, this is a piece of paper. Can you please use your thumb to press down on the piece of paper and don't let me pull it out. Testing motor function, there is positive Froment's sign on the right hand. There is also weakness of finger abduction and of the flexor digitorum profundus of the ring and little finger.

Doing a quick screen for the other nerves I note that there is no finger drop and power of abductor pollicis brevis is intact.

Mister, can you please bend (flex) your elbow fully, hold it there for a minute, and tell me what you feel. There is a tingling sensation over the ring and little finger.

I will now like to assess the patient's function. Mister can you squeeze my fist as hard as you can - pretend it is now a doorknob, can you turn it. [*Ulnar nerve mainly affects ring and little finger - main impairment is grip strength. Cf median nerve which mainly affects thumb and index impairing pincer grip*]

Summary: In summary my patient is a young Malay gentleman with right cubitus valgus deformity and right ulnar nerve palsy at the level of the elbow. He has a surgical scar suggestive of previous anterior transposition of the ulnar nerve. His function is not limited.

ULNAR NERVE: QUESTIONS**Tell me about the course and supply of the ulnar nerve.**

The ulnar nerve begins as the medial cord of the brachial plexus carrying C8 and T1 nerve fibres. It does not have branches in the arm and enters the forearm via the cubital tunnel posterior to the medial epicondyle. It then supplies the flexor carpi ulnaris and the ulnar half of the flexor digitorum profundus in the forearm. It gives off a sensory branch which supplies the sensation over the hypothenar eminence, before entering the wrist and the Guyon's canal. In the hand it supplies sensation over the ulnar one and a half digits, as well as all intrinsic muscles of the hand except the flexor pollicis brevis, abductor pollicis brevis, opponens pollicis, and the radial two lumbricals.

Can you tell me the other locations of an ulnar nerve lesion and how your examination will differ?

Lesions of the ulnar nerve can be divided into high and low lesions. High lesions include a cubital tunnel syndrome, elbow trauma such as dislocations or paediatric lateral condylar fracture which can lead to cubitus varus and tardy ulnar nerve palsy. Examination findings are as presented in this patient. Low lesions include compression in the Guyon's canal or wrist trauma, which will differ from high lesions in that there will be more pronounced claw hand, intact sensation over the hypothenar eminence, and no weakness of flexor digitorum profundus of little and ring finger.

What is the ulnar paradox?

The ulnar paradox states that a low ulnar nerve lesion results in a more pronounced claw hand. This is because a high lesion affects both the lumbricals and ulnar half of the flexor digitorum profundus while a low lesion affects the lumbricals only. Hence in a low lesion, the flexor digitorum profundus acts unopposed, resulting in a more obvious claw hand (hyperextension of MCPJ and flexion of IPJ)

Hand: Wrist Laceration

SAMPLE OSCE

Instruction to candidate:

You are the A&E MO.

Ms Emo has just cut her wrist and now complains of hand weakness and numbness. Please take a short history and examine her hands.

Instruction to SP:

You are Ms Emo, a 24 year old lady recently discharged from IMH for depression b/g borderline personality disorder. You have just been dumped your boyfriend and just cut your wrist with a clean pen knife to “relieve the emotional turmoil inside” as well as get his attention again. You do not have any real intention to take your life.



Examination findings

- Your median nerve and flexor digitorum superficialis have been cut.
- Both ulnar and radial pulses are intact. Capillary refill time is <2s. Hand is still warm.
- You have sensory loss over the left radial 3.5 fingers, sparing of sensation over the thenar eminence and the radial forearm. Sensation elsewhere is normal.
- You have weakness of the LOAF muscles - 1st and 2nd lumbricals, opponens pollicis, abductor pollicis and flexor pollicis brevis (but this is compensated by the longus) → cannot oppose thumb and little finger, cannot abduct thumb against resistance, cannot do twist sign.
- As your flexor digitorum superficialis has been cut, you cannot flex the 2nd to 4th PIPJ. 2nd to 4th DIPJ is still strong, and compensates for fist closure to some extent. Froment's negative.
- You have reduced grip strength, and cannot perform any functional tests the candidate may ask you to do.
- You are right hand dominant.

Marking rubrics:

		Performed and competent	Performed but NOT fully competent	Not performed or incompetent
History				
1	Greets patient and introduces self	1	0.5	0
2	Elicit history of injury, PMHx, drug allergy	2	1	0
3	Elicit hand dominance	1	0.5	0
4	Ask about tetanus vaccination and what was used to cut	1	0.5	0
5	Bonus: suicide risk assessment	2	1	0
Examination				
6	Inspection: skin wound, visible structures, pallor, no wasting	2	1	0
7	Assess vessels: pulses, temperature, cap refill	2	1	0
8	Assess sensation (findings as above)	2	1	0
9	Assess power (findings as above)	2	1	0
10	Assess hand function	1	0.5	0
Discussion				
11	Diagnosis and assessment	2	1	0
12	Differentiate high vs low lesion	1	0.5	0
13	Investigations and Management	2	1	0

Examiner's comments:

- Many students didn't check vascular status early → should check early; must examine according to 5 tissues of the hand (skin / vessels / nerves / tendons / bone and joint)
- This is not a straightforward median nerve; the pattern of neurological deficit does not fit → should suspect something else
- Same case can come out as a chronic lesion.

Knee: ACL Tear

GENERIC SCRIPT

Stem: This patient complains of left knee instability; please take a brief history and examine

Brief history:

- What symptoms do you experience?
- How long has it been?
- How did it start?
- Trauma
- How does this affect you now?

Examination: My patient is a young Chinese gentleman who is alert and comfortable. Inspecting from the front, side, and back with the patient standing; I do not note any scars, skin changes, swellings, or deformities of his knee. His gait appears grossly normal and is not antalgic.

[Lie pt down] The left knee is not warm. Measuring thigh circumference 10cm superior to the lateral femoral condyle, both thighs are of the same circumference. Palpation is unremarkable with no point tenderness in the extensor mechanism, and medial and lateral joint line. Palpation of the collateral ligaments is also unremarkable. Patellar grind is negative and bulge test for effusion is negative.

I will now like to demonstrate movements. Passive movement is full ranging from 0 degrees to 160 degrees.

I will now perform the drawer tests. [Position patient]. There is no posterior sag. Anterior drawer test is positive with a spongy end point. I will now perform the Lachmann test. It is positive with a spongy end point.

I will examine the other ligaments. I am now performing the valgus stress test in full extension, and again in 20 degree of flexion. The test is negative. I am now performing the varus stress test in full extension and again in 20 degrees of flexion. This is also negative.

I will now like to perform the McMurray test for the medial meniscus. I am flexing and externally rotating the knee, applying a valgus stress and now gradually extending the knee. There is no clunk and the test is negative.

I will like to complete my examination by examining the hip, lumbar spine, and doing a neurovascular examination of the lower limb.

Summary: Sir, my patient is a young chinese gentleman who is a serious but non-competitive soccer player. He complains of a 3 month history of left knee instability after a sports injury in which he heard a pop sound and was unable to continue playing. He had left knee swelling immediately after the injury but this has since resolved. Functionally, the knee instability interferes with his ability to play soccer. Examination revealed a positive anterior drawer and Lachmann test suggestive of torn anterior cruciate ligament. I do not note any other ligamentous injury and there is no medial joint line tenderness to suggest meniscal injury.

QUESTIONS

What are the injuries associated with an ACL tear?

- MCL injury
- Meniscal tear

How would you investigate?

I would start with weightbearing AP and lateral X-rays of the knee. I expect this to be normal however there may be a second fracture. A more definitive imaging modality will be MRI.

How would you manage?

- This is a young patient with high functional demand. Therefore, while I would give a try of conservative management, I am keen to offer surgical management after discussion with the patient.
- Conservative management includes lifestyle modification, wearing a knee brace, physiotherapy.
- Surgical management is that of ACL reconstruction using a patellar tendon or hamstring autograft, and thereafter immediate rehabilitation.

Shoulder: Rotator Cuff Tendinitis

GENERIC SCRIPT

Stem: this patient complains of shoulder pain, please take a brief history and examine

Brief history:

- Demographics: age, handedness, PMHx, drug allergy
- Symptoms and what makes it worse: distinguish between pain and stiffness or instability; if pain or stiffness find out whether in one or all planes or movement. (*See approach to shoulder pain and instability in Approaches to Symptoms of disease*)
- Onset; any trauma
- Function.

Examination: My patient is an middle aged Chinese gentleman who appears alert and comfortable at rest. On inspection I do not note any scars, skin changes, swellings, or deformities.

I am now palpating along the SC joint, the clavicle, the AC joint, the head of the humerus; now posteriorly, the spine of the scapula... I do not note any deformity or tenderness.

Examining movements, I note that range of flexion, extension, abduction, adduction, and external rotation are full. There is however a painful arc from 15 degrees to 90 degrees of abduction. Functional internal rotation of the right shoulder is limited.

I am now testing the supraspinatus muscle in 90 degrees of flexion and 15 degrees of abduction. There is mild supraspinatus weakness of power 4 upon 5, as compared to the contralateral side. I am now testing the infraspinatus muscle by resisted external rotation. Power is full. I am now doing belly press... subscapularis power is full.

[Special tests]

- I will now like to do the Neer's test by internally rotating the arm and slowly flexing to 90 degrees. There is pain which is a positive neer's test suggesting impingement.
- I will now like to do the Hawkin's test by flexing arm and elbow to 90 degrees and then internally rotating. There is pain which is a positive Hawkin's test suggesting impingement.
- I will now do a Speed's test to look for concomitant brachial tendonitis; on resisted shoulder flexion with elbow extended, there is no pain / I will now do Yergason's test; on resisted supination there is no pain.

I will like to complete my examination by examining the cervical spine and doing a neurovascular examination of the upper limb.

Summary: In summary, my patient is a middle aged Chinese gentleman who complains of mechanical right shoulder pain, worse on reaching overhead; not significantly affecting function. Examination reveals signs of rotator cuff tendinitis as evidenced by positive Neer's / Hawkins's test, painful arc. There is mild supraspinatus weakness but otherwise power and range of movement is full.

QUESTIONS:

How would you manage this patient?

I believe that conservative management is most appropriate for my patient in view that this is his first episode of tendinitis which is neither persistent nor affecting function significantly, and there is no significant loss of power suggesting a cuff tear. Conservative management would involve activity modification, analgesia, and physiotherapy +/- subacromial H & L injection. If tendinitis is recurrent or bothersome the option of arthroscopic debridement and acromioplasty can be offered.

Medicine Short Cases

thinking on your feet



Overall Strategy

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Strategy

What are medical short cases like? These are 10-minute stations in which you have 6-8 minutes to examine (finish early if you can), and the remaining time to summarize findings, offer diagnosis and differentials, and (if you are fast) discuss investigations and management.

Examiners want you to:

- Demonstrate proficient examination technique (running commentary not advisable)
- Accurately pick up key signs (*but please do not invent*)
- Be able to synthesize your findings in terms of diagnosis – severity – etiology – complications – management as appropriate

Your strategy: Medical short cases are more difficult than surgery ones because: (1) range of cases is wide and not predictable, (2) signs take work to elicit and can be subtle, (3) there can be multiple findings for which the interpretation is not obvious. Our advice is:

- *There is no alternative to seeing many cases.* You need experience to confidently pick up signs, especially in subtle or difficult cases. As you hit the wards, don't just focus on picking up signs, but do complete timed examinations (with your friends as critics) to also hone your thought process.
- *Examine with brain turned on.* 'Pick up signs now and synthesize later' is a doomed strategy because you have no time to consolidate, and you might find yourself wanting to go back to confirm certain signs or look for others. Rather, form and continually refine an impression as you examine – when you pick up a sign that 'doesn't fit', question either the sign or your impression. Also look for additional signs along the diagnosis – severity – etiology – complications – management chain. You can only look for what you think about, and you can only find what you look for.

All this sounds daunting, and indeed the cognitive load is immense - so a mental framework is helpful. This chapter aims to provide this reasoning process that will allow you to think on your feet. It is *not* a script (unlike surgical short cases), but a roadmap to guide you from starting bell to diagnosis, and some further notes on your tasks from there. Once you have the diagnosis, jump ship to Jansen Koh's excellent scripts and Q&A for the common medicine short cases.

We have integrated the pediatric approaches into the adult ones where possible. It is probably better to learn by compare and contrast, rather than learning an entirely separate set of approaches.

FAQ: HOW SHOULD I PRESENT?

This is a common question. The baseline is, you need to report key signs (not regurgitate everything from start to end) and offer a reasonable impression. Our suggested templates could look like -

If you are sure of the diagnosis:

- Sir my patient is a [demographic] with [diagnosis], I say this because [supporting features]
- This is [severity] as evidenced by [supporting features]
- In terms of etiology...
- I note complications of ...
- In summary...
- I will like to complete my examination by...

If you are not sure:

- Take a deep breath: this does not always mean the case is going south, sometimes cases are complex or atypical. Examiners understand and will guide you.
- Do not try to waste time so as to be 'saved by the bell': you will instead be 'killed by the bell'. It is to your advantage to place your bet and present your findings, even if you are unsure. If you are incorrect, you will be prompted to re-examine and thereby get a second chance.
- You can present sequentially but group your findings in a sensible manner.
- Still attempt to offer one or two possible differentials, elaborate on what fits and what doesn't.

Neurological System

ADULTS AND PAEDIATRICS

[Main text discusses adults; unique paediatric considerations are annotated at the side]

The neurological examination strikes fear in many, but once learnt, it is quite logical and intuitive to work through. Your main task is to localize the lesion (where is the lesion), and then suggest possible etiologies (what is the lesion). A polished examination takes some finesse, especially when all findings are visible not only to you, but also to the examiner.

It is especially crucial to examine with brain on, as there is never time to do a 'complete neurological examination', and what you do is directed by what you think is the likely diagnosis, differentials and complications.

1. Understand the stem & identify your task

The neurological examination comes in a variety of stems which you must first grasp. Have an approach to each common stem -

(a) General stems

- Please examine the lower / upper limbs
 - Examine the stated limb in the rehearsed fashion - first task is to decide if UMN or LMN (Beware of non-neurological examination e.g. joint deformity)
 - If UMN, examine cerebellar first and proceed upwards i.e. examine key features in the upper limbs (if lower limbs) and/or cranial nerves.
 - If LMN, examine sensory carefully.
- Please examine the cranial nerves
 - Begin with the quick screen - greet the patient with a big smile (can the patient look at you? Is there ptosis or gaze deviation? Can he smile? Can he talk?)
 - If anything obvious is amiss, zero in on the abnormality and related cranial nerves (e.g. if obvious facial droop, examine CN 5-8 first) rather than screening from CN2 to 12.
 - Identify if multiple cranial nerves are involved or there is a single abnormality.
 - You may need to examine cerebellar system and long tracts.

PAEDIATRIC POINTERS

Paeds neuro is a common short. Begin by looking for

- Dysmorphisms
- If child is well thrived: but hedge 'I would like to plot height and weight against gender specific percentile progressive charts'.

Localization is the same in paediatrics but etiologies are more limited. Begin with the main approach; this column pops up where approaches or etiologies diverge.

- This patient has difficulty walking / frequent falls.
 - This is an invitation to start with gait (see gait analysis in *Approaches to Symptoms* notes). You may need to stress the gait by running, tandem gait, tiptoe, running, squat and stand.
 - Difficulty walking may be due to weakness, incoordination, sensory loss, or musculoskeletal causes - don't assume it's pyramidal weakness.
 - Look at the arm for decreased arm swing (hemiplegia, parkinsonian).
 - After gait, continue with the lower limbs focusing on the likely system identified, or go straight to the abnormality you suspect.

(b) More specific stems: these are meant to suggest a specific diagnosis so have that in mind and go with the flow. For example,

- This patient has difficulty seeing, please examine → look for ptosis, thyroid eye disease, ophthalmoplegia, pupillary disorders (including relative afferent pupillary defect), and visual field defects (often skipped but important here). Remember to always check for visual acuity first!
- This patient has red eye, please examine → exposure keratitis from seventh nerve palsy
- This patient has difficulty writing, please examine → hand nerve lesion (medial / radial / ulnar, see ortho short case), cervical spine disease (radiculopathy, myelopathy, syringomyelia, etc), parkinson's disease

Beware the non-neurologic exam, e.g. 'examine the eyes' which turns out as an exophthalmos!

2. Examine carefully

Being clear on your task, proceed to examine the patient carefully. Technique is important if you are to be able to elicit the signs accurately, and experience allows you to be confident of your findings. As always, think as you examine - refining your hypotheses as you gather new information, and being alert to what 'doesn't fit'.

PAEDIATRIC POINTERS

Young kids may not be able to cooperate with examination; in that situation you will have to rely on tone, reflexes, and other hard signs. How the child interacts with you provides information: note his/her apparent IQ

It always helps to go in having something in your mind to look for. Certain conditions should be spot diagnoses -

- Movement disorders e.g. Parkinsonism, chorea (see section 3)
- Chronic UMN lesion with spastic contractures and stigmata of immobility
- Chronic LMN lesion with fasciculations
- Certain cranial nerve lesions: Ptosis, facial droop

A note on communication: Patient cooperation is essential to the neurological examination and therefore you must excellent communication that also demonstrates rapport and empathy - not just ‘uncle, 大力!’ but telling the patient clearly what you are trying to test, what to expect, and how he can help you. For example,

- Uncle, I want to see how relaxed you can be. I am going to move your arms/legs, please relax completely and do not resist me.
- Uncle, I want to see how strong you are. Can you please hold your hands up like this (demonstrate) as strong as you can, and do not let me push you down.
- Uncle, this is the blunt end of a satay stick (demonstrate on yourself). I will now scratch the bottom of your foot - it may be slightly ticklish.
- Uncle, I want to see how well you feel. This is a satay stick, it should be sharp but not painful (demonstrate on yourself). Let me test on your forehead, can you feel? Ok, this is 100%. I will now test your legs, please point to where I touch and tell me how many %.

3. Identify the clinical picture and proceed

From the initial examination, identify one of the following clinical pictures:

Limbs +/- CN or cerebellar	Cranial Nerves alone	Movement Disorders
a) UMN Hemiplegia b) UMN Diplegia / Quadriplegia c) UMN, bizarre distribution d) LMN + normal sensation e) LMN + abnormal sensation	f) Multiple CN +/- cerebellar g) Isolated ptosis h) Ophthalmoplegia i) Isolated facial droop	j) Hypokinetic (Parkinsonian) k) Hyperkinetic (Chorea) l) Isolated cerebellar disorder

Work through each syndrome, thinking ‘where is the lesion’ and ‘what is the lesion’. Always fall back on the neuroaxis - from brain (cortical, subcortical), to brainstem, spinal cord, anterior horn cell, root, plexus, peripheral nerve, neuromuscular junction, and muscle.

(a) UMN Hemiplegia

This is a CNS lesion above the spinal cord. First examine cerebellar system and cranial nerves

-

- Ipsilateral¹ cerebellar dysfunction → Ataxic hemiparesis, a lacunar (subcortical) syndrome.
- Contralateral¹ cranial nerve palsy → Crossed hemiparesis, lesion is in the brainstem
 - Contralateral CN VI or contralateral LMN CN VII → Contralateral pons
 - Contralateral CN III +/- ipsilateral UMN CN VII → Contralateral midbrain.
- Ipsilateral cranial nerve palsy (e.g. UMN CN7, CN6) → Lesion is above the brainstem and may be in the subcortex or cortex, proceed as below

Then proceed to look for cortical signs (especially if no cranial nerve / cerebellar lesions, or ipsilateral cranial nerve lesion). Cortical signs include gaze deviation, aphasia, hemineglect, and cortical blindness. One useful test is to hold your stethoscope in front of the patient and ask him to use his arm to divide it in half (line bisection test, which would indicate either hemi neglect or hemianopia).

- Cortical signs present → lesion is in contralateral cerebral cortex.
- No cortical signs present → Clinically this is a subcortical lesion.

What is the etiology? Think vascular, infective, neoplastic, inflammatory - look for clues:

- Atrial fibrillation → embolic large-vessel stroke
- Obvious vasculopath e.g. CABG scar, leg amputations, → both small vessel lacunar infarct, or large vessel disease are possible
- Request for blood pressure → Hypertensive bleed.
- Craniectomy scars → stroke with haemorrhagic conversion, traumatic head injury, tumor
- Young lady with malar rash → SLE with antiphospholipid syndrome, complicated by stroke.
- Offer to take history for time course of lesion - e.g. acute: vascular, subacute: inflammatory, chronic: neoplastic, any category: infective

PAEDIATRIC POINTERS

Proceed as for adult and also

- Feel neck for ventriculo-peritoneal shunt → hydrocephalus requiring decompression
- Look for port-wine stain on contralateral side (Sturge-Weber)

Ddx are as in adults, plus

- Spastic hemiplegic cerebral palsy (e.g. from intrauterine stroke, sequelae of meningitis)
- Congenital: Sturge-Weber, cortical dysplasia

¹ N.B. by convention, as well as in this discussion, contralateral means opposite to the side of hemiparesis, and ipsilateral means on the same side of hemiparesis.

(b) UMN diplegia or quadriplegia

In adults, diplegia or quadriplegia is usually a spinal cord lesion (a lesion must be quite large to affect both halves of the brain or brainstem, but a much smaller lesion may affect the entire spinal cord).

Where is the lesion? The main task is to find a spinal cord *level*. In diplegia, move upwards from lower limbs, to superficial abdominal sensation and reflexes, then to upper limbs. In quadriplegia, examining

- The level of lesion = the myotome/dermatome at which UMN findings become LMN and sensory becomes normal
- If patient requires ventilatory support, think high cervical cord lesion (above C5) affecting the phrenic nerve.
- If no level is found, reconsider a parasagittal cerebral lesion (e.g. meningioma) causing diplegia, or a diffuse cerebral process.

What is the lesion? Proceed as follows depending on whether sensation is normal or abnormal-

Sensation abnormal:

- Look for dissociated sensory loss:
 - Syringomyelia: pain & temperature loss in a 'shawl' distribution', fine touch & proprioception spared. UMN weakness in UL > LL²
 - Anterior cord syndrome: UMN paraparesis + isolated loss of pain and temperature, dorsal column spared. Usually due to infarct.
 - Subacute combined degeneration: UMN paraparesis + isolated proprioception and vibration loss with UMN paraparesis.
 - Posterior cord syndrome: isolated proprioception and vibration loss, pain and temperature spared, no UMN paraparesis (rare).

PAEDIATRIC POINTERS

Spastic diplegia ranges from mild to classic scissoring gait.

Unlike adults, diffuse cerebral processes feature more often - cerebral palsy - although spine disease is still possible.

If a level is found, think hard of spine disease.

If no level is found, cerebral palsy is likely. Identify the distribution:

- Spastic diplegic CP: IQ usually normal.
- Spastic quadriplegic CP: IQ usually affected.

Beware the Friedreich Ataxia with upgoing plantars and pes cavus but absent ankle jerks - this is not just a spastic diplegia (see discussion in isolated cerebellar)

It is probably quite difficult to examine sensation in a child, unless he is quite old. In any case these etiologies are not common in paediatrics

² Pathophysiology: the syrinx, expanding from the centre of the cord outwards, affects decussating spinothalamic fibres within C5-T7 segments before it affects the dorsal columns, and also affects the corticospinal fibres of the ULs before the LLs.

- Turn patient and examine the back/neck
 - Look for operation scars → spinal trauma, repaired congenital spinal defects
- Examine eyes +/- do fundoscopy
 - Look for multiple sclerosis / neuromyelitis optica: internuclear ophthalmoplegia, optic neuritis.
- Offer other lesions: tumor, infection, TB

Sensation normal:

- Older patient: look for cervical myelopathy --
 - Supportive findings are ataxic gait, slow grip and release, +ve Hoffman's sign. Loss of two point discrimination but no overt numbness.
 - If RA hands, think atlantoaxial subluxation
 - Central cord syndrome may be superimposed. This is usually post-traumatic, weakness UL > LL, proximal > distal
- Consider hereditary spastic diplegia: isolated spastic gait, difficulty walking, and UMN signs. None or very mild other neuro findings. Ask for family history.
- If nothing else consider
 - Reconsider parasagittal brain lesion.
 - Reconsider other lesions (tumor, infection, TB), less likely to involve motor only

Look for complications: Bladder and bowel function is commonly affected in spine disease - look for urinary catheters and offer digital rectal examination.

PAEDIATRIC POINTERS

Please do look carefully for scars on the back;

Less likely in paediatrics

Cervical myelopathy is not relevant in the child

Cerebral palsy is the most common cause and sensation is normal. Also look for

- Ventriculoperitoneal shunt
- Signs of prematurity e.g. plagiocephaly.
- Hearing loss: CP due to meningitis sequelae

(c) Scattered UMN signs in bizarre distribution

This category of patients do not have a classic hemiparesis or diplegia; specific myotomes may be affected almost at random.

PAEDIATRIC POINTERS

You are rather unlikely to see this pattern in a child.

Key differentials:

- Multiple strokes - a lacunar lesion may affect a very small area, and multiple lacunar strokes may cause isolated patches of UMN.
- Multiple sclerosis - dissemination in space is a diagnostic criterion after all. If suspecting, examine eyes for internuclear ophthalmoplegia, optic neuritis.
- Motor neuron disease - the characteristic paradox of wasting *and* UMN weakness / signs *in the same myotome*. Pause and inspect carefully for fasciculations - best place to look is the tongue at rest *in the mouth*. Sensory will be absolutely and utterly intact. Be aware that MND is a heterogenous group, some have the classic bizarre mix of UMN and LMN, others (less common) are purely LMN or purely UMN.

Upgoing plantars and absent ankle jerks: this paradox arises due to the combination of a UMN and LMN lesion. There are several well-defined causes:

- Two common pathologies, often with common risk factors
 - Stroke with DM neuropathy
- One pathology affecting both UMN neurons and anterior horn cells
 - Motor neuron disease
 - Multiple sclerosis (possible but less common)
- Spine lesion causing UMN signs + affecting reflex arc.
 - Subacute combined degeneration
 - Conus medullaris lesion
 - Friedreich ataxia (see isolated cerebellar)
 - Tabes dorsalis (syphilis)

(d) LMN weakness with normal sensation

In LMN weakness, first identify if sensation is normal or impaired. Causes of symmetrical LMN weakness with normal sensation are

- Neuromuscular junction disease
- Muscle disease
- Anterior horn cell disease
- Isolated motor neuropathy (less likely)

Use the distribution of weakness (proximal vs distal), and reflexes (preserved vs lost) to distinguish lesions -

PAEDIATRIC POINTERS

This pattern is common in paediatrics; the main ddx are

- NMJ: myasthenia gravis
- Muscle: muscular dystrophy (Duchenne, Becker, etc)
- AHC: spinal muscular atrophy

Localize as in the adult.

Lesion	Case	Weakness	Reflexes	Unique feature
Neuromuscular Junction	Myasthenia Gravis (MG) Lambert-Eaton myasthenic syndrome (LEMS)	Proximal	Relatively preserved	Fatigable or improves with exertion.
Myopathy	Muscular dystrophy (e.g. Duchenne, Becker) Autoimmune (dermatomyositis, polymyositis) Endocrine (Cushing, thyroid) Metabolic (alcoholic) Drug induced (statins)	Proximal	Distal is preserved	Pseudohypertrophy Rash, muscle pain Endocrine features Parotid, Dupuytren
	Myotonic dystrophy	Distal	Decreased	Slow to relax
Anterior horn cell	Spinal muscular atrophy (SMA)	Proximal	Decreased	Fasciculations
	Polio	Isolated	Decreased	Very isolated
	Motor neuron disease (MND)	Mixed	UMN	See (c)
Isolated motor neuropathy	Acute motor axonal neuropathy (a type of Gullian-Barre)	Distal	Decreased	

Look closely for clues that may be visible on inspection:

- In the face
 - Ptosis → MG or myotonic dystrophy
 - ‘Hangdog’ facies: Frontal balding, temporalis and masseter wasting, bilateral ptosis, bilateral facial droop, expressionless → myotonic dystrophy
 - Proptosis → Thyroid myopathy
 - Tongue fasciculations (look at tongue inside mouth) → SMA vs MND
- In the limbs
 - Pseudohypertrophy, biopsy scar → think muscular dystrophy
 - Isolated wasting → think polio
 - Fasciculations → again, SMA vs MND.
- General appearance
 - Rash → Dermatomyositis
 - Cushingoid appearance → Cushing’s myopathy

Then perform specific tests to confirm what you suspect:

- MG: do prolonged upward gaze and repeated shoulder abduction for fatigability. If strength improves instead of fatigues, be wary of LEMS.
- Myotonic dystrophy: test for percussion myotonia, slowness to open clenched fist or eyes after firm closure
- Muscular dystrophy: attempt Gower’s sign.

(e) LMN weakness with abnormal sensation

This localizes to peripheral nerve, root, and plexus. The distribution of weakness is crucial:

- Symmetrical bilateral LMN weakness, usually distal with sensory loss in a 'glove and stocking pattern' → most likely peripheral neuropathy
- Symmetrical bilateral LMN weakness, with patchy sensory loss (sometimes symmetrical) → think of a cauda equina lesion (spina bifida, trauma)
- Asymmetrical or in a specific myotomal / dermatomal distribution → lesions affecting specific nerves, roots, and plexuses.

Patchy peripheral neuropathy: consider cauda equina lesions. Turn the patient and examine the spine (including the gluteal region), look for

- Spina bifida: dimple or tuft of hair
- Scar: old spina bifida s/p surgery, trauma, or large prolapsed disk with cauda equina.

Symmetrical peripheral neuropathy: differentials may be hard to ddx on physical examination -

- Diabetic neuropathy: look for neuropathic ulcers, DM dermopathy
- Hereditary sensorimotor neuropathy (Charcot Marie Tooth disease): ask for family history.
- Guillain-barre syndrome
- Toxic: alcoholic, drugs, paraneoplastic

PAEDIATRIC POINTERS

It is particularly important to rule out spina bifida. Signs can be subtle so look closely.

In a well outpatient kid (or young adult), put your money on Charcot Marie Tooth. Ask to examine the mother too.

For specific nerve / root / plexus lesion: this is a true test of how much anatomy you remember. For example:

- LMN unilateral foot drop (rule out bilateral or UMN): sciatic nerve, common peroneal nerve or L5 lesion. Check ankle jerk and plantarflexion - affected in sciatic nerve lesion but spared otherwise. Hip abduction distinguishes the two - it is affected in L5 lesion, but not in a peroneal nerve lesion.
- Hand weakness: elicit the features of ulnar / median / radial nerve lesions, attempting to distinguish low vs high lesions (see ortho short case). However beware of root lesions, e.g. wrist drop can be due to both radial nerve and C7 lesion - but the sensory distribution affected is different in both.
- If you cannot explain the lesion with a single nerve / root / plexus, consider mononeuritis multiplex. Causes of mononeuritis multiplex include -
 - Endocrine: Diabetes, acromegaly
 - Rheumatological: RA, SLE, vasculitis (Wegener's, Churg-Strauss)
 - Infective: retroviral, leprosy
 - Infiltrative: amyloid, sarcoidosis

(f) Multiple cranial nerve palsy +/- cerebellar signs.

Various lesions may affect defined 'clubs' of cranial nerves - it makes a lot of sense once you recall the anatomy. Unfortunately life is not always straightforward and at times you may get an incomplete 'club' (simply because some nerves are affected before others). In any of the brainstem lesions you may also have cerebellar involvement. Here goes:

- CN III - IV: Midbrain lesion
- CN V - VIII: Pontine lesion
- CN IX - XII: Medullary lesion
- CN VII, VIII +/- V: Cerebellopontine angle lesion (usually acoustic neuroma)
- CN III, IV, VI, V₁: Superior orbital fissure lesion
- CN III, IV, VI, V₁, V₂: Cavernous sinus syndrome
- CN V, miosis, nystagmus: think of lateral medullary syndrome → examine for crossed sensory loss (ipsilateral face and contralateral limb), ipsilateral Horner's (miosis, ptosis) ipsilateral cerebellar signs + lower CN palsies (hoarseness, dysphagia, palatal deviation away from side of lesion).

The universal diagnoses: these lesions may also affect multiple cranial nerves in any pattern. Always think of them, and examine long tracts for additional information.

- Base of skull disease: e.g. tuberculosis, meningitis, nasopharyngeal carcinoma
- Myasthenia gravis → look for fatigability
- Peripheral nerve: Guillain Barre syndrome, mononeuritis multiplex
- Multiple sclerosis

Subsequent clinical scenarios assume that multiple cranial nerve pathology has been excluded.

(g) Isolated ptosis

First decide if unilateral or bilateral, and always rule out the universal diagnoses above.

Unilateral ptosis:

- CN III palsy: a down and out eye, patient will not be able to overcome ptosis. Look for the etiology, paying attention to the size of the pupil
 - Is it a midbrain lesion? -- Examine long tracts for crossed hemiparesis, and for other cerebellar signs. Etiologies include vascular, inflammatory (MS), mitotic, and infective
 - Is it a surgical third? --- Pupil is blown. This is potentially life threatening - causes include posterior communicating artery aneurysm, tumor, raised intracranial pressure
 - If medical third -- classically, complete ptosis with a normal reactive pupil. This is most likely ischaemia from microvascular disease.

- Horner's syndrome: no ophthalmoplegia, pupil is small, patient is able to overcome ptosis, may have anhidrosis. Trace the course of the sympathetics to look for an etiology - scars from neck surgery, pancoast tumour at apex of lung (also with intrinsic muscle wasting of hand), cavernous sinus (other cranial nerves), brainstem lesions (assess other cranial nerves, screen long tracts).
- Myasthenia gravis: nonconforming ophthalmoplegia, variable, fatigable. Determine if only ocular or if there is generalized involvement. Don't forget to percuss for thymoma!

Bilateral ptosis:

- Myasthenia gravis is the top differential: examine prolonged upward gaze for fatigability.
- Myotonia dystrophica: be alert for the myopathic facies and frontal balding
- Miller-Fisher syndrome: examine for areflexia, ataxia
- Bilateral occurrence of unilateral causes is possible but much less likely

(h) Ophthalmoplegia

Technique is especially important when testing EOM: be sure to hold your finger/pen *at least* an arm's length from the patient's face, ask the patient to tell you when he/she sees double (you may not be able to see some of the more subtle ophthalmoplegias), align your finger/pen vertically when moving in a horizontal plane and horizontally when moving in a vertical plane, be sure to stress the ends of the range of each extraocular movement (or again, you will miss the more subtle ophthalmoplegia).

Ensure that there is no multiple cranial nerve involvement (examine all branches of CN V particularly carefully), then identify one of these characteristic patterns:

- Down and out, ptosed eye → CN III
- Failure to abduct → CN VI
- Failure to adduct, contralateral eye abducts with nystagmus → Internuclear ophthalmoplegia, one and a half syndrome (localizes to ipsilateral pons)
- Nonconforming ophthalmoplegia → myasthenia gravis, miller-fisher syndrome
- Exophthalmos, proptosis → thyroid eye disease

Then attempt to localize further and identify etiology

- Examine other cranial nerves -- consider cranial nerve 'clubs', base of skull disease, meningitis, myasthenia.
- Examine long tracts -- consider crossed hemiparesis which localizes to the brainstem.
- Look for stigmata of the vasculopath which may provide the etiology of an ischaemic CN III or VI
- Beware of the false localizing VI due to raised intracranial pressure.
- Always consider the *universal diagnoses* for cranial nerves

(i) Isolated facial droop

This is quite obviously a CN VII lesion. First identify whether the lesion is a UMN VII (frontalis and orbicularis oculi spared) or a LMN (frontalis weak, eye closure weak)

UMN CN VII: examine limbs for weakness ipsilateral to the CN VII palsy → lesion is contralateral to the facial droop, in the midbrain, subcortex, or cortex

LMN CN VII:

- Examine limbs for weakness contralateral to the CN VII palsy → this is crossed hemiparesis, lesion is in the pons on the side of the CN VII palsy.
- Look for parotidomegaly or parotid surgery scar
- Look into ear for vesicles suggestive of Ramsay Hunt Syndrome
- Examine hearing → think of cerebellopontine angle lesion e.g. acoustic neuroma
- Examine CN V-VIII and eyes for internuclear ophthalmoplegia → pontine lesion
- Examine other cranial nerves → think clubs, base of skull disease, myasthenia.
- Look for complications → exposure keratitis, saliva drooling.
- End by offering to do otoscopy looking for vesicles (Ramsay Hunt syndrome)
- Always consider the *universal diagnoses*

(j) Movement disorder, Parkinsonian (hypokinetic)

Elicit the Parkinsonian features present - Inspect carefully and examine tone delicately as rigidity can be subtle and then the diagnosis will be missed.

Inspection

- Greet patient: monotonous, soft speech (hypophonia)
- Face: hypomimia (mask like)
- Hands: pill-rolling tremor
- Examine gait: patient has difficulty getting up, gait is shuffling with freezing and festination, posture is stooped with reduced arm swing, and patient needs to turn in numbers -- all features of bradykinesia.
- Examine tone: leadpipe rigidity, cogwheel rigidity (distinguish from spasticity which is found in diseases affecting the pyramidal tract)
- Elicit bradykinesia: ask patient to open and close hands ('twinkle stars')
- Examine handwriting: micrographia
- Other features: frontal lobe release signs e.g. glabellar tap (ask patient first)

Is this idiopathic Parkinson's disease? The Parkinsons-plus syndromes include multi-system atrophy (MSA), progressive supranuclear palsy (PSP), corticobasal degeneration (CBD), lewy body dementia (LBD), vascular parkinsonism.

These atypical features may suggest a Parkinsons-plus syndrome (*see full table under Parkinson's Disease in long cases*):

- Marked symmetry of signs
- Dystonias → MSA, PSP, CBD
- Pyramidal tract signs → MSA, CBD, old strokes (vascular parkinsonism)
- Cerebellar signs → MSA
- Inability of upward gaze, overcome with doll's eye → PSP
- Autonomic symptoms (postural hypotension, incontinence, severe constipation) → MSA, late PD
- Early dementia or psychiatric features → LBD

What complications are present? / How advanced is this patient in the disease?

- Mobility: gait, walking aids used (stick vs wheeled walker)
- Dyskinesias from L-dopa: look especially for tardive dyskinesia of chronic use (fly-catching tongue, gyrating hips)
- Urinary catheter: suggests urinary symptoms in late PD or MSA.

Requests:

- Postural BP → for autonomic symptoms as in MSA
- History of antipsychotic use → for EPSE
- Mini mental state examination → for dementia
- Ask about response to L-dopa.

(k) Movement disorder, Choreiform (hyperkinetic)

This is obviously abnormal even to the layman, but is not commonly encountered for most of us. Attempt to elicit the features of chorea (patient cannot sustain a posture) -

- Inspect at rest: describe the movement, note whether symmetrical or unilateral
 - Chorea: abrupt and involuntary movements that flow from one body part to another, seeming to be purposeless and non-rhythmic.
 - Athetosis: slow writhing involuntary movements
 - Myoclonus: sudden shock-like muscle contractions, often repetitive
- Hold forearms extended → look for dish-spooning (pronate forearm like dishing soup from a pot)
- Ask patient to grip fingers → patient will alternately squeeze and release (milkmaid's grip, like milking a cow)
- Ask patient to protrude tongue → look for darting (Harlequin) tongue which alternately protrudes and retracts.
- Walk the patient → effeminate gait.

Then complete the examination with the aim of looking for the etiology of chorea. For instance

- Inspection
 - Hypomimia → Parkinson's disease with levodopa-induced dyskinesia
 - Rash → SLE
 - Goitre → thyroid disease
 - Stigmata of infective endocarditis (in hands, erythema marginatum)
- Neurological examination of limbs
 - Weakness → Stroke (be especially mindful if chorea is unilateral)
 - Tremor → Parkinson's disease with levodopa-induced dyskinesia
- Eyes & Face
 - Kayser-Fleischer rings → Wilson's disease
 - Thyroid eye signs → thyroid disease

One (of many) mnemonic for etiologies is CHOREADS:

- Copper (Wilson's disease)
- Huntington's
- Oestrogen (OCP)
- Rheumatic fever (Sydenham's chorea)
- Endocrine (thyroid, glucose, calcium)
- Autoimmune (SLE)
- Drugs (antipsychotics, levodopa)
- Stroke

(I) Isolated cerebellar signs with no other findings.

This patient may present with falls or an unsteady gait. If ataxia alone, distinguish from sensory ataxia due to dorsal column disease (which will have +ve Romberg's sign, loss of vibration and proprioception, no other cerebellar signs). Decide if cerebellar signs are unilateral or bilateral -

Unilateral cerebellar lesion:

- Lesion affecting only half the cerebellum: cerebrovascular, tumor, multiple sclerosis
- Brainstem lesions: not common to affect cerebellum alone - be sure that your examination has excluded all pyramidal, extrapyramidal, and cranial nerve lesions. Significant negatives and should be presented: no motor symptoms (ataxic hemiparesis, multiple sclerosis), no tremor (MSA), no cranial nerve lesions (posterior fossa lesions, lateral medullary syndrome).

PAEDIATRIC POINTERS

In children also think of dystonic cerebral palsy due to

- Kernicterus
- Hypoxic ischaemic encephalopathy

In a child or young person, pay special attention to friedreich ataxia, ataxia telangiectasia, and Wilson's disease. In a child also think of congenital malformations and ataxic cerebral palsy.

Bilateral cerebellar lesion: bilateral lesions (e.g. bilateral strokes) are possible though less likely. A diffuse process (degenerative, metabolic) is more likely to cause bilateral cerebellar disease. Examine long tracts (pyramidal, extrapyramidal, sensory), eyes (jaundice, Kayser fleischer rings), look for telangiectasia, and features of alcoholic disease.

Type	Lesion	What to look for
Degenerative	Multiple system atrophy	Tremor, postural hypotension (older patient)
	Friedreich ataxia	Pyramidal tracts: spastic paraparesis, pes cavus Posterior column: proprioception & vibration loss Peripheral neuropathy: absent reflexes
	Ataxia Telangiectasia	Telangiectasia (check face, behind ears) Extrapyramidal: dystonia, tremor, chorea
Metabolic	Wilson's disease	Jaundice, Kayser-Fleischer rings
	Chronic alcohol use	Jaundice, parotidomegaly, Dupuytren's.
	Drugs	Epilepsy - overdose of carbamazepine, phenytoin
Inflammatory	Multiple sclerosis	Other UMN lesions, internuclear ophthalmoplegia
	Encephalitis	Request for history of varicella
Congenital	Arnold-Chiari malformation	Ventriculo-peritoneal shunt Other surgical scars
	Cerebellar hypoplasia (Dandy Walker syndrome)	
	Ataxic cerebral palsy	Diagnosis of exclusion.
Vascular	Bilateral strokes	Bilateral UMN signs - less likely Acute onset
Paraneoplastic		Cachexia

4. Examine function and complications

Finally you have come up with the intelligent diagnosis - but don't stop there. Gaze into the eyes of the patient in front of you and spare a thought - how has this affected his/her life?

- Examine function: e.g. mobility (any aids at bedside), simple motor tasks (writing, buttoning shirt, handling a coin), communication and social interaction
- Can patient swallow? > Especially for brainstem disease, motor neuron disease, myasthenia. Look for NG tube, open the abdomen for PEG tube, and look for thickened feeds. Offer a bedside swallowing test.
- Complications of immobility & spasticity: e.g. sacral sores, contractures
- Complications of LMN weakness (e.g. myasthenia, GBS): is there respiratory distress or desaturation? (request pulse oximetry)

Abdominal System

ADULTS AND PAEDIATRICS *[Main text integrates adults and pediatrics]*

The abdominal examination is reasonably straightforward to perform; its challenge lies in synthesizing the findings and suggesting likely differentials - given the large number of different diseases that could present with abdominal findings.

What is different in the child? A similar strategy works, although the priority of differentials changes slightly. In a child, always look for (and present):

- If the child appears dysmorphic (see long case dysmorphic child)
- If the child appears well thrived, but hedge that 'I would like to plot his height and weight against gender specific percentile progressive charts'.

1. Peripheries - what type of abdomen is this?

	The Chronic Liver	The Renal Abdomen	Non-obvious Abdomen
Disease	Cirrhosis of varying etiologies	Patient is currently on, or has previously required, renal replacement therapy	A mixed bag: haemato, other liver and renal, misc.
Recognition *	Jaundice [^] Stigmata of chronic liver dx: clubbing, asterixis, palmar erythema, spider naevi, hyperestrogenism, (adults only - axillary hair loss, gynaecomastia, testicular atrophy) Stigmata of ethanol use: dupuytren's contracture, parotidomegaly	Dialysis access - HD, PD, permcath scars (no dialysis does not exclude) Sallow appearance: dirty brown - impaired excretion of urinary pigments + anaemia) Characteristically grumpy - you would be too if you have thrice weekly dialysis	All other patients
Implication	Concentrate on liver, spleen, ascites. Look for etiology and cx.	Look carefully for ballotable kidneys, renal transplant.	Identify clinical picture below and proceed.

* Features of multiple types of abdomen, not helpful: pallor, ecchymosis, ascites, pedal edema.

[^] Mild jaundice can be a manifestation of chronic hemolytic anaemia, so look for other features of chronic liver disease

In the abdominal short case, there are three main types of abdomen - the renal abdomen, the chronic liver disease, and the non-obvious abdomen. From the peripheral examination, attempt to identify these three characteristic 'types of abdomen' (this will be very helpful later). The fourth main type of abdomen is the surgical abdomen with scars, stomas etc - for a medicine exam any surgical features should rightly be a sideshow, but may also offer important clues.

2. Abdomen - be sure of your signs.

The technique is an art - kneel by the bedside, palpate deeply yet gently (don't jab), scoop in with inspiration, and let the organ gently kiss your eager fingertips. If you don't feel anything - be patient, don't remove your hand till the patient finishes inspiration, and try again slightly closer to the costal margin. Deep palpation may often reveal the enlarged organ so do it carefully. *Never* take your eyes off the patient's face, because the examiner's eyes *will* be on yours to see if you are watching for pain.

If you feel something - be sure you identify the right organ and fully characterise the mass (see table). Confidence in eliciting signs may be an issue when you start out - this comes with experience but equally it comes with knowing what to look for - hence the importance of recognizing what type of abdomen this is.

Organ	How do I know it's this?	Cautions
Liver	RHC mass Cannot get above Descends on inspiration Dull to percussion	If not palpable but dull - please palpate again Beware isolated left lobe enlargement - trace the entire liver outline Characteristics of liver are imp: hard/nodular? pulsatile? tender? Adults - measure entire liver span; kids - measure cm below costal margin.
Gallbladder	Like liver but globular outline	<i>Always</i> think of this as a differential to liver
Spleen	LHC mass (notch present only if massive) Cannot get above Moves inferomedially on inspiration Dull to percussion	Start palpating from RIF - don't miss a massive spleen If -ve, trace costal margin - some spleens are better felt laterally. Resonance in Traube's space confirms no splenomegaly
Kidney	Flank mass Can get above Bimanually ballotable Resonant on percussion	To increase the chance of balloting a kidney - try to sandwich it between your fingers before balloting
Transplant kidney	Iliac fossa mass Kidney shaped outline	Rather obvious but easy to miss if you do not expose the groins!

3. Identify the clinical picture - generate etiologies & complications

From your examination, slot the patient into one of these clinical pictures:

- a) Chronic liver disease with stigmata
- b) Hepatosplenomegaly or splenomegaly alone, no stigmata of chronic liver disease
- c) Isolated hepatomegaly
- d) Ballotable kidney(s)
- e) Transplant kidney
- f) Ascites alone.

Use these clinical pictures to begin narrowing down your differentials, and to look for features that make particular differentials more likely.

(a) Chronic liver disease with stigmata

Recognize this from the peripheral findings (signs of hyperestrogenism will not be present in paediatrics). Expected abdominal findings are hepatosplenomegaly (spleen > liver) or isolated splenomegaly, +/- ascites.

Etiologies: consider these questions in turn

- With scar
 - Cholecystectomy scar: probably unrelated
 - Kocher scar (smaller): Biliary atresia post-Kasai (paeds), HCC resection (adult)
 - Mercedes Benz scar (larger, rooftop): think liver transplant
- *No scar, adults* -- look for peripheral clues:
 - Needle marks, tattoos → Chronic viral hepatitis (B/C)
 - Parotidomegaly, Dupuytren's contracture → Alcoholic hepatitis
 - Xanthelasma in a lady → primary biliary cirrhosis
 - Kayser-Fleischer rings, chorea, tremor → Wilson's disease
 - Thalassaemia facies, short stature → hemochromatosis
 - No peripheral clues → still offer viral, alcoholic, drug induced, autoimmune
- *No scar, paediatrics* -- top differentials
 - Unrepaired biliary atresia -- may have scratch marks from cholestasis
 - Autoimmune hepatitis
 - Wilson's disease -- look for Kayser-Fleischer rings, chorea, tremor
 - Beware the chronic hemolytic anaemia with jaundice.

Complications: look for them, mention as significant negatives if absent

- Signs of hepatic decompensation (think child's score)
 - Significant ascites, edema, leukonychia → hypoalbuminaemia
 - Significant jaundice (especially in adults)
 - Asterixis, confused patient → encephalopathy
 - Bruises → coagulopathy
- Significant pallor → Think variceal bleed (ddx: chronic hemolytic anaemia)
- Enlarged, craggy liver → HCC (liver should be shrunken in chronic liver disease)
- Abdominal pain → spontaneous bacterial peritonitis (won't come for exam)

Requests:

- Offer to look for the peripheral clues if you did not have time to do so.
- Do DRE for stool colour (pale, malena) and look at urine (dark)

(b) Hepatosplenomegaly or isolated splenomegaly but no chronic liver disease

The range of possible etiologies is wide, including haematological, infective, rheumatological, endocrine, and other causes. Try to narrow down based on characteristic findings and age group and offer the top few differential diagnoses -

- Massive spleen → CML, myelofibrosis (adults), malaria, Kala-Azar
- Pallor + thalassaemic facies (frontal bossing, dental malocclusion), chelation marks → Chronic hemolytic anaemia (especially paed)
- Pallor + bone marrow biopsy scar → myeloproliferative & lymphoproliferative disease, chronic hemolytic anaemia
- Lymphadenopathy → Viral (EBV, CMV - request throat examination), lymphoma
- Cachexic or bruises → myeloproliferative and lymphoproliferative disease
- Portal HTN (ascites, caput medusae) but no other stigmata of chronic liver disease → Budd Chiari syndrome.
- Joint deformities, rash, lupoid hair → Rheumatological: SLE, RA with felty syndrome
- Spade-like hands, tremor → Endocrine: thyrotoxicosis, acromegaly
- Often no obvious signs: infiltrative disease (amyloidosis, sarcoidosis), metabolic storage diseases

How to proceed depends on the likely underlying etiology. For example -

Paeds chronic hemolytic anaemia

- Think of the differentials
 - Thalassaemia major: less jaundiced (unstable Hb do not leave bone marrow)
 - Thalassaemia intermedia: more jaundiced
 - Hereditary spherocytosis: usually splenomegaly without much hepatomegaly
 - Others: autoimmune (less likely G6PD as those tend to have hemolysis in response to an insult)

- Look for complication of hemochromatosis
 - Bronzing of skin
 - Stigmata of chronic liver disease
 - Any signs of heart failure
 - Endocrine: growth failure, delayed puberty, abdominal lipodystrophy (from DM - injection marks alone can be deferoxamine)

(c) Isolated hepatomegaly

The characteristics of the liver is key -

- Hard, nodular -- mitotic lesion, consider mets vs HCC (especially if chronic liver dx- liver should be shrunken in chronic liver disease)
- Tender -- hepatitis
- Pulsatile -- tricuspid regurgitation
- Smooth, regular -- few distinguishing factors but can offer
 - Fatty liver
 - Hepatic cysts
 - Hepatic adenoma (females on oral contraceptive pill)
 - Glycogen storage diseases
 - Chronic liver disease with no stigmata (esp alcoholic, PBC, hemochromatosis)

Systemic features may also give a clue

- Systemic fluid overload -- right heart failure
- Obese patient -- consider fatty liver.

(d) Ballotable kidneys

Etiologies:

- Bilateral: ADPKD (adults), hydronephrosis 2^o bladder outlet obstruction (paeds), any unilateral cause x2
- Unilateral: RCC (adults - esp if cachectic), hydronephrosis, solitary functioning kidney (look for nephrectomy scars), ADPKD with asymmetric enlargement (adults)

Complications: especially

- If on dialysis: look for complications -
 - General complications: pallor, fluid overload,
 - Specific to HD: thrill, access issues (multiple AVF, on permcath)
 - Specific to PD: abdo pain (SBP)
- Patient with ADPKD
 - Hepatomegaly: other cysts
 - Any obvious neurological deficits (intracranial aneurysms)

Requests:

- Blood pressure
- Fundoscopy for hypertensive or diabetic changes
- Dipstick for hematuria, proteinuria, glucose
- ADPKD: neuro examination for intracranial aneurysms (e.g. 3rd nerve palsy)

(e) Transplanted kidney

Although this is unfamiliar to many, it is a relatively easy examination once you know something about transplant -

- Look for clues that could suggest etiology: e.g. ADPKD, DM (dermopathy, neuropathic ulcers, charcot joints), DM or HTN (CABG), SLE (rash, joint deformity).
- Is graft functioning? > Look for permcath or AVF with recent needling (patient back on dialysis), fluid overload (failing), graft tenderness (rejection)
- Complications of treatment: cushingoid habitus (steroids), gum hypertrophy & hypertrichosis (cyclosporin)
- Requests as per (d)

(f) Ascites alone

This situation calls for careful deep palpation as enlarged organs are often there but hard to palpate under massive ascites. Peripheral clues may make the etiology more apparent. Consider -

- Chronic liver disease -- should be obvious in the peripheries
- Renal disease -- ESRF should also be obvious in the peripheries, nephrotic syndrome and PKD may not have peripheral clues
- Heart failure -- examine the JVP
- Local exudative causes e.g. peritoneal metastasis, TB -- any cachectic patient, peritoneal nodularity

Complications wise, there should be no abdo pain (SBP), and look for complications of the likely cause.

Respiratory System

ADULTS AND PAEDIATRICS

[Main text discusses adults; unique paediatric considerations are annotated at the side.]

The respiratory examination is not easy. It takes lots of practice to be smooth enough to complete the examination within 7 minutes, yet be confident of signs elicited. If signs are subtle or localized to one area of the chest (e.g. subtle pulmonary fibrosis affecting only the lung bases), you may only auscultate the abnormality twice.

1. Get a diagnosis as early as possible

There is a limited repertoire of lung diagnoses -- as you go through the standard steps of the respiratory examination, try to clinch the likely diagnosis as early as possible; this gives your mind a great relief and frees it to look for complications and etiology. However always keep an open mind and be alert for any further findings that do not fit.

Aim to diagnose the following by this stage of examination:

- On inspection alone: COPD, pneumonectomy/lobectomy
- On chest expansion & tracheal deviation: collapse, fibrosis, large effusions
- By time of percussion: pleural effusion, pneumothorax
- May not pick up until auscultation: bronchiectasis, interstitial lung disease, localized tumors.

PAEDIATRIC POINTERS

The commonest conditions - asthma and bronchiolitis - have somewhat transient signs and are less common in exams. The same approach to the adult respi exam is handy, but alter the differentials.

Begin by looking for

- Dysmorphisms
- If child is well thrived: but hedge that 'I would like to plot his height and weight against gender specific percentile progressive charts'.

(a) At the foot of the bed: can I see or hear pathology?

Bedside interventions are a give away -

- Chest tube → certainly a pleural disease, but which? Look what is draining - is the tube to drain a pneumothorax (underwater seal and bubbling) or is it to drain an effusion (haemoserous fluid)?
- Nebulizers or inhalers by the bedside → COPD, asthma, sometimes bronchiectasis.
- Long-term oxygen therapy → end stage COPD, interstitial lung disease, occasionally cancer

Look and listen: these scream for attention -

- Barrel chested, pursed lips, audible wheezes → suspect COPD or asthma
- Cachexia, wasting, ptosis → suspect mitotic lesion
- Joint deformities, sclerodermic facies, lupoid rash, Cushingoid → think of interstitial lung disease

(b) Go closer: any scars or clubbing?

These signs are rather specific and should not be missed

- Lateral thoracotomy scars: implies lobectomy or pneumonectomy (if missed, the findings may confuse you significantly)
- Clubbing: indicates mitotic lesion, chronic suppurative lung disease (bronchiectasis, abscess), or interstitial lung disease.
- Elevated JVP, pedal edema → Fluid overload.

(c) Palpation: do chest expansion and tracheal deviation give an answer?

- Chest expansion is best elicited on inspection and confirmed on palpation; the side that expands less is always abnormal.
- Tracheal deviation may be to or away from the abnormal side.
- The constellation of chest expansion and tracheal deviation findings may allow identification of a unilateral lung pathology (but absence of chest expansion or trachea deviation does not exclude pathology)

PAEDIATRIC POINTERS

COPD does not happen in children; think asthma vs bronchiolitis

Stridor may be heard.

Cachexia: not cancer, think of failure to thrive 2' cystic fibrosis or chronic lung disease of prematurity (see under wheeze later)

Look also for a subcostal scar - hypoplastic lung 2' congenital diaphragmatic hernia s/p repair

Clubbing: examine both fingers and toes. Cancer and ILD are rare in kids

Harrison's sulcus indicates chronicity

Hypoplastic lung presents with a 'collapse' picture - decreased expansion, and tracheal & mediastinal shift to same side. If due to congenital diaphragmatic hernia there may be subcostal scar.

	THIS side expands less	
	No scar	Scar
Trachea deviated to THIS side	Fibrosis Collapse	Pneumonectomy Lobectomy
Trachea central	Consolidation	
Trachea deviated to OPPOSITE side	Pneumothorax Pleural effusion / Hemothorax	

Note: if the trachea is not deviated, beginning palpation from the back may be of higher yield.

PAEDIATRIC POINTERS

(d) Percussion: what is abnormal?

- Remember to percuss all the way down to T12 (lower than you think!) and also in the axillae, comparing both sides. If not you might miss a small effusion.
- Dull: pleural effusion (stony dull), consolidation (dull).
- Hyperresonant: pneumothorax if unilateral, COPD if bilateral (may be asymmetrical due to emphysematous bullae).
- Confirm your findings on auscultation.

In kids, hyperresonance means pneumothorax

(e) Auscultation: if everything is normal thus far.

By this time you should be looking for relatively subtle pathology - gross effusions, collapses, and obvious COPDs should have been ruled out. Auscultation is difficult so if you can try to make a diagnosis before reaching this step!

Wheezes:

- Diffuse polyphonic wheeze: asthma or COPD
- Localized monophonic wheeze: be alert, this suggests bronchogenic carcinoma (monophonic because only one airway is narrowed)

Stridor is uncommon in adults.

Wheezes: think of

- Asthma: look for atopy - eczema, allergic shiners
- Bronchiolitis: a ddx in a younger child
- Chronic lung disease: in a small infant with plagiocephaly

Stridor is harsher, often inspiratory (supraglottic) over expiratory (infraglottic).

Think:

- Well kid: laryngomalacia, vascular ring, craniofacial abnormality
- Sick kid: epiglottitis, croup
- Premmie with biphasic stridor: glottic stenosis 2' prolonged intubation
- Abdo scars: laryngeal strictur 2' reflux.

PAEDIATRIC POINTERS

Crepitations: important but a source of stress for many.

- Interstitial lung disease: classically fine end-inspiratory crepitations. If you struggle to identify what crepitations are fine, they can be identified by their (1) Character: like the sound of velcro tearing, (2) Number: fine crepitations are many (10+) while coarse crepitations are few (3-7), and (3) Timing: fine crepitations start in the middle of inspiration and continues to end inspiration. If a patient with known interstitial lung disease (or obvious rheumatological disease) has coarse crepitations, suspect superimposed infection.
- Bronchiectasis: loud coarse early inspiratory crepitations (start early in inspiration, continue to mid inspiration, fade by end of inspiration). There is a characteristic sound like air bubbling through water - which *is* what bronchiectasis is! Ask the patient to cough - crepitations may change on coughing.
- COPD: although wheeze is more classical than crepitations, crepitations are often heard in several situations (1) scattered coarse crepitations due to bronchi opening, as part of COPD, (2) coarse crepitations due to concomitant bronchiectasis, (3) coarse crepitations due to infective exacerbation.
- Pulmonary edema / fluid overload: pan-inspiratory; elevated JVP and pedal edema is the give away.

Paediatric ILD is uncommon so it becomes less important to consider fine vs coarse creps.

Bronchiectasis is the most likely cause of creps in paediatrics

- Exclude active asthma
- Also consider chronic lung disease of prematurity (small infant with plagiocephaly)

Children do not get COPD

Heart failure is important to rule out

Bronchial breathing: louder and harsher than vesicular (normal) breath sounds. Pay attention to the duration of inspiration and expiration - bronchial breathing is characterised by both phases of equal duration, not separated by a pause. Bronchial breathing implies consolidation.

Differential in paed: hypoplastic lung

2. Consider etiology

This is quite specific to the underlying disease. Start thinking and quickly examine for further features -

Pleural effusion: think unilateral vs bilateral.

- Unilateral
 - Mitotic lesion: cachexia, lymph nodes, miosis, hand intrinsic muscle wasting,
 - TB: may also be cachetic with lymph nodes
 - Parapneumonic: ask for temperature chart
- Bilateral: causes of fluid overload - cardiac (JVP), renal (examine for dialysis access, ascites, request dipstick)

Interstitial lung disease

- Examine for secondary causes - features of RA, SLE, scleroderma, AS.
- Consider based on distribution.
 - UZ: occupational (pneumoconiosis, silicosis), TB / sarcoidosis, rheumatological (AS), post RT
 - LZ: idiopathic (IPF), occupational (asbestosis), rheumatological (RA, scleroderma), drugs (methotrexate, amiodarone, etc).
- Ask for occupational history, medications history

Bronchiectasis:

- Diffuse: due to inability to clear secretions or another predisposition to recurrent infections
 - Cystic fibrosis
 - Primary ciliary dyskinesia (Kartagener's): a triad of sinusitis, dextrocardia, bronchiectasis - examine apex beat, request ENT for otitis media
 - Other immunodeficiencies
- Focal: due to local damage.
 - Post infective (pneumonia, TB) - look for dysphagia causing aspiration pneumonia, if pt is foreign looking think TB.
 - Tumor
 - Foreign body

Consolidation:

- Infective (bacterial, TB): listen for productive cough, look for sputum mug, request temperature chart
- Neoplastic: look for cachexia, lymphadenopathy, SVC obstruction, clubbing.

PAEDIATRIC POINTERS

Unilateral effusion in kids: TB vs parapneumonic. Less likely mitotic lesion.

ILD is uncommon in kids

Almost always diffuse in paediatrics

- Cystic fibrosis: Look also for PEG feeding, failure to thrive.
- Kartagener: examine for apex beat and liver.
- Ddx: bronchiolitis obliterans

Ddx: hypoplastic lung - consolidation plus subcostal scar (repair of diaphragmatic hernia), midline shift

Cardiovascular System (adult)

ADULTS

[The paediatric cardiac examination is quite different and is presented separately]

Among the medicine short cases, the cardiac station perhaps seems the most straightforward. But under exam pressure, much depends on your ears (and what is between them) - it is easy to score in a cardiac short case, but also easy to make a mess of should you get flustered, lose your technique, miss or (gasp!) invent signs. It is particularly dangerous to be going through the steps without attempting to piece together the overall picture from the start. Know what you are doing each step, convince yourself of the sign, and think of what it means before you move on.

The standard sequence of examination is inconvenient because it brings you to complications before diagnosis and so on; but follow it you must. As you do so, consider these questions -

1. What is the diagnosis?

There are only two types of adult cardiac examinations - a prosthetic valve or a murmur. In the rare occasion you see a cyanosed adult - turn to the paediatric approach.

Is there a scar? Time spent on inspection - if you know what you are looking for - never goes to waste. The first and most crucial question in every cardiac case is: is there a scar?

- Midline sternotomy -- These are either prosthetic valves or coronary artery bypass. Go close to the patient and listen hard for the metallic clicks of a prosthetic valve; subsequently, at auscultation, listen hard for metallic heart sounds which you might otherwise miss.
- Anterior thoracotomy scars -- Possibly mitral valve repair / replacement, or interventions for congenital heart disease (e.g. blalock-taussig shunt)
- Complicated pattern of scars -- start suspecting repaired congenital heart disease (see approach to paediatrics).

Peripheral clues: classic teachings but we are seldom able to use these with confidence

- Radial-radial and radial-femoral delay (Coarctation) -- actually quite important because coarctation would otherwise be hard to suspect (and confusing on examination)
- Collapsing pulse (Aortic Regurgitation) -- highly informative if convincingly present, but difficult to be convinced unless you have palpated many.
- Slow-rising pulse (Aortic Stenosis) -- usually gives a suspicion but a soft sign.

- Nature of apex beat -- heaving (forceful, sustained) in AS, thrusting (diffuse, nonsustained) in MR, tapping (MS).
- Clubbing -- be very careful for congenital cyanotic heart diseases, alternatively, mention as a complication of possible infective endocarditis.

Auscultation and dynamic maneuvers: the make-or-break of the cardiac examination. Time any murmur you hear and identify the classic features of each murmur

- Aortic Stenosis: ejection systolic murmur best heard over aortic area, radiating to the carotids (if not radiating to carotids, consider aortic sclerosis).
- Mitral regurgitation: blowing pan-systolic murmur best heard at apex, radiating to axilla
- Tricuspid regurgitation: pan-systolic murmur best heard over lower left sternal edge, with giant c-v waves on the JVP, +/- pulsatile liver
- Ventricular septal defect: pan-systolic murmur best heard over lower left sternal edge with no radiation and normal JVP.
- Aortic Regurgitation: early diastolic murmur best heard on lower left sternal edge, accentuated by leaning forward in full expiration
- Mitral stenosis: soft mid-diastolic murmur (+/- late diastolic accentuation if not in AF) best heard in apex, with the bell in the left lateral position.
- Patent ductus arteriosus: continuous systolic and diastolic murmur.
- Coarctation of aorta: systolic murmur at *upper* left sternal edge, radial-femoral delay, radiation to back (interscapular region)

Prosthetic valves:

- Please identify which valve is prosthetic - the mitral, aortic, or both. Listen to S1 and S2 and identify which sound is metallic.

Tips and traps:

- *Beware:* a soft murmur may become inaudible if auscultated in the wrong location; palpate carefully for the apex beat before plonking your stethoscope down, and in all positions attempt to move your stethoscope around slightly to hear more clearly. Dextrocardia is an exam favourite: if you can't palpate the apex, check the right side!
- *What if you're not sure?* Listen again - at the very least you must be able to tell systolic vs diastolic murmurs. Pay attention to nuances - in an ejection systolic murmur you can hear S1 and S2; in a pansystolic murmur you cannot. If really unsure present findings and offer differentials.
- *Are auscultation findings consistent with other clues?* For example, if there is a systolic murmur you aren't sure about, but the patient's apex is terribly displaced and the patient is in AF, MR is more likely than AS.
- *Multiple pathology:* patients may have multiple murmurs, or prosthetic valve with a murmur, so keep an open mind even after you identify one diagnosis.
- *Presentation:* murmurs should be described in full, as above but also including grade of murmur, e.g. 'this patient has mitral regurgitation, I say this because he has a blowing grade 3/6 pansystolic murmur best heard over the apex, radiating to the axilla'.

2. How severe is this murmur?

Attempt to grade the severity of the murmur -- it is not always how loud the murmur is.

- Mitral regurgitation: Loud murmur, apex displacement
- Aortic Stenosis: A long murmur, late peak, weak pulse, soft A2 signifies severity. Can also request to ask the patients for syncope, shortness of breath, or chest pain.
- Aortic regurgitation: long murmur, wide pulse pressure, soft A2.
- Mitral Stenosis: Severity is implied if there is a soft 1st heart sound, long murmur, early opening snap (immobile valve cusps)
- Presence of complications (CCF, pulmonary hypertension) usually signifies severity.

3. What is the etiology?

- *Prosthetic valve*: the apex beat is key - displaced (more likely regurgitation) vs not displaced (more likely stenosed)
- *Multiple valve pathology, MS*: likely rheumatic heart disease or infective endocarditis (but these can cause any pattern or affect any other valve)
- *AR: (causes of aortic root dilatation)* - Marfans syndrome (look for Marfanoid features, ask patient to spread arms to side), aortic dissection, ankylosing spondylitis (ask patient to bend forward)
- *AS*: calcific degenerative AS (older), congenital bicuspid valve (younger)
- *MR*: ischaemic heart disease (look for DM dermopathy, obesity), mitral valve prolapse in a younger person, degenerative, dilated cardiomyopathy

4. What complications are present?

In all cases: look for these peripheral features

- Atrial fibrillation (and if present look for overwarfarinization)
- Cardiac failure: raised JVP (*pressure* not pulse), pedal edema, basal crepitations
- Stigmata of infective endocarditis: Janeway lesions, Osler nodes, clubbing, splinter haemorrhage
- Pulmonary hypertension: palpable P2, parasternal heave.

In prosthetic valves also look for:

- Are the valves crisp → if not have to consider endocarditis or thrombosis
- Any flow murmur: a flow murmur is systolic for the aortic valve and diastolic for the mitral valve, if soft it may be normal
- Any regurgitant murmurs → abnormal
- Overwarfarinization: bruising etc

5. Requests

- All cases - blood pressure (as part of the cardiac examination)
- All valves and murmurs - further bedside investigations for infective endocarditis (fundoscopy for roth spots, dipstick for hematuria, temperature chart)

Cardiovascular System (paeds)

PAEDIATRICS

[The cardiac examination in adults is presented separately]

The paediatric cardiovascular examination is quite a different ball game from its adult equivalent; as a whole new world of congenital conditions have to be considered. You must inspect hard and narrow down your differentials even before you pick up your stethoscope; if not, you will struggle to hear a murmur tucked between the child's rapid S1 and S2 beats, or be utterly confused by the complicated murmurs of complex congenital heart disease. When you examine, do so opportunistically (with the child in his/her mother's arms if possible). Examination becomes difficult if the child cries, but do not lose heart. Try to placate the child (a skill in itself) and if all else fails at least you can discuss the differentials you generated on inspection.

Opening moves

As always in paediatric cases, begin by looking for (and presenting)

- Dymorphisms: congenital heart disease is associated with many genetic syndromes; at least know the association of Down's syndrome with VSD (most common) and AVSD (pathognomonic).
- If the child is well thrived, but hedge that 'I would like to plot his height and weight against gender specific percentile progressive charts'.

The critical question in this examination - *is the child cyanosed?* Sometimes it is obvious from the foot of the bed - the child has blue face, blue lips, may be on supplemental oxygen or an SpO2 monitor. At other times it may be less obvious. Examine the hands and feet - if there is clubbing, the child is almost certainly cyanosed. If there is peripheral cyanosis (even if no central cyanosis), there may be polycythaemia masking central cyanosis and the child is likely cyanosed. Examine the tongue and oral mucosa closely (and use a neutral-coloured torchlight). Do not move on until you are sure about whether this critical sign is present or absent!

I. The Cyanotic Heart

1. Recognize a cyanotic heart. ‘Sir my patient has cyanotic heart disease because he is cyanosed, clubbed, and looks small’.

2. Identify any surgical palliation. Look for the scars of heart surgery - if the child is still cyanotic these procedures must have been palliative; repaired congenital heart disease should be pink.

- Lateral thoracotomy: think of Blalock-Taussig shunt (subclavian - pulmonary artery bypass). This may be unilateral or bilateral. Auscultate in the subclavicular region for a continuous murmur.
- Midline sternotomy: other palliative surgical procedures

3. Etiology: RVOTO or not.

- Cyanotic congenital heart disease can pathophysiologically be divided into two groups: those with right ventricular outflow tract (RVOT) obstruction and those without (usually too sick for exams) -
- If dysmorphic: think of associated cardiac defects.

	RVOT obstruction	Non RVOT obstruction
Pathophysiology	RVOTO, right to left shunt	Common mixing
Pulmonary blood flow	Reduced, hence - Lungs: no creps - Not in respiratory distress	Increased, hence - Lungs may sound wet - Respiratory distress may be marked
Palliation	B-T shunt bypasses RVOT obstruction (presence of B-T shunt implies an RVOTO)	B-T shunt not used
Examples	Tetralogy of fallot Tricuspid atresia Pulmonary atresia (single S2)	Atrioventricular septal defect Truncus arteriosus Totally anomalous pulm venous return Hypoplastic left heart disease VSD/ASD with Eisenmenger’s syn*

* Think of this in a teenager or young adult; with cyanosis + pulmonary hypertension

4. Complications. Look for

- Heart failure
- Stigmata of endocarditis
- Failure to thrive
- Neurological deficit (observe whether child is moving all 4 limbs): due to paradoxical emboli across a right to left shunt.

II. The Acyanotic Heart

Examine as for an adult, taking special care to palpate the apex bilaterally so as not to miss dextrocardia, and paying attention to the second heart sound. The approach is similar to that in adult, although the priorities of differentials change.

1. What is my diagnosis?

- Identify one of the following clinical pictures and proceed to consider differentials for each (diastolic murmurs probably uncommon) -
- Paediatric heart disease may be complicated; if you are unsure, trust your ears over your guessing - present what you hear and offer differentials.

(a) Scars present

- Cyanotic heart disease, s/p complete surgical repair. There may be further residual lesions (e.g. PS-PR murmur from tetralogy of fallot repair)
- Non-cyanotic surgery.

(b) Dextrocardia

- Examine liver: distinguish situs inversus vs isolated dextrocardia (worse prognosis, usually unrepairable).
- Look around for other deformities or surgical scars as per the VACTERL association: vertebral, anorectal, cardiac, tracheoesophageal, renal, and limb.

(c) ESM at Aortic / ULSE: look for clues -

- Atrial Septal Defect: fixed split S2
- Coarctation of Aorta: radial-femoral delay or just diminished femoral pulse, radiation to back (auscultate between the scapulae).
- Aortic Stenosis: Suprasternal thrill or radiation to carotid
- Pulmonary Stenosis: nothing special.
- Innocent murmur: soft, short murmur whose character changes with maneuvers (e.g. getting patient to sit up while auscultating). It may have a distinctive vibratory character (Still's murmur) but you are unlikely to recognize this character unless you have heard many. There will be no complications

(d) PSM at LLSE

- Ventricular septal defect: radiation to right of sternum.
- Mitral regurgitation: radiation to axilla
- Tricuspid regurgitation: giant c-v wave on JVP, pulsatile liver

(e) Apparently continuous murmurs.

- Coarctation of Aorta: radial-femoral delay or just diminished pulse, radiation to back.
- Double murmurs: a 'to and fro' murmur e.g. AS-AR, PS-PR. Listen carefully for the pause between both components of the murmur.

2. How severe is this murmur?

- As in adults - a louder murmur is not necessarily more severe
- For VSDs in particular a large defect causes a soft murmur.

3. What is the etiology?

Much less to comment on than in the adult

4. What complications are present? As in adults -

- Atrial fibrillation
- Congestive cardiac failure:
- Infective endocarditis stigmata:
- Pulmonary hypertension: except for PS where there will be no pulmonary HTN.
- Failure to thrive
- Eisenmeinger syndrome in a L>R shunt: would have cyanosis, clubbing.

Other Systems

ADULTS AND PAEDIATRICS

[Main text is an integrated discussion of both adults and paediatrics]

You may be asked to examine other systems (rheumatology, endocrine, dermatology, miscellaneous). These diseases tend to have quite unique features and multi-system manifestations. The key is to recognize the diagnosis on inspection and proceed with a disease-specific examination - in this way it is a test of how well you understand the manifestations and associations of each condition.

This approach provides a general framework. Apply the thought process here unto your knowledge of each disease's unique features. Scripts for each condition can be found in *Jansen Koh, Baliga 250 cases*, and other resources.

1. Understand the stem

The stem is usually there to guide you so listen to it carefully. Think broad - do not restrict your differentials to a single system. For instance -

(a) Please examine the hands

- Joint deformities: rheumatoid, gout, psoriatic arthropathy, osteoarthritis
- Skin changes: scleroderma, dermatomyositis, tophaceous gout, psoriasis.
- Nerve lesions: ulnar, median, and radial, lower cervical spine (myelopathy, root, Horner's syndrome)
- Clubbing: cardiovascular (IE, cyanotic heart disease), respiratory (bronchiectasis, ILD, cancer, abscess), gastrointestinal (cirrhosis, IBD), thyroid, pseudoclubbing (scleroderma)
- Dupuytren's contracture

(b) Please look at the face and proceed

- Endocrine: Cushing's, acromegaly
- Rheumatological: scleroderma
- Neurological: cranial nerve lesions (facial droop, ptosis)
- Goitre -- see approach to surgical short cases.

(c) This patient complains of skin changes, please examine

- Rash: psoriasis, lupus, dermatomyositis, purpura, vasculitis (and other - see approaches notes)
- Neurocutaneous diseases: neurofibromatosis, tuberous sclerosis

(d) This patient has a deformed large joint:

- DM: Charcot's joint
- Rheumatological causes: seronegative spondyloarthropathy (or JIA in a child), crystal arthropathy
- Hemophilia

(e) Look and proceed: a very broad stem which includes all spot diagnoses e.g.

- Neurology: movement disorder (Parkinsonism, chorea), cranial nerves, myotonic dystrophy.
- Endocrine: Cushing's disease, acromegaly, goitre
- Rheumatology: ankylosing spondylitis, scleroderma
- Skin: neurocutaneous, dermatomyositis, SLE, purpura, psoriasis.
- Others: Marfan's syndrome

(f) Presenting complaint type stems, for example

- This patient complains of headaches → think acromegaly
- This patient complains of clumsiness → cervical myelopathy, cerebellar, rheumatoid hands
- This patient complains of feeling hot → thyroid.
- This patient has back pain → ankylosing spondylitis (or other spondyloarthropathy)
- This patient has difficulty swallowing → scleroderma, dermatomyositis, huge goitre.
- This patient has joint pain and rash → psoriatic arthropathy; consider doing GALS screen (especially since you do not yet know whether it is axial or appendicular predominant)

2. Recognize the diagnosis

- This is the key to any such examination and allows you to hone in on disease-specific features.
- You must begin with a high index of suspicion and that will allow you to look for the relevant features.
- If a diagnosis does not appear obvious - think in terms of systems (endocrine, rheumatological, neurological, etc).

3. Elicit and describe the features present

- Specific examinations have fixed sequences, e.g.
 - Joints or hand: look - feel - move (plus do nerve screen) → refer orthopaedic short cases
 - Thyroid → refer to surgical short case thyroid
- The others tend to be more fluid and rely very heavily on inspection and simple tests. Whatever you do, appear to have a system (e.g. face - hand - torso) rather than jumping around randomly; and make a show of what you are looking for.
- Be wary of differential diagnoses, e.g.
 - Rheumatoid arthritis → Make sure no psoriatic plaques, gouty tophi around

4. Discuss severity or disease activity

- Rheumatoid hands: describe activity (i.e. tenderness, bogginess)
- Scleroderma: distribution of sclerodactyly (proximal to elbow or distal)
- Look closely for any surgical scars.

5. Look for complications: depending on the multiple systems involved, for example

- Rheumatoid arthritis: examine eyes (uveitis), look at face (Cushing's), auscultate lung (interstitial lung disease), palpate spleen (Felty)
- Graves' disease: pulse (AF), screen neuro (stroke 2' AF)
- Acromegaly: neck (goitre), hands (carpal tunnel), heart (cardiomegaly), abdomen (organomegaly, lipodystrophy from insulin), request BP, urine dipstick.
- Psoriasis: look for joint deformities or swellings.
- Marfan's syndrome: murmur (AR), chest tube scar (pneumothorax)
- Tuberous sclerosis: murmurs (cardiac rhabdomyoma), ballotable kidneys (angiomyolipoma), IQ.
- Complications of treatment -- steroids (autoimmune diseases)

6. Look for etiology if possible, for example

- Cushing's syndrome: examine visual fields for bitemporal hemianopia (pituitary tumor), hyperpigmentation (ACTH secretion), and look for virilization (adrenal tumor). Look for signs of systemic disease for which steroids are given - dialysis (glomerulonephritis), rash (lupus), joint deformity (rheumatoid arthritis), edema (nephrotic syndrome).
- Acromegaly: examine visual fields for pituitary adenoma.

7. Examine function if applicable

- Target this to the likely disability - e.g. deformed hands, examine writing, buttoning, fine motor.

Developmental Assessment (paeds)

INTRODUCTION

Developmental Assessment is not difficult but you must know what you are doing. It requires both that you have a systematic method, as well as the flexibility to go with the flow, varying your examination according to what the child is doing.

Start by general inspection. As for any paediatric short case, answer these 4 questions:

- Is the child alert and comfortable?
- Are there any interventions present? (e.g. IV drip, NG tube, etc)
- Does the child look well-thrived for his age?
- Are there any dysmorphic features?

Then proceed to assess development in each of the 4 domains – vision and fine motor, hearing and speech, gross motor, and social development. Note that exams test DA up to an age of 2.5 years old (but please learn until at least 3 yrs)

- In an infant, one method is to start with vision, hearing, then fine motor, and speech. For gross motor, do the 180 degrees flip examination – but leave this till the end as it may upset the child. Plenty of information about the gross motor domain can be gathered from inspection alone. If the child appears to be <6 months, also assess the primitive reflexes. Observe social development as you interact with the infant
- In a toddler 1-2 years old, do vision, hearing, fine motor, speech, and gross motor by getting the child to walk/move around. Observe social development as you interact with the infant. It is no longer appropriate to do primitive reflexes – you do not expect these to be present.
- In an older toddler who is already talking and playing, it is no longer necessary to start with vision and hearing – if the child is already talking, the inference is that he/she is able to hear so as to learn how to talk. Do the other domains, being sure to engage the child.

In each domain, find the cutoff between what the child can and cannot do, so as to pinpoint the developmental age. ‘Child able to stack 6 blocks’ implies that the child is at least 2 years old, but does not tell you exactly how old the child is. ‘Child able to stack 6 blocks but not 8’ implies the child is at least 2 years old, but not yet 2.5 years old. If there is any delay – is it isolated to one domain or global?

Prepare your DA kit with thought – know what each object in it is for and how to interpret: do not offer the child a toy and not know how to interpret what age the child is shown to be!

At the end, it may be helpful to talk to the parent to gather any information you were not able to (e.g. if the child would not talk to you) – do ask the examiner for permission first.

STEPS & MILESTONES

Key milestones for each domain must be memorized as a sequential progression; unfortunately there is a fair amount of memory work and there are no real shortcuts. You may find that milestones vary slightly between references; distinguish between median ages and limit ages, and do appreciate that there is a 'range of normal'. Reference used for this document is mainly the NUH guidebook 'paediatrics on the go'. A more comprehensive milestone chart is the DDST Singapore (attached at the end of this guide).

Vision

Approach with a red pom pom ball. Does the child fixate on the object? Can the child track as you move it laterally?

- Fixate and follow to 90° - 6 weeks
- Fixate and follow to 180° - 3 months
- Fixate and follow vertically - 4 months
- Reaches out for the object - 5 months
- Object permanence - 9 months

Hearing

Distract the child and ask the examiner to ring a small bell on either side of the child, away from his/her view.

- Watch for startle, changes in facial expression - newborn
- Localizes at same level (turns head) - 6 months
- Localizes above/below level (turns head) - 9 months

If there is no response, there are parental concerns or any doubt, request formal audiometry.

Fine motor

Offer small objects (e.g. raisin, sticker)

- Holds rattle (hand unfisted) - 3 months
- Palmar grasp - 6 months
- Transfers - 7 months
- Immature pincer grasp - 9 months
- Mature pincer grasp - 12 months

Offer cubes:

- Stacks 2 cubes - 15 months
- Stacks 3 cubes - 18 months
- Stacks 6 cubes - 2 years
- Stacks 9 cubes - 2.5 years
- Makes a bridge - 3 years

Offer a pen and paper. Describe the grasp – an adult pen grasp is tripod, many children younger than 2 years are able to scribble with a palmar (supinator) grasp, or a pronator grasp.

Invite the child to copy shapes:

- Scribbles - 15-18 months
- Draws line - 2 years
- Draws circle - 3 years
- Draws cross - 3.5 years
- Draws square - 4 years
- Draws triangle - 5 years

Offer picture book:

- Turns 2-3 pg at a time - 18m
- Turns 1 pg at a time - 2y

Beware of premature hand preference < 18 months. This may be a sign of cerebral palsy.

Speech & language

Observe the child's language:

- Babbling - 6 months
- Indiscriminate dada, mama - 7 months
- Discriminate dada, mama - 10 months
- 2-3 other words - 12 months
- 2-3 word phrases - 2 years

Attempt to interact with the child as you play with him/her asking him/her to -

(a) Point to body parts:

- Point to object of interest - 15 month
- Points to 2-3 body parts - 18 months
- Points to 4-5 body parts - 2 years

(b) Naming

- Name some picture cards - 18 months
- Names 3 objects - 2 years
- Names 1 colour - 2.5 years
- Names 2 colours - 3 years
- Names 3 colours - 4 years

(c) Commands

- Follows 1 step commands - 1 year
- Follows 2 step commands - 2 years
- Follows 3 step commands - 3 years

(d) Other general things to ask

- Name, age, sex - 3 years
- Count to 10 - 3 years
- Count to 20 - 4.5 years

Personal, social, behavioural

This is mostly observation, comment on what you see along the way.

- Social smile - 6 weeks
- Stranger anxiety - 6 months
- Separation anxiety - 9 months
- Waves byebye, claps hands - 10 months
- Mouthing - up to 12 months

Look for diapers:

- Dry by day - 2 years
- Dry by night - 3 years

Offer a play object:

- Object permanence (look for hidden toy) - 9 months
- Casts toys - 12 months
- Pretend play - 18 months
- Plays with others - 3 years

Ask about ADLs

- Feed self with spoon - 9 months
- Drink from cup - 15 months
- Helps with dressing - 2 years
- Dresses and undresses fully - 3 years
- Can make cup of milo - 3.5 years
- Can go toilet independently - 4 years

Be alert for Autism spectrum disorder features, e.g.

- Cannot point to object of interest
- No eye contact, no interaction
- No pretend play
- Repetitive actions, preoccupation with certain objects
- Language delay

Gross motor – older child

Observe walking:

- Walks independently or with 1 hand held - 12 months
- Walks steadily, can stoop to pick toy - 15 months
- Walks backwards - 2 years
- Runs - 2 years
- Tiptoes - 2.5 years
- Jumps on both feet - 3 years
- Stands on 1 foot for 5 seconds - 4 years

Offer a ball:

- Throws ball while standing - 18 months
- Kicks ball - 2 years

Staircase (ask parent):

- With assistance or railing - 18 months
- Alone up stairs, 2 feet per step - 2 years
- Alone up stairs, 1 foot per step - 3 years
- Alone down stairs, 2 feet per step - 3 years
- Alone down stairs, 1 foot per step - 4 years

Observe fine motor and beware of the child who only uses one limb.

Gross motor – 180° flip examination

Do this for children who are yet to be able to walk independently

Supine: inspect posture

- Normal: moves all 4 limbs and rests in a slightly flexed posture.
- Spastic: Asymmetrical limb movements, persistent fist clenching, all 4 limbs extended
- Flaccid: frog-leg posture

Pull to sit:

- Less head lag - 2 months
- No head lag, good head control - 4 months
- Lifts head in anticipation - 6 months

Sitting:

- Sit with straight back, tripod stance - 6 months
- Sits steadily, no support - 7 months

Attempted weight bearing:

- Bears full weight - 6 months
- Pull to stand - 9 months
- Observe also for scissoring, suggesting spasticity

Ventral suspension: look for tone and head control (vs flaccidity – rag doll appearance; or spasticity). *Always ask for permission - request the mother to hold if you are not confident of doing so.*

Prone:

- Lifts head - 2 months
- Rolls over - 4 months
- Supports weight on hands - 6 months
- Creeps and crawls - 10 months

Primitive reflexes

- Sucking/rooting - up to 4 months
- Palmar grasp - up to 3 months
- Moro - up to 4 months
- Asymmetric tonic neck reflex - 2 to 6 months
- Parachute - 6-12 months

SAMPLE SCRIPTS**6-month old**

Alice is a pleasant Chinese infant, alert and comfortable at rest. I do not note any active interventions, and she does not appear dysmorphic. She appears well thrived for age but I will like to plot her height, weight, and occipital-frontal circumference against gender-specific progressive percentile charts.

On examination of vision and fine motor, she is able to fixate and follow up to 180 degrees, and reaches out for my object. She has a palmar grasp but not yet a pincer grip, and is able to transfer objects across the midline.

On examination of hearing and speech, she is able to localize sound to the same level, both left and right. She babbles but has yet to call daddy and mummy.

I performed a 180 degree flip examination for gross motor. Alice moves all 4 limbs normally. On pulling to sit, she has no head lag. She sits with a straight back in a tripod stance. She is able to weight bear and I do not note scissoring of the legs. Prone, she lifts her chest off the bed by supporting her weight with her elbows, but has yet to crawl.

In terms of social development, she is pleasant and has started to develop stranger anxiety. She is not yet able to wave bye-bye to me.

In summary, my examination suggests a developmental age of 6 months in all domains.

1-year old

Benny is a 18-month old Chinese boy. I do not note any active interventions. He appears small for age and I will like to plot his height, weight, and occipital-frontal circumference against gender-specific progressive percentile charts. He appears to have plagiocephaly and I note an old tracheostomy scar.

On examination of vision and fine motor, he is able to fixate and follow objects 180 degrees. He has a mature pincer grasp but is not yet able to scribble or to stack 2 cubes.

On examination of hearing and speech, he is able to localize sounds above and below ear level bilaterally. He is able to call daddy and mummy specifically, and can say 'no'. He is not yet able to name any picture cards or body parts.

On examination of gross motor, he walks unsteadily, and is not yet able to run or jump. He cannot throw a ball.

In terms of social development, he is able to wave goodbye to me. He demonstrates object permanence, however he is not yet able to engage in pretend play with a toy car.

In summary, Benny is an 18-month old, small for his age, with features to suggest that he was born premature. His developmental age is about 1 year in all domains. I will like to know his corrected age.

18-month old

Charles is a 2-year old Chinese boy, comfortable at rest with no interventions. He appears dysmorphic with upslanting palpebral fissures, hypertelorism, flat nasal bridge, low-set ears, broad neck, single palmar crease, and a wide sandal gap.

On examination of vision and fine motor, he is able to fixate and follow objects 180 degrees. He has a mature pincer grasp, and is able to stack 3 blocks but not more. He holds a pen with a palmar grasp and can scribble, but is not yet able to draw a line.

On examination of speech and language, he is able to localize sound above ear level bilaterally. He calls daddy and mummy specifically, but has yet to develop 2-3 word phrases. He can name some picture cards and point to 2 body parts, but not yet follow 2-step commands.

On examination of gross motor, he walks well, but cannot run or jump or throw a ball. He cannot climb staircases.

In terms of social development, he is very friendly and waves bye-bye. He demonstrates object permanence and is able to engage in pretend play with a toy car. He is still on diapers both day and night.

In summary, Charles is a 2-year old with a developmental age of 18 months. He has global developmental delay with dysmorphic features suggestive of Down's syndrome.

2-year old

Danny is a 2-year old Eurasian toddler, alert and comfortable at rest, with no active interventions and no dysmorphisms. He appears well thrived for age but I will like to plot his height, weight, and occipital-frontal circumference against gender-specific progressive percentile charts.

In terms of fine motor, he has a mature pincer grasp, is able to draw a line but not a circle, and stacks 6 but not 8 blocks.

In terms of gross motor, he walks well and runs, but is not yet able to tiptoe or jump with both feet off the ground. He throws and kicks a ball.

In terms of social development, he is very sociable, and is no longer on pampers in the daytime. He is able to pretend play with a toy car, but has yet to help with dressing

In terms of speech and language, he is not able to say 2-3 word phrases, understand commands, or name picture cards. His parents report that he has only just begun to call daddy and mummy specifically and has no other words. I note that he is able to localize sounds above the level of his ears bilaterally.

In summary, Danny is a 2-year old with isolated speech and language delay. His developmental age is 10 months in speech and language, but 2 years in all other domains.

2.5-year old

Elizabeth is a playful Chinese toddler, alert and comfortable at rest with no active interventions. I do not note any dysmorphisms. She appears well thrived for her age but I will like to plot her height, weight, and occipital-frontal circumference against gender-specific progressive percentile charts.

In terms of fine motor, she is able to stack 8 blocks but not yet build a bridge. She holds a pen with a mature tripod grip and can draw a line and a cross, but not yet a circle.

In terms of speech and language, she is able to express her demands for my stickers in 2-3 word phrases, can point to more than 5 body parts, and follow 2-step commands. However she is not yet able to give her name and age, or count to 10.

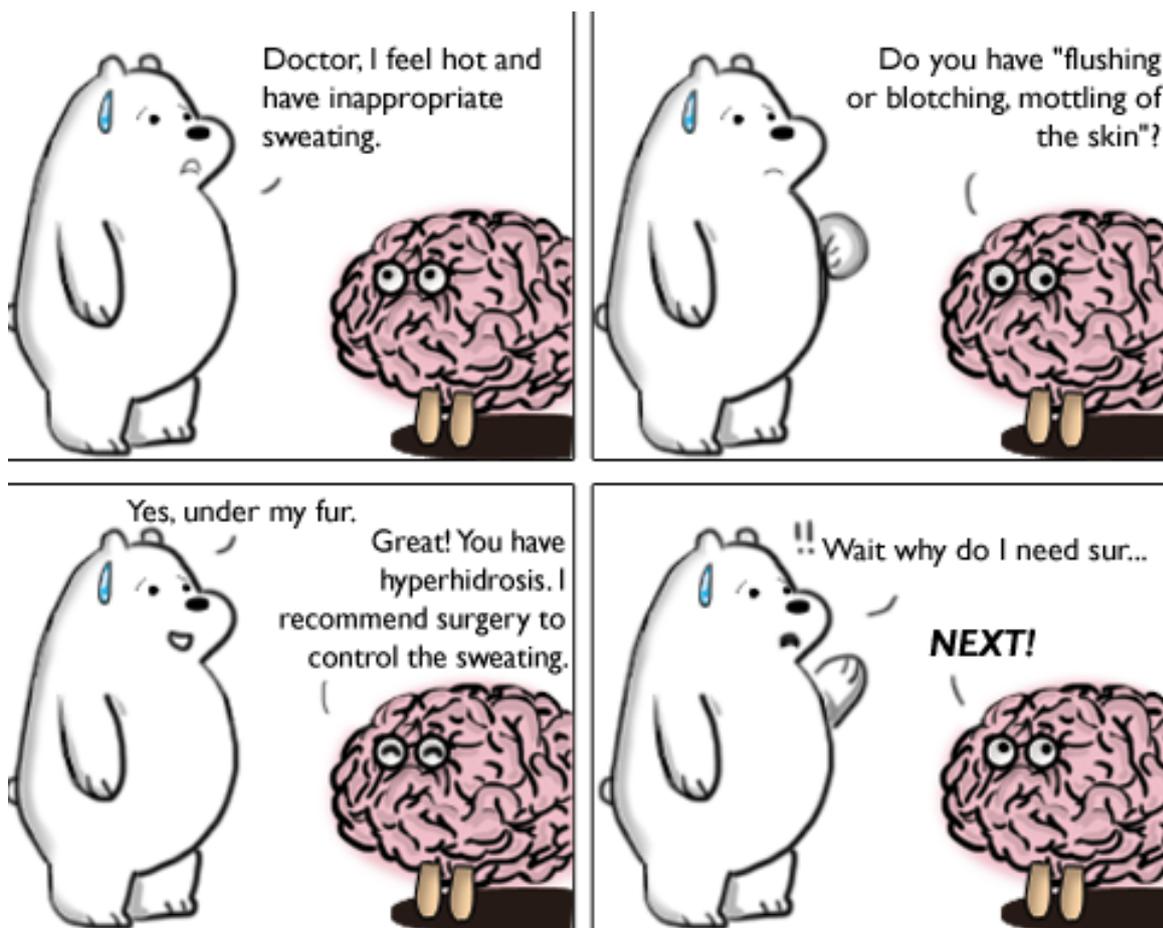
In terms of gross motor, she is highly active, able to run, tiptoe, and kick a ball. She is not yet able to jump with both legs off the ground. Her parents report that she can climb up stairs, 2 feet per step, but not yet down stairs.

In terms of social development, she is very sociable and interacts well with me. I note that she is not on pampers at the moment.

In summary, Elizabeth is a pleasant Chinese toddler with a developmental age of 2.5 years in all domains.

Surgery Long Cases

presentation scripts & Q&A



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Strategy

What are surgical long cases like? You begin with 20 min for history taking, targeted physical examination, and consolidation. Examiner 1 observes you. Examiner 2 arrives at the 21st minute and begins a 20min discussion, which is more in-depth than the medical longs. [For orthopedics: 10min + 10min, only 1 examiner] *

Examiners want you to:

- Take a good history: have a reasonable approach to the presenting complaint, be able to diagnose and consider differentials, etiology and complications. Explore how the patient has been managed, including his/her progress up to your exam day.
- Interact with the patient, not simply use a memorized template (a memorized template will inevitably lead to an examiner's comment along the lines of "appear to have little experience or interactions with patients in real life")
- Do targeted physical exam (tailor to your history), with good technique and accuracy. Please do not neglect the physical exam as this has a rather high weightage; do not take 'shortcuts' e.g. examine through clothes!
- Show professionalism (e.g. after doing per-rectal exam, please wipe the patient).
- Present your case in full (more detailed than in medicine longs), not merely repeating what the patient told you, but 'adding value' with your interpretation and synthesis.
- Discuss just about anything: e.g. your clerking, what is atypical in the history, read lab results and X-ray / CT scans, suggest management, and answer basic science questions. You are expected to tailor your answer to *this* patient. Many examiners like to probe if you have been in the wards, and will be a safe HO.

Your strategy: Surgical long cases are quite high-stakes (because of the points system), although patients are generally less complicated than the medical ones. The longer presentation and in-depth questioning can also be a challenge. Our advice is:

- *Be comfortable with clerking patients.* History-taking is not easy. You need to be logical and comprehensive (covering diagnosis, differentials, severity, etiology, complications, and management), and yet have a conversation with the patient (not do a checklist). Patients are very different from textbooks; it takes experience to make sense of what they say, to probe for information, and piece things together on the go. There is no shortcut to practice with real patients - we only became comfortable after clerking ~50 cases. As you practice, do so intentionally – blind yourself to the case, take full histories as you would in an exam and also practice presenting. Get a friend or senior to observe and give feedback. Think about whether you covered what you need to achieve, what you did well and what you can do better (we too have made 'rookie' mistakes like clerking 'dark urine' as approach to hematuria when it was obstructive jaundice). Develop your own style and template, and at MBBS stick to what you have practiced.

- *Think through cases you have seen.* The long discussion tests your overall ability to clinically assess and apply knowledge to *this* patient (from history till management). Do not simply float through your SIP posting, but ask your team the ‘why’ questions – why this diagnosis, why not the other diagnosis, why this treatment? For every patient think – if this is *your* patient, how will *you* manage? Only then, flip the file and ask *why* did the team’s approach differ from yours. Read up on what you do not know.

Refer to *Approaches to Symptoms of Disease* for how to get from presenting complaints to diagnosis. This chapter begins at diagnosis, samples possible presentations and discussion. Common things being common, they will cover about 80% of all possible surgical long cases – but there are oddballs. These cases were mostly written based on real patients we clerked, to help us learn from what we saw. Please do not take them as a gold standard to memorize - they are just an example. You would do far better to attempting the exercise yourself - think through or write out the cases you clerk in this manner, in your own style.

** Orthopaedic long cases are also covered here. These are shorter and a lot more standard (fewer permutations), but otherwise the approach and strategy is not too different.*

Troubleshooting:

- *Don’t ask the patient for his/her diagnosis at the start.* This causes you to fixate on certain ideas and clouds your thinking – at best you are highly likely to be less comprehensive in approaching the presenting complaint, neglecting to consider differentials, at worst you may be led down an entirely wrong track. You should trust in your ability to arrive at a diagnosis more than you trust the patient’s ability to give you one! It is, however, generally acceptable to ask a patient towards the end of your history, ‘what do you understand about your condition’ – you can use this to gauge patient’s understanding of disease, verify your suspicions, and probe any discrepancies.
- *Patient can’t give a good history.* Despite due diligence to list good patients, some do not turn up and are replaced by random patients grabbed from the wards. Those who have clerked many *real* patients generally handle suboptimal patients better. For the long-winded patient, guide him/her along with targeted questions, and try to stay focused on what is important. For the reserved patient, keep probing and tease out information patiently. For an unsure patient, ask direct questions that will allow you to form one clinical impression or the other. Do not depend on patients to be primed, or to give you answers (if you need to know about a specific past medical history, ask specifically, and do not assume a ‘no’ if the patient does not respond to an open-ended question on ‘what medical history do you have’). The bottom line: clerk real patients, not just your medically-trained friends!
- *Patient can’t speak English.* You are expected to clerk in English or your mother tongue (translator may not be provided if patient speaks your mother tongue). In any case, having a translator is bad news – it cuts your time in half. Therefore, please practice clerking your mother tongues (and better, in other languages and dialects).

- *I have difficulty presenting:* Remind yourself not just to report, but to interpret and to put things together – ready to present. For each case you clerk, practice presenting to someone (preferably a tutor, or a senior, if not then a batchmate). Try to present immediately after clerking, do not sit down to ‘prepare’ a presentation – you do not have this luxury in the exam.
- *I cannot read CT scans:* Start practicing early in SIP. Look at the scans of your patients, first attempting them yourself, before reading the report.
- *I am scared of difficult questions:* Taking a good history and putting things together puts you in good stead for any questions on history or diagnosis (always go back to what the patient tells you). As for investigation and management questions, focus on being a safe HO – if you do not know, admit it humbly and offer to escalate to seniors.
- *I don't have enough time:* Manage your time well, be disciplined and know when to move on. Practice, practice, and you will take a history more effortlessly. You need to do this without talking too quickly (neither patient nor examiner understands you) or appearing rushed/flustered.
- *I don't know what to expect.* Look at the senior's accounts from the hospital you are going to. Each hospital has a slightly different character. Your aim is to get the idea and flavor, not freak out at every single question that is asked.

Abdo Pain: Acute Cholecystitis

PRESENTATION

Sir, my patient Mdm Liew is a 50 year old Chinese lady with past medical history of DM, HTN, HLD, presents now with 3 days of right hypochondrium pain and low-grade fever.

Her presentation first started as right hypochondrial pain, sharp, constant, gradually becoming worse. It does not radiate to the back or shoulder and is not better on sitting forward. This was associated with low-grade fever of Tmax 37.8 degrees, and 2 episodes of nonbilious nonbloody vomiting. She is not jaundiced and does not have dark coloured urine or pale stools.

[Risk fx / etio] This is on a background of recurrent episodes of biliary colic. In the last 2 years, she has had at least 5 episodes of right hypochondrial pain which begin after a fatty meal, reach a crescendo over 3-4 hours, and then subsided. These episodes have been much milder than her presentation currently, and she was never febrile during these episodes. [Etio] She does not have history of mechanical heart valve replacement, or family history of jaundice and anemia (hemolytic anemias)

[Ddx]

- [Hepatitis] I note that she has had raw oysters last week and has not been vaccinated against viral hepatitis. She is in a stable monogamous relationship, has no hx of IV drug abuse, and has never received blood products from outside singapore.
- [PUD] Otherwise she does not have hematemesis, coffee grounds vomitus, PR bleeding, or malena. She is not on long-term NSAID or steroid therapy and has no known peptic ulcer disease.
- [Referred] On systematic review there is no chest pain / shortness of breath / palpitations to suggest a coronary event, and no cough / shortness of breath to suggest right basal pneumonia. She has no cough, shortness of breath, positive travel history or sick contact

Other than DM HTN and HLD, all of which are well controlled on medications, she does not have any other past medical or surgical history. She has no known drug allergies.

She stays with her husband and children in a HDB flat, works as a business executive and is financially well to do.

On examination, Mdm Liew is alert, comfortable. She is currently receiving IV fluids as well as IV rocephin and flagyl. She has no scleral icterus and no conjunctival pallor. Her urine bag is draining clear yellow urine. Abdomen was not distended. There is tenderness and guarding over the right hypochondrium and epigastrium. Otherwise the rest of the abdomen is soft and non-tender, no signs of peritonism. Murphy's was positive. There was no organomegaly. Bowel sounds were present. Cough impulse over bilateral groins were negative. Breath sounds were clear and equal bilaterally. Heart sounds were dual, no murmurs, pulse was regular at 95. I would like to complete my examination by doing a digital rectal examination looking for PR bleed or melena.

In summary my patient is a 50/Chinese/lady with recurrent episodes of biliary colic, now presenting with right hypochondrial pain and low-grade fever of 2 days duration, with a positive murphy's sign. My primary diagnosis is cholecystitis and my differentials include biliary colic, hepatitis, pancreatitis, peptic ulcer disease, and dengue.

QUESTIONS

How would you investigate?

Sir, I need to investigate to confirm my diagnosis, rule out differentials and find the etiology.

Initial bloods include

- FBC for raised TW
- LFT which should be normal or at most mildly raised. If severe cholestatic LFTs, I will suspect cholangitis. If hepatocellular picture I will suspect hepatitis.
- UECr for prerenal AKI since patient has been vomiting / to guide fluid therapy
- Blood cultures if the patient looks septic.
- CRP, procalcitonin

For differentials, I will do

- Amylase, lipase to look for pancreatitis (if present, to do full glasgow bloods).
- Hepatitis viral serologies for viral hepatitis
- Cardiac enzymes, ECG - to rule out AMI.
- CXR - looking for basal lobe consolidation, rule out air under diaphragm

Imaging modalities include

- US HBs for evidence of cholecystitis
- CT abdomen for the same especially overnight where US cannot be easily obtained.

Can you please read this ultrasound?

Sir I note there is a thickened gallbladder wall with intraluminal gallstones casting a posterior acoustic shadow. There is pericholecystic fluid and I will like to ask if there is positive sonographic murphy sign. I will also like to look at the common bile duct which I expect to be not dilated.

You are the HO on call. How would you manage?

Sir, my main goals would be to (1) Resuscitate the patient according to ABC (2) Confirm the diagnosis and rule out differentials (3) Initiate treatment and plan for definitive treatment.

1. Resuscitate the patient according to

- A Assess airway
- B Ensure breathing, saturation
- C Take vitals
 - Set IV plugs
 - IV fluid rehydration
 - Keep NBM
 - Transfer GW or HD
 - Chart: strict I/O, Q2h paras
 - Serial monitoring for complications

2. Confirm diagnosis and rule out differentials

As discussed above.

3. Initiate treatment

Empiric IV antibiotics with gram negative and anaerobic coverage - 3rd gen cephalosporin like ceftriaxone, and metronidazole.

4. Definitive treatment

> Laparoscopic KIV open cholecystectomy

When would you perform cholecystectomy?

Sir I would prefer early cholecystectomy within first 72h as opposed to interval cholecystectomy because there is no increased risk of bile duct injury, and we do not risk the patient having a recurrence while waiting for interval cholecystectomy

If patient too sick for op then how?

Alternative is percutaneous cholecystostomy

- Involves percutaneous catheter placement in the gallbladder lumen under imaging guidance (alternative to surgical cholecystectomy)
- Indications: moribund patients who are not fit for surgery or when early surgery is difficult due to extensive inflammation
- Drains the gallbladder and alleviates the inflammation (resolves acute episode)
- Followed by elective cholecystectomy 4-6 weeks later

Please counsel Mdm Liew on cholecystectomy.

Mdm Liew, I am Dr _____. I'm going to explain to you (1) your current condition (indication) (2) What is a cholecystectomy and why we recommend it (3) risks and complications of cholecystectomy (4) risk of not going for cholecystectomy and alternative. Do you have any other questions or concerns you would like me to address along the way? If not, still feel free to stop me any time along the way to clarify anything you don't understand.

**Recommend drawing it out!*

(1) Your current condition

You have inflammation of your gallbladder. This is likely due to irritation and infection of the stones that your gallbladder had formed. Thankfully none of the stones have dropped out into the bile ducts and caused any blockage.

(2) Cholecystectomy

So the best solution is to just remove the whole gallbladder with the stones inside. This will both remove the infection and also prevent the possibility of any more such infections in the future! The operation is called cholecystectomy. It will be done under general anesthesia so you do not feel any pain. We normally try to do this by a camera through keyhole surgery, but that sometimes does not work so we need to make it open through a small cut (draw).

(3) Risks and complications of cholecystectomy

(a) Risk from general anesthesia

Can get heart attack and stroke, but there is a lower risk of this as you have no previous heart or blood vessel problems

(b) Risk from procedure itself

*If lap, risk of conversion to open

Injury to common bile duct, injury to cystic artery, injury to bowel

Spillage of bile → peritonitis, sepsis

Infection, hemorrhage

(4) Risk of not going for cholecystectomy and alternatives

The alternative to cholecystectomy is percutaneous cholecystostomy, poking a hole through the skin into the GB to drain it. However this is just a temporary measure and once the tube is removed without removing the gallbladder, there is a risk that this can happen again. Furthermore, the stones can drop out and cause cholangitis or pancreatitis.

The last option is to not do anything, but other problems may arise: the GB can become gangrenous and burst, the stones can burrow a hole through to the intestine and cause blockage of the intestine, mucous or pus can accumulate there, the infection can spread to the whole body and cause sepsis & death, and in rare cases GB cancer can develop after a long time.

So let's say the OT very full, and we have not been able to do the cholecystectomy on the same day. The next day you see the patient. It is now day 4 of illness, the patient is more tender in the RHC and the temperature is going up. What is happening?

Sir I am worried about local complications of cholecystitis such as mucocele → empyema → gangrenous → perforation. I think the patient needs early cholecystectomy.

Start suspecting these when there are signs of failure of conservative mx (peritonism, non-resolving fever/pain)

OK what are the other complications of cholecystitis?

- Gallstone complications: cholecystoenteric fistula → gallstone ileus
- Long term: GB cancer.

Abdo Pain: Acute Pancreatitis

PRESENTATION

Sir, my patient Mdm Liew is a 50 year old Chinese lady with past medical history of DM, HTN, HLD, presents now with acute epigastric pain of 2 days.

Her epigastric pain was sudden in onset, constant, sharp and excruciating with radiation to the back, slightly better when sitting up and leaning forward. This was associated with a low grade fever of 37.8 no chills no rigors, as well as nausea and 2 episodes non-billous non-bloody vomiting over the same duration.

[Ddx]

- [Acute cholangitis] She did not have jaundice, tea coloured urine, pale stools.
- [PUD] There is no hematemesis, coffee ground vomitus, PR bleed, melena, nor does she have risk factors of peptic ulcer disease such as long term NSAID or steroid use, known peptic ulcers or H. pylori.
- [Aortic dissection] No migratory pain from chest to abdomen, no suggestion of distal embolization.
- [DKA] She has no polyuria, polydipsia, no intercurrent illness or non-compliance to medications that could precipitate DKA.
- [AMI] She did not have crushing chest pain with shortness of breath, palpitations, diaphoresis.

As for underlying etiology,

- [Gallstone disease] She has longstanding post-meal dyspepsia symptoms.
- [Instrumentation] She has never had an OGD or ERCP.
- [Alcohol] She drinks only occasionally and in small quantities
- [Metabolic] She does not have past medical hx of hypercalcemia or parathyroid problems. She is however hyperlipidemic and may have high triglycerides.
- [Drugs] There is no long term use of TCM, corticosteroids, NSAIDs, loop diuretics
- [Viral] No recent history of bilateral jaw pain/swelling (mumps)

Her past medical history is that of well-controlled DM, HTN, HLD. Her latest HbA1c is 6.0 and compliance is good. She has no past surgical hx and has no drug allergies.

Socially, she stays with her husband and children in a HDB flat, works as a business executive and is financially well to do. She does not smoke.

On examination, Mdm Liew is alert and sitting forward, not in respiratory distress. She is currently receiving IV fluids. She is not jaundiced, has no scleral icterus or conjunctival pallor, and her urine bag is draining clear yellow urine. I do not note any abdominal scars. Abdomen was not distended, soft and non-tender on palpation with no guarding or rebound. Murphy's was negative. There was no organomegaly. No pulsatile expansile mass. Bowel sounds were present. Cough impulse over bilateral groins were negative. Breath sounds were clear and equal bilaterally. Heart sounds were dual, no murmurs, pulse was regular at 95. I would like to complete my examination by doing a digital rectal examination looking for PR bleed or melena.

In summary my patient is a 50/F/Chinese who presents with epigastric pain radiating to the back on a background hx of longstanding post-meal dyspepsia symptoms. My top differential is acute gallstone pancreatitis. My ddx include cholecystitis, and I want to make sure it is not cholangitis.

QUESTIONS

You are the HO on call. How would you manage this lady?

Sir, on call my initial goal will be (1) resuscitate the patient, (2) confirm my diagnosis and rule out differentials, (3) stratify severity to determine disposition and prognosticate and to look out for complications, and (4) identify the etiology so that this can be treated.

(1) Resus

- A Assess airway
- B Ensure breathing, saturation
 - Supplemental oxygen
 - Be wary of systemic inflammatory response syndrome
- C Take vitals
 - 2 large bore IV plugs
 - Aggressive fluid rehydration - 2 pint normal saline fast
 - Keep NBM
 - Insert IDC
 - Transfer GW (if mild) or HD/ICU (if severe)
 - Chart: strict I/O, Q1h paras + SpO2 + hypocount
 - Also ensure patient has adequate analgesia (tramadol, pethidine)

I would then do investigations to:

- (2) Confirm diagnosis and rule out differentials
- (3) Stratify severity using Glasgow's scoring system
- (4) Determine etiology
- (5) Prepare for ERCP

Blood work:

- FBC - raised TW (Glasgow)
- RP - raised urea (Glasgow) and Cr in pre-renal AKI (SIRS), raised glucose (Glasgow), rule out DKA (low bicarb, high glucose)
- Ca/Mg/Phos - hypocalcemia (Glasgow)
- LFT - looking for cholestatic picture (?choledocholithiasis, cholangitis), albumin and AST/ALT and LDH (Glasgow)
- Amylase Lipase -- *normal range <100, <140*
- ABG - PaO₂ (Glasgow)
- CRP - to trend
- Cardiac enzymes ECG - to rule out AMI
- GXM PT/PTT (ERCP)
- Dengue serology - since we are in the midst of an outbreak.

Imaging:

- Ultrasound for evidence of cholelithiasis, rule out dilated CBD, look for cholecystitis
- Also do CXR - looking for whiteout (ARDS) or air under diaphragm
- Supine AXR - for sentinel loop (ileus secondary to para-pancreatic inflammation)
- Acutely there is no role for CTAP, this is not required for diagnosis of pancreatitis in the presence of a typical hx and raised amylase/lipase, and I don't expect complications like necrosis so early. In practice however I often see CT abdo being ordered for radiological confirmation and to rule out other ddx.

What are the complications of acute pancreatitis?

The complications of pancreatitis can be divided into local and systemic complications.

Local complications include

- Pseudocyst formation which can cause mass effect (GOO, obstructive jaundice), pain, persistently increased amylase.
- Pancreatic necrosis which is initially sterile but can become secondarily infected (pain, sepsis). *CT scan look for gas > necrosis. If suspect infection, aspirate and culture and cover with Abx. If not infected discontinue Abx. Infected necrosis may also require surgical necrosectomy.*
- Pancreatic pseudoaneurysm
- Functional complications like exocrine or endocrine deficiency.

Systemic complications

- Organ failure e.g. ARDS, AKI, DIVC
- Hypocalcemia
- Pancreatic ascites

You may be asked this content in various permutations... e.g.

- When rounding the patient the next morning, you notice that he has desaturated. CXR shows bilateral infiltrates... [ARDS]
- 1 week later the patient complains of abdominal pain, and develops a high fever. Amylase is still elevated... [infected necrosis]

What is the definitive treatment for Mdm Liew?

- ERCP within 48-72h, especially if severe, evidence of ductal stones, cholangitis, or not responding.
- Cholecystectomy (same admission)

No ductal stones were seen on the CTAP, just some in the GB. No dilated ducts seen. Pancreatitis improves with supportive treatment. Please counsel Mdm Liew on cholecystectomy.

Mdm Liew, I am Dr Tsang. I'm going to explain to you (1) your current condition (indication) (2) What is a cholecystectomy and why we recommend it (3) risks and complications of cholecystectomy (4) risk of not going for cholecystectomy and alternative. Do you have any other questions or concerns you would like me to address along the way? If not, still feel free to stop me any time along the way to clarify anything you don't understand.

*I recommend drawing the HBS system in this case!

(1) Your current condition

You have inflammation of your pancreas, which is a gland you have in your body that helps produce juices for digestion and also helps control blood sugar. This inflammation happened because a stone inside your gallbladder dropped out and blocked the pancreas. The pancreas juices cannot flow out, get stuck inside the pancreas and cause irritation and inflammation, sometimes the tissue will die and form fluid collections. Currently your pancreas inflammation has improved. The stone causing blockage has passed out by itself OR has been removed by the scope (ERCP).

(2) Cholecystectomy

Now, the next step is to remove the source of the stones - the gallbladder. The name of the operation for this is 'cholecystectomy'. This can be done laparoscopically (if ERCP already done) or open (if ERCP was not done and need to do CBDE). We will leave a drain from the surgery area coming out of your skin, but only for a few days then we will take it out. Therefore after the operation you should have a scar like this and a drain coming out (draw!).

If CBDE: We will also need to open the bile ducts to make sure there are no more stones left behind. After that we will close back the bile duct and leave a another drain from the bile duct.

(3) Risks and complications of cholecystectomy

(a) Risk from general anesthesia

Can get heart attack and stroke, but there is a lower risk of this as you have no previous heart or blood vessel problems

(b) Risk from procedure itself

*If lap, risk of conversion to open

Injury to common bile duct, injury to cystic artery, injury to bowel

Spillage of bile → peritonitis, sepsis

Infection, hemorrhage

Steatorrhea on eating fatty food

(4) Risk of not going for cholecystectomy and alternatives

The alternative to cholecystectomy is conservative management.

However the risk if you don't remove the gallbladder is that it can happen again. The source of gallstones will still be there... stones can still drop out and cause pancreatitis.

There is a 40% of recurrence within 6 weeks.

Abdo Pain: Aortic Aneurysm

PRESENTATION

Mr Muthu is a 69 year old gentleman who is retired. He is a vasculopath with HTN HLD IHD, and previous cardiac bypass. Other medical problems include BPH.

He now presents with a 4 day history of epigastric pain radiating to the back. He describes this as multiple episodes of dull aching pain, pain score 5/10, each episode lasting 30 minutes and then resolving. He has not had any prior episodes

[Ddx]

- [Spine]: Pain is not related to spine flexion or extension, and
- [Biliary: cholelithiasis, cholangitis, pancreatitis]: is not relieved on leaning forward. He has no jaundice, tea coloured urine, or fever. He has no known gallstone disease, past episodes of fatty dyspepsia, or recent alcohol binge.
- [Peptic ulcer / Dyspepsia]: There are no gastrointestinal symptoms of vomiting or diarrhoea, and no melena, hematemesis, or per-rectal bleeding.
- [Urolithiasis]: There is no hematuria and no past episodes of loin to groin colic.
- [AMI]: There is also no diaphoresis, palpitations, dyspnoea,
- [DKA]: new-onset polyuria/polydipsia,
- [Dengue]: and does not stay in a dengue cluster.

He was first seen in primary care and treated as for gastritis with proton pump inhibitors, but did not improve. He then came to the accident and emergency, where he was diagnosed with abdominal aortic aneurysm. He has received endovascular aneurysm repair and is post-op day 5, recovering well in general ward.

In terms of complications, he was not hemodynamically unstable when admitted, and has no chest pain / shortness of breath (backward dissection / AR), no giddiness / blackouts / weakness / facial droop (dissection involving carotids). He has no sudden severe abdominal pain (rupture), no oliguria (renal artery involvement), no sudden leg gangrene (distal embolization).

In terms of past medical history, he is a vasculopath with ischaemic heart disease requiring coronary artery bypass 10 years ago, and has not had anginal symptoms since. He does not know his latest ejection fraction. He has hypertension and hyperlipidemia, for which he is on multiple medicines. He misses his medications about twice a week and latest clinic blood

pressure was systolic 160. He is an ex smoker of 30 pack years and has quit smoking since his bypass.

His is functionally good, able to walk 10 bus stops.

Socially, he is a retiree. He stays with his wife in a condo and they do not have any financial concerns.

On examination, he is alert, comfortable, not on any external intervention at present. I note an old midline sternotomy with old saphenous vein graft scar, likely from old coronary bypass. I also note fresh surgical scars over bilateral femoral arteries, both of which are healing well with no hematoma. His abdomen is soft non-tender, there is a mass palpable deep in the epigastrium - this is a firm 3cm x 2cm mass, not expansile or pulsatile, regular margins and not nodular; it is likely the repaired AAA. On examination of the lower limbs, there were no gangrene, ulcers, arterial skin changes. Pulses were well felt and capillary refill time was < 2 seconds. A brief cardiac examination was unremarkable.

In summary, Mr Muthu is a 69 year old vasculopath presenting with symptomatic aortic aneurysm status post endovascular repair.

QUESTIONS

At his initial presentation, what would be your differentials for his epigastric pain?

See above; remember to cast your net wide.

How do AAA present?

- Asymptomatic, detected incidentally on (hopefully) abdominal palpation or (sadly) CT scan for some other reason
- Symptomatic but unruptured: pain due to local compression, rapid expansion; distal embolism causing trash feet
- Rupture: a catastrophic event with hypotension, hemoperitoneum and rapid demise

OK so you are the HO on call, what will you do?

Sir, my initial goals are to (1) resuscitate the patient, (2) establish the diagnosis and consider differentials (as AAA may be incidental and pain may be due to another pathology, and (3) prepare the patient for urgent intervention.

Resus A Assess airway
 B Ensure breathing, saturation
 C Take vitals
 Set 2 large bore IV plugs
 Start fluids

Bloods FBC - Hb drop, Plt
 PT/PTT
 GXM
 UECr - for contrast scan
 LFT - ddx hepatobiliary pathology
 Amylase, lipase - ddx pancreatic pathology
 Glucose, dengue duo
 Troponins

ECG

Imaging CT aortogram (if unstable - bedside ultrasound).
 Looking for to confirm AAA, exclude ddx, identify size and branches involved
 Look for dissection or contained leak/rupture and plan for stenting

Initial Mx To HD/ICU with hourly monitoring and strict i/o
 Insert IDC
 Keep NBM
 Analgesia
 (Stop antiplatelet)

CT aortogram shows a 5cm aneurysm. What is the management?

Sir, this patient has a symptomatic aneurysm and needs to be repaired urgently (regardless of size). I would opt for endovascular aneurysm repair over open repair in view of lower perioperative risk, assuming endovascular repair is anatomically feasible (These days even suprarenal AAA can be repaired endovascularly).

What complications will you counsel the patient on?

Aortic surgery is not low risk.

- Anaesthetic/general risks include myocardial infarction, stroke, and death.
- Surgical risks can be divided into aortic and systemic organ risks.
 - Aortic risks include endoleak, stent migration, stent occlusion, stent infection.
 - Organ risks include *paralysis* (spinal artery involvement), *kidney injury* (renal artery involvement or contrast nephropathy), bowel ischaemia, trash feet from distal *embolization*.
- Transfusion risks: hemolytic transfusion reaction, hepatitis/HIV, and ARDS.
- If endovascular repair, post-op complications also include stent migration, stent infection, endovascular leak

Let's say he has the same 5cm aneurysm but this was asymptomatic and detected on CTAP performed for some other reason. What will you do?

Sir, for an asymptomatic AAA, my threshold to repair is 5.5cm (UK small aneurysm trial). At 5cm therefore I will follow up with ultrasound in 3-6 months with a view to repair when the aneurysm reaches 5.5cm.

Abdo Pain: Diverticulitis

PRESENTATION

Mdm Verasamy is 35 year old Indian lady with a 2 day history of right iliac fossa pain with fever and vomiting.

With regards to her abdominal pain, this started as a mild ache 2 days ago and got worse over the course of the day, reaching a pain score of 5/10 at presentation to A&E. She describes it as a constant dull pain localized in the right iliac fossa. There is no prior migration from umbilicus to right iliac fossa [appendicitis], and no radiation from loin to groin [urolithiasis], to the back [pancreatitis or AAA], or anywhere else.

This was associated with fever, also of 2 days' duration; she did not measure her temperature at home. She also had 3 episodes of non-bloody non-bilious vomiting over these 2 days. There is no constipation, diarrhoea, or jaundice [cholecystitis]. There was also no pyuria [pyelonephritis], hematuria [urolithiasis], no per-vaginal bleed [ectopic pregnancy] or discharge [PID], or back pain [spine dx]. She has been well until these 2 days, with neither prior episodes of abdominal pain, nor any loss of weight or appetite. *Her last menstrual period was last week (remember to ask!).*

Since admission she has received IV antibiotics with symptomatic improvement. She has also undergone a computed tomography scan, but has not had any interventional procedures or surgeries.

In terms of past medical history, I note that she has childhood asthma for which she no longer requires follow up. She has also had one termination of pregnancy 10 years ago. Otherwise there is no past medical history, no past surgeries and no drug allergy. She does not have any family history of malignancy or inflammatory bowel disease.

Socially, she is single, unmarried, and is sexually active with multiple partners without use of condoms. She works as an advertising executive and is financially comfortable. She stays in a 4 room flat with her parents and does not smoke or drink.

On examination, she is alert and comfortable, receiving IV ceftriaxone and metronidazole via an IV plug which is clean. Peripherally there is no cachexia, jaundice, or pallor. On inspection of the abdomen, there is no scar, no distension. She has mild tenderness in the right iliac fossa with no guarding or rebound, and the abdomen is otherwise soft. There is no RIF mass. There is no flank tenderness and no positive Murphy. Renal punch is negative.

I will like to complete my examination by doing digital rectal examination to rule out a bleed, and doing per-vaginal examination looking for cervical excitation and cervical discharge.

In summary my patient is a 35 year old Indian lady with 2 days of right iliac fossa pain and fever. She is not peritonitic and has clinically improved after 2 days of IV antibiotics.

QUESTIONS

What are the differentials you will like to consider for her initial presentation?

Sir, I will like to consider gastrointestinal, urological, gynaecological, and miscellaneous causes (cast net broad as presentation nonspecific).

- The gastrointestinal causes include diverticulitis and appendicitis, although there is no classic migratory pain.
- I must also consider gynaecological causes especially due to her sexual activity with multiple partners, and I feel that her reported menses does not exclude ectopic pregnancy.
- In terms of urological causes, I am considering pyelonephritis or urolithiasis complicated by pyonephrosis, although my patient has no hematuria or loin to groin pain to suggest urolithiasis.

How will you investigate the patient?

Sir, my investigations are directed to (1) confirm dx, (2) look for cx, and (3) guide further mx.

- At the bedside I want a urine pregnancy test, capillary blood glucose, and urine dipstick.
- I will then send initial blood work such as FBC looking for raised TW in infection, UECr for hypochloreaemic metabolic alkalosis due to vomiting, LFT to rule out hepatobiliary causes, amylase to rule out pancreatitis, CRP and cultures since she is febrile, dengue duo just to be sure, and GXM and PT/PTT in case the patient needs interventional procedure.
- I will send urine for formed element microscopy looking for evidence of hematuria.
- If vaginal examination shows discharge I will also like to send for culture.
- I will order erect chest X ray and KUB, looking for urolithiasis, and seeking to exclude air under diaphragm or Rigler's sign in perforation.
- I will discuss with my senior and the patient will likely eventually need a CTAP.

OK here is the CTAP please read -

Sir this is a slice of my patient's CTAP taken yesterday morning. I note that there is fat stranding surrounding the cecum, with thickening of the colonic wall and multiple diverticulae. I also note a small hyperdense collection adjacent to one of the diverticulae and I wonder if this is an abscess. Otherwise I do not note any free air in this cut. Overall this scan confirms my impression of diverticulitis, with a paracolic abscess.

Can you explain how colonic diverticuli form?

Diverticuli are outpouchings of the colonic wall at a point of weakness where the vessels penetrate the circular muscle layer of the colon. It arises due to increased intraluminal pressure especially in patients with chronic constipation and low fibre diet.

What is diverticular disease?

Outpouchings in the colonic mucosa and submucosa through weaknesses in the muscles layers where colonic arteries penetrate the colonic wall.

How can diverticular disease present?

Diverticular disease can be asymptomatic, present as lower gastrointestinal bleeding, or present as diverticulitis. Diverticulitis may be simple with just abdominal pain which is classically LIF in textbooks but more commonly RIF in asians. Alternatively diverticulitis can be complicated and present with perforation, palpable mass due to abscess formation, or fistulation causing pneumaturia, fecaluria, per-vaginal feculent discharge. Chronic diverticulitis can form benign strictures, causing intestinal obstruction

OK so how would you manage the patient?

Sir, my patient has Hinchey 1 diverticulitis (pericolic perforation) so I would initially attempt conservative management most importantly with IV antibiotics. Supportive measures include hydration, keeping patient NBM, and analgesia. If she fails to improve she may require percutaneous drainage by interventional radiology. I could consider offering an interval colonoscopy to rule out underlying colorectal malignancy.

You give your patient IV antibiotics. On call you are called to see her because of severe abdominal pain. When you palpate the abdomen it is rigid. How now brown cow?

Sir, I am worried about perforated diverticulitis. I will attend to the patient immediately and resuscitate the patient. I will inform my senior and obtain a stat erect chest X ray unless the patient is crashing. The patient needs to go for laparotomy with surgical resection for example right hemicolectomy with defunctioning ileostomy.

(If you know the diverticulum is right sided, please don't say Hartmann's!)

Abdo Pain: Urolithiasis

PRESENTATION

Sir, my patient is Ms Jolene, a 33 year old Chinese lady with hypercalcemia from an underlying parathyroid tumor, presenting with recurrent urolithiasis and pyelonephritis, and currently admitted for pyonephrosis.

May I begin with Ms Jolene's significant past medical history. In the past 4 years, she has recurrent pyelonephritis with fever and loin pain, each time treated by GPs with oral Abx. She has also had multiple episodes of loin to groin pain suggestive of ureteric colic, but has never had loss of weight or appetite in all these years.

Five days ago, she presented yet again with loin pain, fever, chills and nausea of one day's duration. No other storage symptoms such as dysuria, frequency, urgency, or urge incontinence; no voiding symptoms; and no hematuria. She was started on IV antibiotics with little improvement. In fact on the 3rd day of admission her loin pain worsened. CTAP was done, which showed pyonephrosis with ureteric stones. Right percutaneous nephrostomy was done, after which her symptoms improved.

In terms of the etiology of her urolithiasis, I note that she was found to be hypercalcemic with elevated parathyroid hormone levels. Sestamibi scan found a parathyroid adenoma although there was no palpable goitre. She is otherwise not on any medications that could cause hypercalciuria. In terms of other etiologies, she has no known hx of gout or joint pains, and drinks at least 2 litres of water each day. She is not known to have congenital structural abnormalities of the urinary tract. She does not have any family history of parathyroid tumours, urolithiasis (RTA), urological cancers, or other endocrine tumors (MEN).

I understand that she has recently underwent parathyroidectomy for her parathyroid adenoma.

In terms of social, functional and financial history,

- She does not smoke or drink
- She has been able to work as per normal in her job as a magazine editor, as it is a mostly desk bound job
- She stays with her husband in a HDB flat in tampines, does not have any children
- Financially okay as she is covered by insurance
- The only concern/struggle she has is with the restrictions that come with the new low purine diet as per dietician recommendation, to reduce risk of kidney stones

On examination, Ms Jolene was alert and comfortable. She is still receiving antibiotics. She does not have an indwelling catheter. I note a horizontal scar in the midline of the neck over the level of the thyroid cartilage. The right percutaneous nephrostomy is draining hemopurulent fluid, the collecting bag currently contains about 200ml of fluid. Otherwise I do not note any other scars, her abdomen is soft and non-tender. The left kidney was renal punch negative and not ballotable. I did not touch the right side in view of the right PCN.

In summary Ms Jolene is a 33 year old Chinese lady with hypercalcemia from an underlying parathyroid tumor, presenting with recurrent urolithiasis and pyelonephritis. She is currently admitted for pyonephrosis and is now status post parathyroidectomy.

What are the risk factors for urolithiasis?

Sir, risk factors include:

- Dehydration
- Diet: high protein and salt intake, high purine diet.
- Drugs: probenecid, loop diuretics, antacids, salicylate acid, indinavir, chemotherapy
- Systemic diseases: Crohn's disease, gout, renal tubular acidosis, patients on chemo for haem malignancies, hyperparathyroidism, inborn errors of purine metabolism
- Infection with urea splitting organism (struvite stone)
- Biodata: Male to female 3:1, age 40-60

Assuming that you are the HO when she first presented, how would you manage her?

Sir, pyonephrosis is a urological emergency and requires drainage as soon as possible. I would see her immediately, get a set of vitals and assess her airway breathing and circulation, set an IV plug and start IV fluid resuscitation. I would then send off the following basic investigations:

- FBC for raised total whites
- RP for raised Cr (both pre and post renal AKI), electrolyte abnormalities
- Ca/Mg/Phos, for hypercalcemia
- CRP for trending
- Blood cultures, to guide antibiotic treatment
- ABG and lactate if she is sick
- PT/PTT and GXM as she is going for an invasive procedure
- UFEME, Urine gram stain and cultures
- XR KUB looking for radio-opaque stones
- Subsequent CT KUB or CT urogram looking for pyonephrosis.

I would then start her on empiric broad spectrum IV antibiotics, insert an IDC and put her on Q1H paras plus strict IO monitoring, transfer her to a HD ward, escalate to my senior and make an urgent urology referral once the CT KUB or urogram confirms pyonephrosis. The patient will need percutaneous drainage.

Note: the diagnosis of pyonephrosis is seldom as apparent from the start! Many patients come in with what appear to be pyelonephritis - think of pyonephritis if they do not get better with Abx (worsening pain, more septic, not responding), have a ballotable kidney. Have a low threshold to scan if the patient is very sick. Be suspicious of pyonephrosis if the patient is known to have urolithiasis.

Outline the definitive treatment of urolithiasis.

My patient has pyonephrosis and will require emergent drainage via percutaneous nephrostomy tube. Thereafter her urolithiasis will need to be managed for example with percutaneous nephrolithotomy or ESWL.

General principle:

Treatment depends on the size and location of stones.

- Conservative for stones <5mm (70% spontaneously expelled):
 - Analgesia, high fluid intake, medical expulsive therapy with alpha blocker
 - If uric acid stone → alkalinize urine.
- Intervention if conservative is unlikely to succeed (larger than 5mm), fails, recurrent, or complicated (e.g. pyonephrosis, hydronephrosis).
 - Extracorporeal shockwave lithotripsy -- for renal and upper ureter stone
 - Ureteroscopy plus laser lithotripsy -- for ureteric stones
 - Percutaneous nephrolithotomy -- for renal stones especially when PCN inserted already.
 - If worried that stone fragments may cause obstruction, add a means to ensure drainage e.g. double J stent.
- Address underlying cause of stone formation: e.g. drink more water, decrease intake of oxalate rich food (e.g. spinach) and purine rich food (alcohol, red meat), normal calcium diet

How do you know when you can remove the PCN?

After successful decompression and drainage, the next step is evaluate the patency of the collecting system. This is usually done with an antegrade urogram, where contrast is injected through the PCN. It is imperative to ensure there is no obstruction before removing the drain, otherwise the patient will leak from the nephrostomy site or the pyonephrosis will recur.

- Therefore if there is free flow of contrast, → can pull out PCN
- If still obstructed/narrowing seen (dDx stone vs benign stricture vs malignant stricture)
 - insert DJ stent via ureteroscopy and ensure free flow before pulling out PCN
 - With DJ stent in place, you can also perform ESWL on any kidney/ureteric stones without worrying about obstructing the system again.
 - DJ stents should be left in only about 6-8 weeks, no more than 3 months, otherwise stent crystallization may occur. Crystallized stents are extremely hard to remove.

How would you evaluate her kidney function?

- Renal panel - Cr, eGFR >> evaluate both kidney overall function, if there is only one working kidney Cr can still be normal. So need to evaluate differential function.
- A MAG3 renogram would be the investigation of choice. This is a dynamic radioisotope study that allows me to evaluate the differential function of right vs left kidney, by looking at how well each kidney excretes the radioisotope tracer. In normal individuals, differential kidney function should be ~50% on each side, out of 100% for both kidneys combined. If one kidney has <15% total renal function, it is not worth saving that kidney.
- What about DMSA? This is a static scan that can allow visualization of scarring. But does not give information about function.

BGIT, Lower: Diverticular Bleed

PRESENTATION

Mr Ng is a 57 year old Chinese gentleman who presents with a 1-day hx of painless per-rectal bleeding.

This is his first episode of gastrointestinal bleeding. He first noticed dark red blood mixed with stool yesterday morning, followed a few hours later by one episode of large amount of dark red blood with no stool. Since admission yesterday evening he has had 5 more episodes, each time passing ~1 cup amount of fresh red blood with no stool. All of these episodes were painless.

[Ddx]

- [UBGIT] His stools are not malenic and he has no hematemesis or melena to suggest bleeding from the upper part of the GIT. No epigastric pain, long term use of NSAIDs or steroids, no personal history of chronic liver disease or peptic ulcer disease.
- [CA colon] There is no recent change in bowel habits (baseline 1x a day), no change in stool caliber, tenesmus, recent loss of appetite or weight.
- [Haemorrhoids] He does not have any history of haemorrhoids and does not notice any lumps or sensation of rectal prolapse while defecating.
- Not associated with any pain on defecation, does not have history of constipation

In terms of complications, he was hemodynamically stable on admission and not in haemorrhagic shock. I do not note any symptoms of anemia such as chest pain, SOB, palpitations, giddiness/syncope.

In terms of past medical history, he has ischaemic heart disease of NYHA class 1 on aspirin; he has never required percutaneous angioplasty or stent (*if angioplasty or stent, must know when, so can decide whether to hold aspirin*). He has well controlled hypertension and hyperlipidaemia on medications. He has never had a colonoscopy but a fecal occult blood test done last year was negative. There are no known drug allergies.

In terms of family history there is no suggestion of a family history of colorectal or colorectal associated malignancies.

Social history wise, he is a high-functioning engineer. He stays with wife and 2 doctor children in a HDB mansionette. He does not smoke or drink.

On examination, Mr Ng was alert and comfortable. He does not have any surgical scars. There was no conjunctival pallor. Abdomen was soft and non-tender, no organomegaly no masses. I would offer to do a digital rectal examination *with proctoscopy*, looking for piles or colonic masses as well as the nature of the blood.

In summary, my patient is a 57 year old gentleman with 1 day of painless per rectal bleed. He is hemodynamically stable and his bleed has stopped spontaneously

QUESTIONS

What are the differentials for lower BGIT?

- Sir, my patient is most likely to have a diverticular bleed given his history of dark blood mixed with stool followed by fresh blood passed without stool.
- I need to rule out massive upper gastrointestinal bleed although I do not note any risk factors suggestive of variceal or peptic ulcer bleed.
- I will also need to rule out colorectal cancer although these tend to present as chronic bleed, mixed with stools, and iron deficiency anaemia rather than as an acute episode of per rectal bleeding.
- Another differential would be hemorrhoids, however this is classically bright red blood after defecation, spraying or dripping blood staining whole toilet bowl and on wiping with toilet paper.
- Other differentials include colitis which may be inflammatory (Crohn's) or infective (parasites), however these tend to be bloody diarrhoea with abdominal pain, which is not what my patient has
- Angiodysplasia is another relatively rare cause that is difficult to elicit on history and is usually picked up incidentally on scopes (can be torrential)
- Coagulopathy (I would not attribute a bleed to coagulopathy alone unless everything else is ruled out!)

If you were the house officer on call, how would you investigate and manage this patient in the acute setting?

I would

- Resuscitate according to airway breathing and circulation
- Get a set of vitals
- Set 2 large bore IV cannulae and commence IV fluid resuscitation
- Keep the patient nil by mouth and start IV omeprazole/pantoprazole
- Suspend his aspirin for now
- Catheterize.
- Send to high dependency with hourly parameters, strict I/O monitoring and stool charting

Then I would send off some basic investigations:

- Full blood count: looking at Hb for anemia and Plt count
- Renal panel: looking at the urea to creatinine ratio that would suggest bleeding from the upper GIT
- PT/PTT: for any bleeding diathesis
- GXM: in anticipation for any blood products the patient may require
- Lactate, ABG
- Cardiac enzymes and ECG: looking out for type 2 MI, especially in view of the patient's history of ischemic heart disease
- Chest XR looking for free air under diaphragm

If patient develops symptoms of anemia or has a Hb below the requirement for scopes (9), then I would call for packed red cells

I would then counsel the patient and take consent for OGD to rule out upper GIT bleed and an interval colonoscopy to confirm the presence of diverticular disease and screen for colorectal cancer.

Your patient has stopped bleeding by the time you clerk him and your registrar says that he will do the scope outpatient. However, that night you are called to see him for a large spontaneous per rectal bleed. He is now tachycardic - he asks you if he can get a scope right away. Can you counsel him?

Mr Ng, you must be worried about suddenly bleeding again. We take this seriously and need to investigate you. I understand that you are asking for a colonoscope - you do need a colonoscope eventually but right now our priority is to find the source of bleeding and stop the bleed. Colonoscopy is not so good in patients who are currently bleeding, it often cannot find the source because you may have more than one abnormal area / outpouching which can bleed and we don't know which one is bleeding. May I suggest we send you for a scan which, if there is currently fast bleeding, will show us exactly where is bleeding?

Principles of subsequent management (after resuscitation)

- Stable patient (either stable from beginning or responded after resus) with no further bleed (*consider that 80% of bleeds stop by themselves*): stop antiplatelets/warfarin, give outpatient / interval scope to rule out cancer.
- Initially unstable, transient response to resuscitation (rebleeds or unstable): CT mesenteric angiogram looking for arterial blush (minimum 0.3ml/min flow to see) -
 - CTMA finds bleed: proceed to conventional angiogram + embolise
 - Negative CTMA: either stopped bleeding or bleed too slow to detect. Continue to monitor, should not be massive bleed.
 - Offer scope if unfit for CTMA, or when bleeding has stopped.
- Does not respond to resuscitation: surgery stat.

When do you need surgery for bleeding diverticula?

- Please please always rule out UGBIT or hemorrhoidal bleeding (done via on table OGD and proctoscopy) before chopping
- Emergency
 - If bleeding cannot be controlled with angiographic or endoscopic therapy, and source of bleed has already been identified on CTMA → targeted segmental resection.
 - If pt unstable despite aggressive resuscitation, and source of bleed is still unknown → total colectomy. Blind segmental resection contraindicated (high rebleed rate).
- Elective – if more than 2 episodes of significant bleeding (symptomatic anemia or requiring transfusion)

So Mr Ng is very happy with you and refers his friend to see you. His friend has been complaining of blood staining his toilet paper, with a 'lump' that prolapses out of the anal canal with defecation but reduces spontaneously. Proctoscopy reveals haemorrhoids. How would you counsel Mr Ng's friend?

Mr Ng's friend sounds like he has grade 2 internal haemorrhoids. I will first attempt conservative treatment with stool softeners and bulking agents, Daflon, and topical nitroglycerine / nifedipine. If this fails I can counsel the patient for rubber band ligation or sclerotherapy. If all else fails the patient can be referred for surgical haemorrhoidectomy.

BGIT, Upper: Peptic Ulcer Disease

PRESENTATION

Sir, Mr Hong is a 40 year old Chinese gentleman with significant past medical history of migraine requiring frequent analgesia. He presented 3 days ago with three episodes of coffee ground vomitus, malena, and epigastric pain of one day duration.

His pain was sharply felt in the epigastrium and did not radiate to the back or shoulder, pain score was 7/10. 4 hours after onset of the epigastric pain, he had 4 episodes of coffee ground vomitus each about 50ml and 3 episodes of malenic stools. (*If possible, note time of last episodes*). There was no hematemesis or fresh PR bleed.

[Etiology]

- [Variceal bleed] He is not an alcoholic and has no known hx of chronic liver disease or alcoholism. He is not jaundiced and does not have dark coloured urine. He does not have known hepatitis but has never been vaccinated or tested.
- [PUD] He has had previous episodes of epigastric pain worse at night. These were milder and self-limiting, and without hematemesis or malena. He has been taking NSAIDs chronically for his migraine. He is also quite stressed at work, often skips meals, and is a smoker of 20 pack years.
- [Gastric CA] He has not had any early satiety, loss of weight or loss of appetite recently.
- [Mallory weiss] There was no retching before the hematemesis started.
- [Reflux esophagitis] He has no history of heartburn or gastroesophageal reflux symptoms]
- Prior to this incident he has not had any past scopes or H. pylori testing or treatment.

[Complications]

- On admission he was hemodynamically stable with no chest pain, palpitations, giddiness, shortness of breath (haemorrhagic shock / anaemia).
- There was no severe abdominal pain to suggest peritonitis.

I note that he was resuscitated, received blood transfusion, and IV medication. An oesophagoduodenoscopy was performed on the same day. He is currently stable and received no further transfusions or repeat OGD. He tested positive for H. pylori and has commenced triple therapy.

In terms of past medical history, he has bad migraine on follow up with neurology. He has been prescribed beta blockers, NSAIDs, and ergot drugs. He has required his analgesics almost daily when he is stressed. Otherwise, he has no other medical or surgical history, no past scopes. He has no known drug allergies.

Socially, Mr Hong is a top lawyer and is married with 2 children. He is financially well to do and stays in landed property. He smokes (as discussed above) and drinks one can of beer a day.

On examination, Mr Hong was alert and comfortable, receiving IV fluids and proton pump inhibitors. He does not have conjunctival pallor. On inspection, the abdomen has no scars, herniae, and was not distended. On palpation, it was soft non-tender without rebound or guarding. No palpable masses or organomegaly. Percussion note over the abdomen is normal, not tympanic. Bowel sounds are present. I would like to complete my examination with a digital rectal examination looking for melena or fresh PR bleed.

In summary, Mr Hong is a middle aged Chinese lawyer with first episode of upper GI bleeding likely secondary to peptic ulcer disease, status post endoscopic hemostasis

You are the HO on call. How would you manage?

Sir, goals for this patient are to (1) resuscitate him, (2) rule out ddx and cx (3) initiate medical therapy, (4) prepare for endoscopy.

- (1) Ensure patient is stable
 - Assess airway, breathing, circulation
 - Set 2 large bore IV plugs
 - Start fluid resuscitation with normal saline.
 - Insert IDC.
 - Q1H parameters with strict IO charting and stool charting
 - Keep NBM.
- (2) FBC - Hb for anemia, Plt
 - RP - raised U to Cr ratio, prerenal AKI, dehydration
 - PT/PTT, GXM - to call for blood
 - Cardiac enzymes, ECG - for cx of AMI
 - Chest XR looking for free air under diaphragm
- (3) Start IV PPI e.g. omeprazole 80mg bolus then 8mg/h infusion
- (4) Prepare for endoscopy

What if the initial Hb is 13?

Sir, I am aware that in acute blood loss the Hb can be normal. I will not be fooled by a Hb of 13 and will still need to resuscitate as above.

If he comes in with free air under diaphragm, what will you do?

Sir, perforation is a surgical emergency. Patient needs laparotomy and

- Duodenal ulcer: omental patch repair +/- vagotomy
- Gastric ulcer: partial gastrectomy including the ulcer, or patch closure with biopsy.

If there is no free air under the diaphragm, what is your definitive mx?

Sir, patient needs Oesophagogastroduodenoscopy. This is diagnostic, therapeutic, and prognostic.

- Diagnostic:
 - Directly visualize source of bleed (diagnostic)
 - Biopsy to do clo testing for H. pylori
 - Biopsy for histology in gastric ulcer.
- Therapeutic: endoscopic hemostasis using two modalities - e.g. clipping, submucosal adrenaline injection, argon plasma coagulation, etc.
- Prognostic: predict risk of rebleed using Forrest classification

What are the indications for emergency (stat) endoscopy?

- Hemodynamic instability
- Evidence of continued bleeding e.g. hematemesis (malena can be existing blood in the GI tract)
- Suspected variceal bleed

if not - scope within 24h.

Tell me about Forrest Classification.

It is a scoring system to predict risk of re-bleeding.

Forrest 1: Active bleed	1a - spurting
	1b - oozing
Forrest 2: Recent bleed	2a - visible vessel
	2b - blood clot
	2c - hematin stain

Forrest 3: Lesion without bleeding: healed ulcer, clean base

Other scores available - Rockall, Blatchford

Please counsel Mr Hong on OGD.

Mr Hong, I am Dr Tsang. I'm going to explain to you (1) your current condition (indication) (2) what is an OGD (3) risks and complications of OGD (4) risk of not going for OGD and alternative. Do you have any other questions or concerns you would like me to address along the way? If not, still feel free to stop me any time along the way to clarify anything you don't understand.

(1) Your current condition

You are currently bleeding from your stomach or intestine. We have given you a transfusion but we must also find and stop this bleeding. We usually do this with an OGD

(2) OGD

This involves putting a small camera through your mouth, down your food pipe into the stomach and first part of the small intestine. With the camera, we can see where the bleeding is, and once we find the problem we can put a clip and injection to stop the bleeding. You will be sedated so that you won't feel any pain.

(3) Risks and complications of OGD**(a) Risk from sedation**

Can get heart attack and stroke, but there is a low risk of this as you have no previous heart or blood vessel problems and the dosage given is lower than for bigger operations

(b) Risk from procedure itself

Perforation (accidentally poke through the wall of the intestine), failure to stop bleeding, re-bleeding, infection

(4) Risk of not going for OGD and alternatives

If you don't go for the OGD then there is a chance you will keep bleeding and keep needing transfusion of blood. The only other way to stop the bleeding is with an operation, but that is even more invasive and comes with even higher risks.

OK so you have done OGD, Mr Hong tests positive for H. Pylori. How would you treat?

- Triple therapy with PPI (Omeprazole) 20mg BD + Clarithromycin 500mg BD + Amoxicillin 1g BD or Metronidazole 400mg BD x 2/52 then Omeprazole 20mg BD x6/52.
- Follow with test for cure with urea breath test. If H. Pylori –ve, then continue PO PPI.

You treat for H. Pylori. Next day Mr Hong rebleeds. How?

- First rebleed: rescope.
- Second rebleed: surgery (oversewing or underrunning of ulcer, duodenotomy/partial gastrectomy)

OGD showed gastric ulcer. How would you follow up?

In addition to following up on H. pylori eradication, need to re-scope 6 weeks later to document clearance. Need to rule out malignant ulcer.

BGIT, Upper: Variceal Bleed

PRESENTATION

Sir, Mr Lee is a 34 year old gentleman with past medical history of chronic alcoholic liver disease, presents now with 2 episodes of painless hematemesis over 1 day's duration. The 2 episodes of painless hematemesis were unprovoked, each time of about 200ml of fresh blood, associated with 2 episodes of fresh PR bleed followed by 2 episodes of melena. This was preceded by worsening abdominal distension and bilateral lower limb swelling of 1 weeks duration.

[Complications] He does not have symptoms of anemia such as chest pain, giddiness, syncope, palpitations. He does have some shortness of breath but only on lying supine, possibly due to diaphragmatic splinting from his ascites.

In terms of etiology, I note a longstanding history of alcoholic cirrhosis with a recent admission 6 months ago for variceal bleed. He required several blood transfusions and intubation then, and bleeding was stopped successfully via OGD. Since then he has defaulted follow up, and did not undergo interval endoscopic variceal ligations to obliterate any remaining varices.

He admits that he still drinks about 6 cans of guinness stout a day (his baseline since before previous admission), and is unable to cut down. CAGE screen for ethanol use disorder was positive. There is no known hepatitis infection or risk factors such as IVDU or sexual exposure; he is however not vaccinated against hep B.

Other differentials that I considered -

- [PUD] he did not have any abdominal pain, no preceding history of gastritis, no risk factors such as long term corticosteroid use, NSAID use, smoking. He has never been tested for H pylori.
- [Gastric CA]. He does not have early satiety or loss of weight or appetite.
- [Mallory Weiss tear] he did not have severe nausea, vomiting or retching before.
- [Hemoptysis] no preceding coughing or history of TB

Otherwise in terms of his social, functional and financial situation,

- He used to work as a cook in a kopitiam but is currently in between jobs
- He is not on talking terms with his wife, children due to his drinking habits, and currently stays with his elderly mother
- He is not coping well financially and has been receiving some financial support from his mother

On examination, Mr Lee is alert and oriented to time place and person. He does not have asterixis. He has conjunctival and palmar pallor. I note peripheral stigmata of chronic liver disease, namely: jaundice, clubbing, scleral icterus, spider naevi, gynaecomastia, loss of axillary hair. His abdomen is grossly distended. It is tense but non-tender, with a positive fluid thrill. I am unable to palpate for any hepatomegaly. Bowel sounds are active. I measured his blood pressure supine and standing and there is significant postural drop. I would like to complete my examination with a digital rectal examination looking for fresh blood or melena.

In summary, Mr Lee is a 34 year old Chinese gentleman with 2nd episode of hematemesis secondary to alcoholic liver cirrhosis. My main issues are (1) hematemesis, (2) alcohol use disorder, (3) liver cirrhosis.

QUESTIONS

You are the HO on call. How would you manage?

Sir, this is a medical emergency. I will attend to the patient immediately and escalate rapidly to my senior. My management will be (1) to resuscitate the patient according to ABC, (2) to initiate therapy for variceal bleed, (3) to send the patient for urgent endoscopic hemostasis, and (4) to treat underlying condition

(1) Resuscitation

- A Assess airway (blood > airway compromise)
If compromised, intubate.
- B Ensure breathing, saturation
- C 2 large bore IV plugs
Take vitals
Fluid - 2 pint normal saline fast
Call blood bank for transfusion
Keep NBM
Insert IDC
Transfer HD/ICU
Chart: strict I/O, stools, CIWA, Q1h paras

(2) Initiate emergent therapy while awaiting OGD

- Bloods: FBC (Hb), UECr (raised urea to Cr, AKI), LFT, PT/PTT, GXM, Trop
- ECG, CXR
- IV pantoprazole (clot stabilization)
80mg bolus, then 8mg/h infusion for 3 days
- IV somatostatin (splanchnic vasoconstrictor → decrease portal flow & thus pressure)
250ug bolus followed by 250ug/h infusion for 3-5days
- IV ciprofloxacin or ceftriaxone (prevent 2ndary bacterial infection, clot stabilization)
Ciprofloxacin 500mg bd or ceftriaxone 1g od for 7 days
- Glucose and thiamine (prophylaxis for wernicke encephalopathy)

(3) Emergent OGD

Always emergent in suspected variceal bleeds

- Endoscopic variceal ligation (preferred for esophageal varices)
- Sclerotherapy (preferred for gastric varices)

(4) Treat underlying condition

See below (treat underlying portal HTN)

QUESTIONS

What is the definitive management for the varices?

Oesophagealgastroduodenoscopy for visual confirmation of varices with red stigmata of recent hemorrhage, with endoscopic variceal band ligation or sclerotherapy. This is followed by 3-weekly endoscopic variceal ligation until all remaining varices are obliterated.

If 2nd bleed → attempt scope again

If 3rd bleed → consider temporizing measures such as sengstaken blakemore tube

How do you treat the underlying portal hypertension?

- Prevention of progression of CLD, prevent further insults to liver
 - Refer NAMS (national addiction management service), pharmacological abstinence therapy
 - Treat any underlying Hep B/C, if no underlying Hep infection then vaccinate
- Medical: non-selective beta blockers i.e. propranolol
- Radiological: transjugular intrahepatic portosystemic shunt (beware hepatic encephalopathy)
- Surgical: surgical shunts (not done anymore)

How do you use a Sengstaken Blakemore tube?

- Secure airway first - intubation
- Measure SB tube as per nasogastric tube (usually ~40cm)
- Advance SB tube via nose
- Fill gastric balloon with 150ml of water or gastrograffin (for position check)
- Tug gently to ensure gastric balloon is sufficiently inflated, then anchor with pulley and traction
- Inflate esophageal balloon to 20-25mmHg
- Can aspirate blood in stomach
- Confirm position of tube with CXR
- Release intermittently to prevent pressure necrosis

Breast Cancer

PRESENTATION

Sir, my patient is Mdm Chin a 73-year-old Chinese retiree who presented with an asymptomatic right breast lump.

The lump was picked up on mammogram during a free neighbourhood breast cancer screening program. The lump is painless. She is unaware of how long she has had it, and does not know if it has been increasing in size. She has not noticed any nipple discharge or skin changes over the lump. She also has not experienced any constitutional symptoms such as LOW or LOA, nor any shortness of breath, constant progressive headache, increase or change in nature of her back pain that might suggest distant spread.

In terms of risk factors, she has a significant family history of breast cancer -- 5 out of her 7 sisters, have breast cancer as well. Age of diagnosis ranges from 55 to 67. She is the youngest in her family. There is no personal or family history of colon, ovarian, pancreas, thyroid or skin cancers. Otherwise she does not have prolonged estrogen exposure -- she had her menarche at 14 years of age, has had 3 children all breastfed, has never taken oral contraceptive pills, went into menopause at 50, and has not been on hormone replacement therapy for her osteoporosis. She also has never received radiotherapy, and being a seamstress does not have occupational exposure to radiation.

She has never had any procedures or surgeries to the breast. She had done a mammogram done 10 years ago when the 1st of her sisters got diagnosed with breast cancer. It was normal at the time. She did not feel the need to do any more mammograms since, and decided to go for the mammogram screening this time because it was free.

Otherwise she has a past medical history of osteoporosis, previous compression fractures in 2013 and left neck of femur fracture in 2014 s/p fixation, HTN HLD and DM well controlled on medications, and bilateral cataracts s/p insertion of intraocular lense in 2015. No drug allergies.

In terms of social history, she does not smoke or drink. She stays with her daughter and son in a HDB flat with a lift landing. She is ADL independent and community ambulant with a walking stick. The family is able to cope financially with the cost of treatment.

On examination, Mdm Chin is alert and comfortable. She does not look cachectic, her temporalis muscles are not wasted. On inspection, the breasts are symmetrical with no obvious masses, scars, skin changes or nipple changes. There is no tethering of the skin accentuated by the patient tensing her pectoralis majors. On palpation, I note a irregular ill-defined mass in the right breast at the 10 o'clock position 5 cm from the nipple, measuring about 4cm by 4cm. It is non-tender and not warm, not fixed to the overlying skin or underlying structures. There were no other masses felt in the right breast, no masses felt behind both nipples, and the contralateral breast was normal. She is not able to express any discharge. On examination of the axilla, I found a small non-tender mobile node about 1.5cm by 1.5cm. There was no cervical lymphadenopathy. There was no tenderness on percussion of the spine, auscultation of the lungs was clear and no hepatomegaly on abdominal exam.

In summary, Mdm Chin is a 73 year old lady with a significant family history of breast cancer, who now presents with a right breast lump for investigation.

QUESTIONS

How would you investigate this lady?

Sir, I would like to complete the triple assessment, which in addition to clinical evaluation would involve radiological assessment with mammogram and ultrasound and histopathological assessment with core biopsy. Tissue should be sent for histology, plus estrogen/progesterone receptor and HER2 receptor staining. After which, if the diagnosis of breast cancer is confirmed, I would proceed to stage the patient with a CT Thorax and liver +/- bone scan.

Please interpret Mdm Chin's mammogram.

Sir, these are the cephalocaudal and medial lateral oblique views of Mdm Chin's mammogram. I see a ill-defined spiculated hyperdensity located in the upper outer quadrant (MLO: lateral, CC: superior). There are pleomorphic microcalcifications around the lesion. There is some architectural distortion causing tethering of the skin.

Yes it was reported as BIRADS 5. What does this mean?

The breast imaging reporting and data system (BIRADS) is a standardized way of communicating mammogram findings. BIRADS 5 means a highly suggestive of malignancy and biopsy should be done.

BIRADS	0 = incomplete assessment
	1 = normal
	2 = benign finding, go back to routine screening
	3 = likely benign, needs close interval follow up in 6 months.
	4 = suspicious for malignancy, must biopsy
	5 = highly likely malignancy
	6 = biopsy proven malignancy.

Please interpret the ultrasound image

Sir, this is an ultrasound image of Mdm Chin's right breast lump. It is solid-cystic, taller than wide. Its margins are irregular and I note internal microcalcification casting posterior acoustic shadow.

CTTAP confirmed no metastasis elsewhere, no other axillary or internal mammary lymphadenopathy other than the axillary node you found on examination. Bone scan is normal. What stage is Mdm Chin?

- T1 <2cm
- T2 2-5cm
- T3 >5cm
- T4 direct ext to chest wall or skin

- N1 ipsilateral axillary level 1-2 nodes that are mobile
- N2 ipsilateral axillary level 1-2 nodes that are matted OR internal mammary nodes
- N3 ipsilateral axillary level 1-2 nodes that are matted
AND internal mammary nodes, infracalvicular nodes OR supraclavicular nodes

- Stage 1: Max T1 N0
- Stage 2: Max T2 N1, T3 N0
- Stage 3: A = T0-3 N2, T3 N1-2, B = T4 any N, C = any T N3
- Stage 4: Any T, any N, M1

According to the TNM classification, Mdm Chin is Stage 2b = early stage breast cancer.

Please outline the principles of management of breast cancer.

Sir my goal of treatment for this patient is curative. She should receive multidisciplinary, multimodality care and needs to be discussed at tumour board.

The options are that of breast conserving surgery with postoperative radiotherapy vs mastectomy. As she has a palpable lymph node, axillary clearance should be done (no role for sentinel lymph node biopsy if LN already palpable). I will need to discuss this with the patient and look for contraindications to breast conserving surgery such as

- Small breast to tumor ratio: small breast, large tumor
- Multifocal tumor
- Contraindication to RT: connective tissue disease, unable to comply, (pregnancy - not in this lady)
- Recurrence of breast cancer (not in this lady)

Following surgery she may be offered adjuvant chemotherapy depending on surgical staging, hormonal therapy if she is hormone receptor positive (aromatase inhibitor since postmenopausal, tamoxifen in younger ladies), or herceptin if HER2 positive. Need to monitor contralateral breast with screening mammogram!

General principles

	DCIS	Early stage (max T2N1)	Locally advanced (T ≥3 or N ≥2)	Unresectable / Metastatic
Goal	Curative			Palliative #
Local Rx	BCT +/- RT or Simple mastec +/- reconstruction	BCT + RT or Simple mastectomy +/- reconstruction	Neoadjuvant chemo/RT Mastectomy (no BCT) + RT (since high stage) +/- reconstruction	Toilet mastectomy
Regional Rx	BCT: no need SLNB Mastec: do SLNB*	Clinical nodes: Axillary clearance No clinical nodes: SLNB; if +ve, axillary clearance	Axillary clearance	
Systemic Rx	No chemo, Herceptin Can give tamoxifen.	Chemotherapy - multiple potential agents. If ER/PR +ve: hormonal therapy Tamoxifen (premenopausal) vs AI (menopausal) If HER2 +ve: targeted therapy - Herceptin		

* Need for SLNB in DCIS is disputed. If the patient is going for total mastectomy, it may no longer be possible to do SLNB at a later date because there is no more breast tissue in which to inject blue dye -- so if invasive cancer is unexpectedly found in resection tissue, a full axillary clearance would be required. Hence DCIS going for total mastectomy, SLNB is favoured. However for DCIS going for BCT, there may not be a need to do SLNB.

^ Axillary clearance prognosticates (to decide on systemic Rx) but not cures

Modalities for symptom control include

- Brain mets: RT
- Bone pain: RT
- #: prophylactic fixation
- Cord compression: RT, steroid
- Pleural effusion: tap

Can you explain what is sentinel lymph node biopsy?

SLNB is the standard of care to assess regional lymph nodes in early breast cancer (patients with locally advanced breast CA do not qualify). As most early stage breast cancers have a lower clinical suspicion for lymph node involvement, the sentinel lymph node biopsy saves many of these patients from the morbidity of axillary clearance, which includes lymphedema and recurrent cellulitis of that arm.

To do a SLNB, dye is injected into the breast tissue and the first lymph node the dye drains to (i.e. the sentinel node) is removed to be assessed under frozen section.

The sentinel lymph node is taken to be representative of the nodal status of the rest of the axillary nodes.

- If the sentinel node is negative, then axillary node clearance is not done.
- If the sentinel node is positive for metastasis, then full axillary clearance is done. This is to determine nodal status, to prognosticate and guide (systemic) treatment (NOT to remove disease).

What are some other variants of breast cancer and when do you think of them?

- Phyllodes tumor - a fibroepithelial tumor which ranges from what resembles a benign fibroadenoma, to a clearly malignant lesion which grows much faster and larger than the other breast cancers
- Inflammatory breast cancer - an aggressive form of locally advanced breast cancer where tumor emboli within dermal lymphatics cause lymphedema. The breast is painful, swollen, with erythematous skin and a peau d orange appearance. This has a poor prognosis.

Dysphagia: Esophageal Cancer

PRESENTATION

Mr Teo is a 60 year old Chinese gentleman who presents with a one month history of painless progressive dysphagia.

He describes his dysphagia as a feeling of food getting stuck in his chest, rather than difficulty with swallowing itself. Dysphagia was initially worse with solid food than with liquids, and he noted a gradual change in food preference from rice and meat, to porridge and soup. There is associated regurgitation of undigested food 5 minutes after swallowing. Over the past month his dysphagia has gotten progressively worse and in the past few days he struggled to tolerate even liquids. What prompted him to seek treatment at A&E was that he vomited liquids yesterday.

He has clinically significant loss of weight of 10 kg over the past month, with very poor oral intake and postural giddiness suggesting dehydration.

There are no symptoms to suggest local invasion such as hoarseness of voice (recurrent laryngeal nerve involvement) or hemoptysis (tracheal-esophageal fistula). He also does not note any hematemesis or melena, no symptomatic anaemia, and has no symptoms of aspiration pneumonia (or esophago-pulmonary fistula) such as fever or shortness of breath. He is not jaundiced (distal mets).

In terms of risk factors,

- [For SCC] I note that he is a heavy smoker of 200 pack years and is also a heavy drinker with frequent binges. He drinks very hot tea. Otherwise, he has no ingestion of caustic chemicals,
- [For adenoCA], no history of gastroesophageal reflux symptoms.

He does not have any past medical or surgical history, nor any past scopes. He is not on any long term medications and has no drug allergies.

Socially and functionally, he works as a delivery man which requires him to be able to carry heavy loads, but he has been struggling to keep up with this work. He cannot afford to stop working, however, as he is the main breadwinner of his family with two kids. He stays with wife and children in a 3-room HDB flat and they are financially tight.

On examination, Mr Teo is cachectic with wasted temporalis and intrinsic muscle of the hand. He is on an IV drip and his mucous membranes appear dry. I find conjunctival pallor but no scleral icterus. I do not note any scars over his neck, chest, or abdomen. His abdomen was soft and non-tender with no hepatomegaly. His lungs were clear. There was no goitre and no cervical lymph nodes palpable.

In summary, my patient is a 60 year old Chinese gentleman, with a 1 month history of progressive esophageal dysphagia and significant LOW 10 kg.

QUESTIONS

What are the differentials you would consider for this gentleman?

Sir my primary differential for this gentleman is that of a malignancy of his esophagus or gastroesophageal junction (may also consider gastric CA). I am also considering other causes such as -

- Benign strictures, although he does not have any history of caustic chemical ingestion.
- Achalasia, although I would expect achalasia to present with difficulty swallowing solids and liquids from the start
- Extraluminal compression by the 4 Ts - thyroid, thymoma, teratoma, or terrible lymphoma.

How would you investigate this gentleman?

Sir my goals are to (1) confirm the diagnosis, (2) stage the disease, and (3) prepare for surgery.

I would like to start with some blood tests

- FBC: looking for anemia, raised total whites in asp pneumonia
- RP: looking for raised urea and creatinine in view of dehydration, as well as hyponatremia (can cause difficulty swallowing, lethargy → misconstrued as decreased effort tolerance)
- LFT: albumin, liver metastasis → ALP, liver enzyme derangements
- CXR to rule out aspiration pneumonia

Definitive confirmation of diagnosis can be undertaken via

- Gastrograffin swallow looking for irregular shouldering, and ruling out the rat-tail appearance of achalasia.
- Esophagealgastroduodenoscopy which will allow me to directly visualize the lesion, measure its distance from the incisors as well as take tissue biopsies.

Staging can be divided into locoregional and distant staging

- Locoregional staging for T and N stage is best undertaken with endoscopic ultrasound. (difficulty: less accurate if the scope cannot pass the tumor)
- Distant staging wise, patient will need CT thorax abdo pelvis +/- PET looking for lung, liver, and adrenal mets.

Pre-operatively patient will also require lung function test, PT/PTT, GXM.

OK can you read this OGD photo please?

- Sir I see a tight stricture with slight erythematous change and contact bleeding of the mucosa. The scope is unable to pass the stricture,
- OR sir I see an ulcerative fungating mass with contact bleeding

OK so a biopsy is taken. What is the likely histological type in this gentleman?

Sir I think a squamous cell carcinoma is more likely than adenocarcinoma in this gentleman given his risk factors are smoking and alcohol and there is no GERD.

How would you treat this gentleman if -

(a) Tumor invades mucosa only (T1a), N0, M0

Sir the patient is suitable for endoscopic mucosal resection with spares patient the morbidity of esophagectomy. However patient will need frequent repeat endoscopes.

(b) Tumor invades submucosa or muscularis propria (T1, T2), N0, M0

Sir this patient should be discussed at a multidisciplinary tumor board. He can receive esophagectomy with complete (R0) resection, adequate proximal and distal margins, and appropriate nodal dissection. Options are

- Transthoracic: either
 - Two-stage (Ivor Lewis): with intra-thoracic esophageal anastomosis, abdominal and right thoracotomy scars
 - Three stage (McKeown):
 - with cervical esophagogastric anastomosis, abdominal, thoracic, and cervical scars
- Transhiatal: done in 4 stages, with cervical esophagogastric anastomosis, abdominal and cervical scars

(c) Tumor invades adventitia (T3) +/- adjacent resectable structures (T4)

As for (b) but with neoadjuvant chemoradiotherapy.

(d) Unresectable or metastatic disease

Sir this patient should receive palliative chemoradiotherapy. For palliative mitigation of dysphagia, he can receive palliative stenting and/or palliative radiotherapy (must be done before stenting or stent will drop). Patient can also have insertion of NG tube or PEG to facilitate feeding.

(e) Gastroesophageal junction tumors

Sir, I am aware that GEJ tumors are mostly staged as esophageal tumors (unless epicentre in stomach >5cm from EGJ or not extending into esophagus). Management depends on location from GEJ

- Siewert 1: Esophagectomy plus partial gastrectomy, plus appropriate lymph node dissection
- Siewert 2 & 3: Total gastrectomy with distal esophagectomy (only abdominal incision), plus appropriate lymph node dissection

How do you optimise the patient for surgery?

- Nutritional: NG vs TPN
- Lung: LFT, chest physiotherapy.

What are the post-op complications?

- Anastomosis-related
 - Anastomosis leak → mediastinitis
 - Anastomotic stricture → GOO symptoms
- Injury to surrounding structure: Recurrent laryngeal nerve, chylothorax
- Cardiovascular: AMI, VTE
- Pulmonary: Atelectasis, pneumonia
- Related to altered anatomy (stomach now functioning as esophagus): GERD, dumping syndrome

Another patient with dysphagia is diagnosed with achalasia. What is achalasia?

Aperistalsis of distal esophagus due to degeneration of ganglion cells in myenteric plexus. This results in failure of lower esophageal sphincter to relax. Diagnosis is via barium swallow showing smooth distal rat-tailing (vs shouldering in esophageal CA), esophageal manometry showing failure of peristalsis, increased lower esophageal sphincter resting tone and failure of lower esophageal sphincter to relax on swallowing.

How do you treat achalasia?

Aim is to decrease lower esophageal sphincter tone. Can be done with medical, endoscopic or surgical means.

- Medical: Calcium channel blockers, nitrates
- Endoscopic: Injection of botulinum toxin, pneumatic balloon dilatation
- Surgical: Laparoscopic Heller's esophagomyotomy with partial fundoplication

GOO: Gastric Cancer

PRESENTATION

Sir, my patient Mdm Goo is a 70 year old Chinese lady with past medical history of peptic ulcer disease, now presenting with symptoms of gastric outlet obstruction of 2 weeks duration, with significant loss of weight.

I note that she has a significant history of peptic ulcer disease. She was admitted in 2007 for what sounds like a perforated peptic ulcer s/p emergency repair (*sounds like patch repair*). She was again admitted in 2010 with malena requiring endoscopic hemostasis. Both times she has received triple therapy but defaulted the follow up urea breath tests and scope. She has been asymptomatic until this year.

Over the last 2 months, however, she has been complaining of early satiety and vague epigastric discomfort which has been getting worse. She has gravitated towards small frequent meals of semi-solid foods like porridge or just liquids like soup. In the last 2 weeks, she also has post-meal vomiting (forceful, vs regurg which is not), 5 minutes after meals. Vomitus is non-bilious, non-bloody, and consists of the undigested food she had just ingested. This is associated with clinically significant weight loss of 10kg over 2 months.

Her presentation sounds like gastric outlet obstruction and I am considering malignant obstruction, benign strictures, as well as extraluminal compression

- First, I am most worried about gastric cancer.
- It may also be benign fibrotic stricture due to previous peptic ulcer disease
- There is no progressive painless jaundice, or dark urine / pale stools, to suggest a periampullary tumor that could compress on the gastric outlet.

In terms of other complications of gastric cancer, there is no hematemesis, coffee grounds vomitus, or malena from a bleeding malignant ulcer; and the patient does not have symptoms of anaemia such as chest pain / SOB / giddiness / palpitations. There is no sudden severe generalized abdominal pain suggestive of perforation. There is no ascites from transcoeliomic spread. Systemic review was unremarkable for distant spread.

In terms of past medical history, she has well controlled DM HTN and HLD, and no other past medical or surgical history. No previous gastrectomy, no history of GERD. She has had no more scopes since the 2010 admission and no known drug allergies.

She is retired, stays with her husband a HDB flat, and spends most of her time helping to look after her grandchildren. Financially, she is well-supported by her children. She does not smoke or drink.

On examination, Mdm Goo is alert and comfortable, slightly cachectic. She is currently on IV drip. She does not have scleral icterus or conjunctival pallor. Her abdomen has no scars, is not distended, and is soft and non-tender on palpation, no rebound tenderness or guarding. There is no masses in epigastrium or anywhere else, no hepatomegaly, no palpable gallbladder. There was no shifting dullness. Succussion splash was positive. Bowel sounds were present and active. No inguinal hernia. She did not have any cervical lymphadenopathy specifically no Virchow's node.

I would like to complete my examination by doing a digital rectal examination looking for PR bleed or melena, and masses in the rectouterine pouch or fixed adnexal masses in the blumer's shelf suggestive of metastatic disease.

In summary, Mdm Goo is a 70 year old Chinese lady presenting with symptoms of gastric outlet obstruction, preceded by 2 months vague epigastric discomfort and loss of weight, and positive succussion splash on examination. There is no palpable abdominal mass or jaundice. I am most worried about gastric cancer, not complicated by bleeding or perforation. My other ddx are benign strictures or extraluminal compression.

QUESTIONS

What are the risk factors for gastric cancer?

Sir, the development of gastric cancer is multifactorial due to the interplay of past gastric conditions, environmental factors including infection, and host factors including genetics.

1. Premalignant and other gastric conditions
 - Peptic ulcer disease
 - Atrophic gastritis
 - Previous gastric resection with bile reflux
 - Gastric polyps
2. Environmental factors
 - Infection: H. pylori infection
 - Diet: nitrosoamines in preserved food, smoked food
 - Smoking
 - Low socioeconomic status
3. Genetic and other host factors.
 - Obesity
 - Family history

What are the various ways gastric cancer can present, and what are their differentials?

Presentation	DDx
Asymptomatic, picked up incidentally	
Nonspecific - Early satiety, dyspepsia	Peptic ulcer disease Biliary colic.
Constitutional - LOW, LOA	
Anemia	Colon cancer Peptic ulcer disease Post-gastrectomy anemia (B12, Fe def) Menorrhagia, endometrial cancer
Epigastric mass	Pancreatic tumor (or pseudoaneurysm etc) HCC of left lobe Transverse colon CA Gastrointestinal stromal tumour
Cx of ulcer - Bleeding, Perforation	Peptic ulcer, variceal bleed
GOO	Peptic ulcer disease cx stricture Head of pancreas cancer
Dysphagia	Esophageal cancer Gastroesophageal junction tumor
Obstructive jaundice	Periampullary cancers
Transcoelomic metastasis: Ascites	Cirrhotic/tuberculous ascites

How would you investigate?

Sir, I would start off with some basic investigations:

- FBC looking at Hb for anemia
- RP looking for hypokalemia hypochloremic metabolic alkalosis with paradoxical aciduria, rise in Cr from pre-renal AKI (dehydration)
- LFT looking for cholestatic pattern (possible in HOP or LN at porta hepatis) vs hepatocellular pattern (liver metastasis)
- CXR looking for free air under the diaphragm
- ECG PT/PTT GXM - pre-op w/u

Then I would like to prepare the patient for an OGD, which is both diagnostic and therapeutic. It will allow us to confirm GOO (presence of food debris, absence of duodenal visualization), directly visualize the lesion and location, and take a biopsy for histological confirmation, and placement of a stent for temporary relief of GOO symptoms.

Can you please read this scope picture?

- Sir this is an endoscopic view of the gastric outlet showing a large fungating tumor with contact bleeding. The gastric outlet is almost entirely occluded and I think the scope cannot intubate past the pylorus.
- Sir this is an endoscopic view of the stomach. I note a large ulcer with heaped up edges. There is contact bleeding from the ulcer.

Histology confirms adenocarcinoma. Please outline your management for Mdm Goo.

I would first stage the lesion with:

- (1) Endoscopic ultrasound for T and N staging
- (2) CTTAP for N and M staging
- (3) PET scan also for M staging

Thereafter the patient should be discussed at tumor board and managed by a multi-disciplinary team.

Further discussion depends on the scans they show you -

(a) Early gastric cancer i.e. superficial to muscularis propria.

- Can be treated with endoscopic mucosal resection

(b) Non-early gastric cancer -- for curative Rx

- Pre-operatively there is a role for laparoscopy to rule out peritoneal metastases which may be occult on CT scan.
- If there are no peritoneal metastases I will offer curative resection involving
 - Wide resection with oncological margins (≥ 6 cm confirmed with frozen section): partial / subtotal / total gastrectomy
 - En-bloc resection of lymphovascular supply and any structures involved by local invasion
- Re-establish GI continuity with Roux-en-Y (alternatives: Bilroth I, Bilroth II)
- Couple with 6 cycles of chemotherapy (3 neoadjuvant and 3 adjuvant)
 - Chemotherapy regimen: 5FU, epirubicin, cisplatin

(c) Palliative Mx:

- Painful bony mets - external beam radiotherapy
- Bleeding - transcatheter embolization, palliative gastrectomy
- Obstruction - palliative gastrectomy, self-expanding metal stent, feeding jejunostomy
- Perforation - palliative gastrectomy

What are the post-op complications to look out for, and what to do about them?

Early: bleeding, infection, injury to surrounding organs, anastomotic leak, stump blowout

Late:

- Pre-meal: Early satiety due to loss of gastric reservoir → take small frequent meal
- Post-meal: early dumping syndrome (hypovolaemic symptoms from fluid shift) vs late (hypoglycaemic symptoms) → take small frequent meal
- Between meals: Nutritional deficiencies - B12 (loss of intrinsic factor), Fe → give B12 injections, Fe supplements
- Afferent loop syndromes
- Retained antrum syndrome
- Bile reflux gastritis

OK apart from adenocarcinomas what other gastric cancers do you know of?

While the majority of gastric cancers are adenocarcinomas (diffuse type or intestinal type - Lauren classification), other histological types include GIST, carcinoid tumors, and lymphomas.

Yay! I love GIST! Tell me about GIST?

GIST stands for Gastrointestinal Stromal Tumour. It is the most common stromal/mesenchymal neoplasm arising from the GI tract. It is characterised by cKIT mutation which causes tyrosine kinase hyperactivity, picked up as CD117 antigen expression (part of the KIT membrane receptor's tyrosine kinase). PDGFRA (Platelet derived growth factor receptor alpha polypeptide) gene mutation is also seen in GIST.

While GISTs occur throughout the GI tract from the esophagus to the anus, they are most common in the stomach (40 to 60 percent) and jejunum/ileum (25 to 30 percent), and less commonly in the duodenum (5 percent), colon/rectum (5 to 15 percent), and esophagus (≤ 1 percent). They can also occur in the retroperitoneum, mesentery and omentum. They frequently metastasize to liver, omentum, peritoneal cavity and rarely to regional lymph nodes. They uncommonly metastasize to the lungs, the most common site of metastasis for most soft tissue sarcomas (dDx for GIST).

The clinical behavior of GISTs is highly variable; the main prognostic determinants for are tumor size, mitotic rate, and tumor location. Tumours with high risk of recurrence are those >2 cm, high mitotic rate and extra-gastric location.

How do GIST present?

GIST tumours can be asymptomatic and discovered incidentally. More often, they are associated with nonspecific symptoms (ie, early satiety, bloating) unless they ulcerate and bleed, or grow large enough to become a palpable mass or cause intestinal obstruction.

What is the diagnostic work-up?

The preoperative diagnosis of a GIST requires a high degree of suspicion and familiarity with its radiologic appearance.

1. *CT/MRI scan*: Appears as a solid smoothly contoured mass that enhances brightly with IV contrast. Very large tumors (>15 cm) may appear more complex/heterogenous due to necrosis, hemorrhage, or degenerating components. It may be difficult to identify the origin of a large mass because of exophytic growth.
2. *OGD*: Appears as a submucosal mass with smooth margins, a normal overlying mucosa, and bulging into the gastric lumen, occasionally with central ulceration
 - Leiomyomas have the exact same appearance
3. *Endoscopic Ultrasound*: Can help to characterize lesion and determine if benign or malignant. Tumors that disrupt the normal tissue planes, contain cystic spaces, and are associated with enlarged lymph nodes are more likely to be malignant.
4. *Pre-op Biopsy, EUS guided preferred*: Preoperative biopsy is not generally recommended for a resectable lesion in which there is a high suspicion for GIST, and the patient is otherwise operable. However, a biopsy is preferred to confirm the diagnosis if metastatic disease is suspected or if preoperative imatinib is considered prior to attempted resection in a patient who has a large locally advanced lesion thought to represent a GIST.

How do you treat a GIST?

- Local control: Surgical resection
- Systemic control: neoadjuvant or adjuvant targeted therapy with tyrosine kinase inhibitors (imatinib) --- TKIs have transformed the treatment of GIST.
- Hypothyroidism is a common side effect of antiangiogenic tyrosine kinase inhibitors

Hematuria: Bladder Cancer

PRESENTATION

Sir, my patient Mr Pradeep is a 55 year old indian man with past medical history of urolithiasis in 2014 status post extracorporeal shock wave lithotripsy. He now presents with a 2 week history of painless gross hematuria.

This gross hematuria is painless, persistent and mixed with urine/throughout micturation, no blood clots. It does not occur in between micturition and he does not notice any blood staining his underwear. This is associated with some storage symptoms such as frequency and urgency, but no voiding symptoms such as hesitancy, slow and intermittent flow, terminal dribbling, incomplete voiding, and double voiding.

[Ddx]

- [RCC/Urolithiasis] It is not associated with any dull flank ache or sharp loin to groin pain
- [Cystitis] There is no fever, dysuria or pyuria
- He has had no recent instrumentation to the urethral tract.
- [Anticoagulant] He is not on any anticoagulants.
- [Renal] I also considered renal causes but he does not have lower limb swelling, decreased urine volumes, or recently diagnosed hypertension. He does not have any joint pain, rashes, or history of autoimmune disease.

Otherwise he has no pneumaturia or recurrent urinary tract infections to suggest local invasion, no constitutional symptoms such as LOW LOA, and no SOB, bone, pain or neurological symptoms to suggest distant spread.

In terms of risk factors, he does not smoke, does not have occupational exposure to industrial chemicals as a cabin crew member, and does not have family history of any urological cancers or colorectal cancers.

In terms of past medical history, he had hematuria and sharp flank pain secondary to kidney stones in 2014 s/p ESWL, as well as previous testicular torsion s/p orchidopexy when he was 18. He has no other past medical, surgical or drug history and no known drug allergies.

Social, functionally and financially, he stays with his wife and 2 children, is able to work as per normal as a cabin crew member and is financially stable.

On examination, Mr Pradeep was alert and comfortable at rest. He is not cachexic. Abdominal examination found no scars and a soft non-tender abdomen. There was no organomegaly, most notably no ballotable masses and no palpable bladder. In the groin there was no left-sided varicocele. Digital rectal exam revealed a normal prostate of 1.5 breadths that was smooth, firm, no nodules, median sulcus felt, overlying rectal mucosa mobile over it. Anal tone was intact. No spinal tenderness, lungs clear.

In summary Mr Pradeep is a 55 year old indian man with 2 weeks of painless gross hematuria for investigation. I am most suspicious for bladder cancer, with a differential of renal cell carcinoma, prostate cancer, and although less likely in the absence of pain or infective symptoms, urolithiasis and urinary tract infection.

QUESTIONS

How would you investigate this patient?

Sir, I would (1) confirm diagnosis (2) look for complications (3) stage disease and (4) plan for treatment

I would start with the following basic investigations -

- FBC looking at Hb for anemia, and total whites
- Renal panel looking at Cr for kidney function and for contrast studies
- PT/PTT, GXM in preparation for procedures
- UFEME looking at RBC WBC bacteria casts and crystals
- Urine gram stain and cultures
- Urine cytology for malignant cells

In terms of imaging I would do

- XR KUB looking for radio-opaque stones
- CT urogram looking for any renal tumours or any synchronous tumours in the upper tracts (field change effect); also to stage N and M.
- CXR to rule out lung metastases

To confirm the diagnosis he will require cystoscopy which can visualize the lesion and its location as well as take a biopsy.

Subsequently if cystoscopy confirms a tumor, he will require transurethral resection of bladder tumor. This is both diagnostic to determine depth of invasion (superficial vs muscle invasive), as well as therapeutic.

If there is muscle invasive disease, then we will also need to look for metastases with CT thorax and with the CT urogram already done.

OK a 4cm exophytic papillary lesion seen. TURBT was performed. Mr Pradeep's histology comes back as superficial bladder cancer. What now?

For superficial bladder cancer, the TURBT would already have been the primary treatment. He would benefit from intravesical therapy with Bacillus Calmette Guerin (BCG) or mitomycin C as a >3cm tumor is high risk*. Following treatment careful surveillance for recurrence and second tumors is required, e.g. cystoscopy and urine cytology in 3 months.

** High-risk features are:*

- high grade
- multi-focality
- multiple recurrences
- tumour size >3cm
- primary or coexisting carcinoma in-situ
- prostatic urethral involvement

Mr Pradeep decided he does not want intravesical therapy. He returned to his job as a cabin crew and was lost to follow up. 4 years later he comes back with the same symptoms, and was found to have recurrence of bladder tumour -- this time muscle invasive. Repeat CT urogram and bone scan show no synchronous lesions and no mets. What would you offer him?

- I would offer him curative management with neoadjuvant platinum-based chemotherapy and then radical cystectomy and pelvic lymphadenectomy with urinary diversion, usually an ileal conduit or with a neobladder.
- If he refuses surgery - I suppose multimodal therapy with TURBT, chemotherapy, and radiotherapy is possible but if this fails then cystectomy becomes difficult.
- *Note: if metastatic, chemotherapy is the mainstay of treatment.*

Hematuria: Renal Cell Carcinoma

PRESENTATION

Sir, my patient Mr Cui is a 75 year old Chinese gentleman on warfarin for old mitral and aortic valve replacement, who presents now with 1 month of painless gross hematuria, left flank discomfort and loss of weight.

With respect to his painless gross hematuria, this has been persistent for 1 month, mixed with urine throughout micturition, and associated with vermiform blood clots. There is no urethral bleed or staining of underwear between micturation. He also complains of dull left flank discomfort, which has been become more prominent recently. Occasionally he also feels a sharp flank pain radiating to the groin, which will be followed by passage of a blood clot. I also note weight loss of 5kg over the past month.

[Ddx]

- Otherwise, there is no fever or pyuria to suggest pyelonephritis
- No LUTS symptoms such as dysuria, urgency, and frequency (cystitis), hesitancy intermittency double-voiding (BPH), no pneumaturia or recurrent UTIs (e.g. bladder tumor with local invasion)
- He does not have any history of urolithiasis, parathyroid tumor, gout, or endocrine tumors (MEN). He is not on any medications that could cause hypercalciuria, has no known hx of gout or joint pains, and drinks at least 2 litres of water each day.
- I also considered renal causes but he does not have lower limb swelling, decreased urine volumes, or new hypertension. He does not have any joint pain, rashes, or history of autoimmune disease.
- His last INR 3 months ago was said to be within range. He has no other symptoms such as per rectal bleeding or easy bruising. He does not drink at all, has not been prescribed any new medications recently, sticks religiously to a stable diet and has never in the past 20 years been admitted for complications from warfarin.

He has not had any jaundice, shortness of breath, bone pain or constant progressively worsening headache suggestive of metastasis. In terms of paraneoplastic syndromes, I note there is no symptoms of hypercalcemia, and no painful leg swelling to suggest deep vein thrombosis, no weakness and rash to suggest dermatomyositis.

In terms of risk factors, he is a smoker of 50 pack years but does not have any family history of kidney tumours. No risk factors such as industrial chemical exposure or pelvic irradiation

As for his past medical history, I note he had aortic and mitral valve replacements 20 years ago for rheumatic heart disease. He is stable on warfarin. Otherwise there is no other medical or surgical history, and no other long term medications other than warfarin. He does not drink because of his warfarin. No known drug allergies.

Socially functionally and financially, he stays with his wife in a 2 room studio flat. Both are retired and well-supported financially by their 3 children.

On examination, Mr Cui is alert and comfortable. His blood pressure is 120/70. I note that he is slightly cachectic. He has a midline sternotomy scar and audible metallic clicks by bedside. I note that he has an IDC, urine bag containing gross hematuria. There is no scleral icterus, conjunctival pallor OR suffusion, no easy bruising. His abdomen is not distended, soft and non-tender. There is a left ballotable mass but no other organomegaly. No palpable bladder. Bowel sounds are present. Hernia orifices are intact. There is a left varicocele. Digital rectal examination revealed a normal prostate and no PR bleeding.

In summary, this is a 75 year old gentleman who has had 1 month of painless gross hematuria with vermiform clots, left flank discomfort and significant loss of weight, associated with a ballotable left flank mass and left varicocele on physical examination. I am most suspicious of left renal cell carcinoma.

QUESTIONS

What are your differentials?

Other differentials include

- Urolithiasis but the sharp loin to groin pain is always in relation to passing of a blood clot and he has persistent hematuria in between the loin to groin pain episodes.
- Bladder cancer

What are some of the paraneoplastic syndromes associated with RCC?

- Hypertension - due to overproduction of renin
- Hypercalcemia - due to production of PTH-like protein
- Polycythemia - due to overproduction of erythropoietin
- Venous thromboembolism
- Non-metastatic hepatic dysfunction.

How would you investigate?

Sir, I would investigate with the aim of (1) confirming diagnosis and ruling out differentials (2) looking for complications (3) staging.

But I would like to start with the following basic investigations:

- FBC looking at Hb for anemia or polycythemia, TW for infection
- UECr looking at Cr in view of contrast studies
- PT/INR make sure it is in range
- GXM as patient may be going for surgery
- UFEME for RBC, WBC, bacteria, casts, crystals
- Urine cytology for malignant cells
- XR KUB looking for radio-opaque stones
- Most importantly a triphasic CT Kidney looking for a renal parenchymal mass with irregular borders and enhancement after contrast injection, any lymph node involvement, perinephric extension beyond Gerota's fascia, renal vein or IVC extension

Then staging should be completed with

- CT thorax and abdomen looking for distant metastasis to the lung, liver
- Bone scan for bone mets
- KIV MRI w gadolinium abdomen and heart (IVC and right atrium)

Mr Cui's corrected calcium is 4.0mmol/L. How would you manage?

Sir this is severe hypercalcaemia (>3.5mmol/L). For severe hypercalcaemia or even if not severe but patient is symptomatic, I will

- Immediately begin aggressive hydration (1st line therapy, onset in hours) with 3L of saline a day
- Give calcitonin (onset in 4-6hrs, has tachyphylaxis after a few days)
- Consider bisphosphonate i.e. IV zoledronic acid or pamidronate (onset in 24-72 hours): used if due to excessive bone resorption e.g. malignancy related hypercalcemia, hypervitaminosis D etc.
- Glucocorticoid (onset in 2-5days): not applicable in this case, better for lymphoma, sarcoidosis, other granulomatous disease
- If all else fails - dialysis (onset in hours)

Mr Cui's CT Kidney shows a 12cm left renal parenchyma mass that enhances with contrast, extension into the renal vein stopping just before the IVC, but contained within the Gerota's fascia. No enlarged lymph nodes seen. No distant metastasis. How would you treat this patient?

This is a T3N0M0 tumour, it is resectable. I would counsel Mr Cui for a radical nephrectomy. This will involve en-bloc resection of the entire kidney with the Gerota's fascia together with its lymphovascular supply, as well as resection of the renal vein. Surgery is the mainstay of treatment. There is no role for adjuvant chemotherapy or radiotherapy.

Hepatocellular Carcinoma

PRESENTATION

Sir, my patient Mr Buang is a 43 year old Malay gentleman with hepatocellular carcinoma on a background of hepatitis B cirrhosis, currently admitted for transarterial chemoembolisation.

4 years ago, he was admitted for symptomatic ascites and diagnosed with cirrhosis secondary to hepatitis B. Since then he has had multiple admissions for decompensated liver cirrhosis, about 2-3 times a year. Triggers are usually fluid and salt indiscretion, or intercurrent illnesses such as upper respiratory tract infection or gastroenteritis. He has never had variceal bleeding. He is compliant to propranolol, frusemide and spironolactone, but misses his clinic appointments sometimes. He is not on antiviral medication at present and does not know his current Child's score, viral load or HBe status.

He was diagnosed with HCC last month when a follow-up ultrasound showed a small lesion which CT confirmed as HCC. He is otherwise asymptomatic with no abdominal pain, worsening jaundice, symptomatic ascites, encephalopathy, or variceal bleeding. There is no loss of appetite or loss of weight.

He does not have any family history of hepatocellular carcinoma. He has no other liver problems such as autoimmune liver or biliary disease. As for his underlying hepatitis B, his mother and siblings do not have hepatitis or cirrhosis. He does not have IV drug use and has not received blood products overseas. He declines to comment on sexual history.

He is currently admitted for transarterial chemoembolisation and is post-procedure day 2. He does not have any bleeding manifestations or fever, and is planned for home tomorrow.

He is functionally good and is able to work normally as a lab technician. Socially, he does not smoke or drink. He stays with wife and children in a hepatitis B flat. His wife is vaccinated against hepatitis B. They are coping well financially.

On examination, Mr Buang was alert and comfortable. He has peripheral stigmata of chronic liver disease such as marked jaundice, scleral icterus, spider naevi, loss of axillary hair, but no palmar erythema, clubbing, or gynaecomastia. There is no conjunctival pallor, no needle marks, no parotidomegaly or Dupuytren's contractures. The abdomen was not distended, had no scars, and was soft non tender even over the RHC. There was splenomegaly of 2cm below costal margin but no hepatomegaly. I did not find any shifting dullness or pitting lower limb edema. I note multiple tattoos over his arms and legs. There was no cough impulse over both groins, and I would like to complete my examination by doing a digital rectal examination.

In summary Mr Buang is a 43 year old Malay gentleman with recently diagnosed HCC on a background of hepatitis B liver cirrhosis, status post transarterial chemoembolization.

QUESTIONS

What are the ways in which HCC can present?

Sir, HCCs can

- Be completely asymptomatic and picked up only on screening (AFP/US).
- Have local symptoms such as right hypochondrial pain, either when the tumour grows fast enough to stretch the liver capsule or when it invades into the liver capsule.
- Precipitate decompensation of chronic liver disease
- Present with constitutional symptoms such as LOW LOA or symptoms of distant metastasis such as SOB or back/bone pain.
- Present with tumour rupture, with sudden severe abdominal pain, hypotension, and signs of peritonism.

How is HCC diagnosed?

HCC is usually diagnosed via surveillance ultrasound and/or AFP* monitoring of patients with chronic hepatitis or cirrhosis; that is the only way to diagnose in a timely fashion.

If the ultrasound finds a nodule >1cm, workup with further imaging modality is carried out - triphasic CT or dynamic contrast MRI. Classic imaging features (see below) is diagnostic of HCC. If the initial imaging features are not diagnostic, a second imaging modality (MRI with liver-specific contrast e.g. primovist, contrast enhanced ultrasound) or biopsy is carried out (otherwise, Biopsy is not routine due to risk of seeding)

A smaller subcentimeter lesion would be followed up on serial ultrasound and investigated if it is growing.

* AFP is routinely measured but not needed for diagnosis and insufficiently sensitive or specific (not all HCCs secrete AFP, and AFP can be elevated in the absence of HCC). AFP is now used more for trending on follow-up.

Please read Mr Buang's CTAP.

Sir, this is Mr Buang's CT scan. The arterial phase (contrast lights up in aorta) shows a 4 cm x 4 cm arterially enhancing lesion in the right liver. The venous phase shows the same lesion which is now hypodense, consistent with venous washout as is expected in HCC. I do not note any other lesions in the liver. The background liver is cirrhotic with nodularity, caudate lobe hypertrophy, and corkscrewing of hepatic arterioles. I do not note any ascites and I do not see any distant metastases in these cuts.

Mr Buang's albumin is 25, bilirubin is 40, and PT/PTT is normal. Please calculate his Child-Pugh Score.

A = 3 pts, B = 2 pts, C = 1 pt, D = 1 pt, E = 1 pt. Total 8 points, i.e. Child's B.

Scoring table:

	1 point	2 points	3 points
Albumin g/L	>35	28-35	<28
Bilirubin $\mu\text{mol/L}$	<34	34-51	>51
Coag: PT prolongation (INR)	<4 (<1.7)	4-6 (1.7-2.3)	>6 (>2.3)
Distension (ascites);	None	Controlled on diuretics	Refractory to diuretics
Encephalopathy	None	West Haven 1-2 1: Change in behaviour alone 2: Disorientation, drowsy, asterixes	West Haven 3-4 3: Marked confusion, mostly sleeping but arousable 4: Comatose

Interpretation: Child's A: 5-6 points, Child's B: 7-9 points, Child's C: 10-15

What is TACE?

TACE is the selective intra-arterial administration of chemotherapeutic agents followed by embolization of major tumour artery (principle: HCC draws vascular supply mainly from artery not hepatic vein). A guidewire is inserted from from groin (femoral artery) going up to the aorta and then into the hepatic artery. Contrast is used to localize the exact artery supplying the tumour. Chemotherapy and oily contrast (lipiodol) is then injected through a catheter, and the artery supplying the tumour embolized.

Suppose Mr Buang has just come back from TACE. You are now the HO reviewing him in the ward. How would you assess him, and what would you do for him?

The main concern is post-TACE syndrome which is usually self-limiting but there is a small risk of hepatic decompensation after TACE, especially if pre-morbid liver function is poor.

- I will get a set of vitals looking for fever.
- Ask for symptoms of right hypochondrial pain, nausea, vomiting. Ensure there is no malena or hematemesis (UBGIT is another cx of TACE)
- Examine general condition: drowsiness, asterixes; palpate abdo for RHC tenderness
- Order FBC UECr LFT PT/PTT; there may be transient elevation of AST ALT and bil.
- Order analgesia, antiemetics, and ensure that he is on antibiotics e.g. 5 days of ampicillin/sulbactam

Why do you think Mr Buang received TACE instead of another treatment option?

Sir, Mr Buang has a 4cm x 4cm nodule and is Child's B at present. Technically a single 4 cm nodule should be resectable. Perhaps there is another contraindication to resection such as poor liver function or inadequate future liver remnant volume should he be resected. Radiofrequency ablation would not be curative in his case as his nodule is too big.

General principles of management:

- Is this a good liver or bad liver? Assess liver function -
 - Child Pugh scoring: most liver surgeons will only be comfortable operating on a Child's A or a good Child's B; surgical morbidity is 40% for Child's C
 - Indocyanine green: Patient must have a r15 of >14% for major liver resection, and a r15 of >20% for segmental liver resection

- Is this a good tumor or a bad tumor?
 - Principle: A good tumor is a tumor that can be resected and yet leave sufficient functional liver for the patient to survive. Leaving insufficient functional liver behind can cause hepatic encephalopathy and liver failure.
 - A tumor with vascular invasion, metastasis is clearly a bad tumor. A multifocal tumor may be good or bad - if they are all on the same side of the liver which can be resected, then it is a good tumor; if all over the place, then bad.
 - Estimate future liver remnant volume: measured by CT volumetry = remnant liver / (current liver - tumor size). The larger the the tumour volume, the smaller the volume of functional liver removed. The minimum % of future remnant liver varies depending on function - about 25% for healthy liver, to 40% for cirrhotics¹

- Various algorithms of varying complexity are available.
 - Can I cure this patient? > Curative options are resection or RFA (if qualify³), or transplant (see note²).
 - If cannot cure > how best to palliate?
 - Motherhood statements: multidisciplinary care, discuss at tumor board... etc.

	Good Liver (Child A, early B) and patient fit	Bad Liver (Child late B or C) or patient unfit (comorbids)
Good tumor (resectable)	Curative resection RFA ³	RFA ³ Transplant: if within Milan ² criteria
Bad tumor: locally advanced	Transplant: if within UCSF ² expanded criteria Local-regional therapies e.g. TACE, SIRT ⁴	Transplant: if within UCSF ² expanded criteria Best supportive care
Extrahepatic metastasis	Sorafenib Best supportive care	Best supportive care

Footnotes:

1. Sometimes, to improve residual functional liver volume, can try portal vein embolization of the diseased lobe. Hope is that shunting blood supply to the contralateral lobe results in atrophy of the diseased lobe and hypertrophy of the normal lobe, improving future liver remnant volume.
2. Transplantation is a curative option for virtually all patients who fulfill the transplant criteria (below) and are fit for a big operation + can tolerate post-operative immunosuppression. It is considered especially in those who cannot be cured by resection or RFA. Unfortunately it is the hardest to come by simply due to lack of supply - donor organs are allocated on a waitlist prioritised according to MELD (model for end-stage liver disease) score. Transplant criteria are:
 - o Traditional milan criteria: 1 lesion no more than 5cm OR 3 lesions less than 3cm AND no lymphovascular involvement AND no distant metastasis.
 - o Can also be considered within expanded UCSF criteria (less strict).
3. Radiofrequency ablation (RFA): curative for lesions that are 3cm or smaller, and cannot be performed on lesions near any major vessels due to heat sink effect.
4. Locoregional therapy options include the following (all are palliative except RFA for a small tumor; and usually should not be offered to a child's C).
 - o Transarterial chemoembolisation (TACE)
 - o Selective Internal Radiation Therapy (commonly with Yttrium-90 microspheres)
 - o Ablative therapies, which include radiofrequency ablation, percutaneous ethanol injection, microwave ablation, cryotherapy

10: Colon Cancer

PRESENTATION

Sir, my patient Mr Mok is a 66 year old Chinese cook. He has no significant past medical or surgical history, and has good premorbid function.

He presents with a 1-month history of change in bowel habit, and loss of weight. In terms of his change in bowel habit, he notes a decrease in stool calibre and progressively watery stools in the last month which is non-bloody and non-fatty. This is associated with clinically significant loss of weight of 10 kg over 3 months. In the last week, he also complains of abdominal distension and mild colic over the central abdomen (pain score 1-3/10), without nausea/vomiting or constipation. There is no tenesmus and no severe unremitting pain that could suggest perforation. He has no per-rectal bleeding and no symptoms to suggest anaemia such as chest pain, dyspnoea, giddiness, palpitations, or lethargy. He also does not have any pneumaturia, fecaluria, or urinary tract infection. Systemically he is well with no jaundice or dyspnoea.

In terms of risk factors, he does not have any personal history of polyps, and no known family history of colorectal cancer or CRC-associated cancers. He had a colonoscopy 10 years ago which was clean, and no further fecal occult blood test or scopes since then.

He was diagnosed with colon cancer and is currently post-op day 4. He is systemically well and tolerating escalation to soft diet. He did not receive neoadjuvant chemotherapy and does not know if he is planned for adjuvant chemotherapy.

Examination revealed a pleasant middle-aged Chinese gentleman who is alert and comfortable at rest. He is not receiving any active interventions at the moment. There is mild temporal wasting, no pallor or jaundice. On inspection of the abdomen, there is a midline laparotomy scar, a surgical drain in the left iliac fossa connected to a closed active drain system and draining small quantities of hemoserous fluid, and a loop ileostomy in the right iliac fossa. I say that it is an ileostomy because it is draining liquid material, and the lumen size is <1cm; it is however less spouted than I would expect and I will like to remove the base plate to check for skin excoriations. Otherwise the stoma is pink. The abdomen is soft and generally non-tender, except for mild tenderness at the surgical scar, with no hepatomegaly or other organ masses felt. Hernia orifices are intact and there are no parastomal or incisional hernias.

Ideally I would like to complete my examination by performing a digital rectal exam however I feel that this may not be wise considering that it is POD4. I do not expect any findings as a loop ileostomy usually serves to defunction a left hemicolectomy or anterior resection; if he had a abdominal-perineal resection he would have had an end-ileostomy instead.

In summary, my patient Mr Mok is a 66 year old Chinese gentleman who presented with change in bowel habit, loss of weight, and symptoms suggestive of partial intestinal obstruction. He was diagnosed with colon cancer which is most likely left-sided, and underwent either a left hemicolectomy or anterior resection with defunctioning loop ileostomy.

QUESTIONS

You mention colorectal cancer associated malignancies, what are those?

These are seen in hereditary non-polyposis colorectal cancer, also known as Lynch Syndrome. This is an autosomal dominant syndrome due to defective mismatch repair (MMR) proteins. HNPCC is divided into

- Lynch syndrome I : familial colon cancer. Defined by Amsterdam Criteria, remembered as the 3-2-1 rule
 - At least **3** relatives with histologically confirmed colorectal cancer, 1 of whom is a first degree relative of the other 2.
 - At least **2** successive generations involved;
 - At least **1** of the cancers diagnosed before age 50.
 - Exclusion of FAP
- Lynch syndrome II : HNPCC associated with other cancers of the GI or reproductive system - namely endometrial cancer and, to a lesser extent, cancers of the ovary, stomach, small intestine, hepatobiliary tract, pancreas, upper urinary tract, prostate, brain, and skin.

OK what are your differential diagnoses in this patient?

Sir, my differential diagnoses for his presentation with intestinal obstruction include other infective and inflammatory pathologies of the colon. These include -

- Diverticular disease complicated by stricture, which can explain the decrease in stool calibre and spurious diarrhoea. However the loss of weight is somewhat unexpected, as is an extensive resection for the condition.
- Infective strictures such as that due to TB.
- Inflammatory bowel disease, especially ulcerative colitis, which can present with toxic megacolon
- Infective colitis

Note: please tailor your differentials to the patient's predominant presenting symptom e.g. intestinal obstruction, lower BGIT / iron deficiency anaemia, or change in bowel habit

Why do you think this patient's tumor is left sided?

This patient presents as intestinal obstruction which is consistent with that of a left sided tumours (in the right side the feces is more liquid, hence tends not to obstruct).

Localizing presentations:

- Left sided - change in bowel habits, decreasing stool calibre, eventually intestinal obstruction +/- bleeding.
- Rectal - tenesmus
- Right sided - tend to present more insidiously with just weight loss and anemia, and typically present with more advanced disease.

If you were the first doctor seeing him at A&E, how would you investigate this patient?

In the acute setting, my goals are to (1) rule out any emergent complications such as perforation or impending perforation, (2) diagnose the underlying cause IO, and (3) prepare for further management.

- First I will need to resuscitate the patient and ensure that he is hemodynamically stable.
- I would begin with simple blood investigations
 - FBC to look for microcytic hypochromic anaemia
 - UECr for electrolyte imbalance for diarrhoea and to ensure renal function for contrast scan
 - LFT to rule out liver dysfunction from extensive hepatic metastases
 - PT/PTT and GXM as pre-procedural investigations.
- My first line imaging modality will be erect CXR to rule out air under diaphragm, and supine KUB on which I expect dilated right-sided large bowel loops. Cecal dilation >9cm or the absence of dilated small bowel loops will be worrisome (impending perforation or close loop obstruction). I must also make sure that I don't see any Rigler's sign.
- The patient will subsequently require a CTAP

The patient will also require

- CXR or CT thorax for metastatic disease
- Colonoscopy to visualize the lesion and biopsy for tissue diagnosis.

When would you worry of impending perforation?

- RIF tenderness
- Diameter >3cm in small bowel, >6cm in large bowel, >9cm in cecum
- Pneumatosis intestinalis.

In this setting would you do CTAP first or colonoscope first?

Sir, in the emergent setting I do CT first.

1. *CT provides more information.* It allows me to confirm a tumor, rule out other causes of obstruction, and look for impending perforation i.e. pneumatosis intestinalis, diameter (or existing sealed perforation). Even if I am considering colonoscopic stenting, the CT helps see if the tumor is stentable (ascending colon, flexures are technically difficult to stent).

2. *CT is safer.* If there is impending perforation, the patient must be sent to OT immediately and not for colonoscopy. In such a situation a biopsy is unnecessary - surgical resection will provide the histology. Colonoscopy involves air insufflation which risks causing an iatrogenic perforation! I need to make sure there is no 9cm cecum or pneumatosis intestinalis before sending an obstructed patient to scope room.

3. *CT is faster.* Preparing the distal colon for a scope takes time and even then there is no guarantee that the tumor can be visualized. In the acute situation a CT scan is much faster.

In contrast in the elective setting (patient presents with PR bleed for investigation) - you would do colonoscopy first.

Suppose he indeed presented with an obstructed tumor, how would you manage him?

- First I will need to resuscitate him and rule out complications e.g. perforation (add in if not previously discussed).
- Will rule out other causes of obstruction such as hernia.
- I would prepare the patient for procedural intervention -
 - Non-surgical: colonoscopy with stenting; this converts an emergency procedure into an elective one.
 - Surgical: resection with primary anastomosis, proximal washout and defunctioning ileostomy. If patient is sick, Hartmann procedure is an option.

What is the benefit of colonoscopic stenting instead of immediate resection?

Sir, colonoscopic stenting may allow decompression of the obstruction and is of value in certain circumstances, assuming expertise and equipment is on standby

1. Interval stenting as a bridge to elective resection (2 weeks later). Benefits are:

- Avoid emergency surgery which has more complications
- To perform a one stage procedure without diverting ileostomy -- in the obstructed setting, a diverting ileostomy is important. This because the proximal bowel is distended and the distal bowel is collapsed, so there is an issue with matching lumen size while making anastomosis. The proximal bowel will also be edematous which results in higher rates of anastomotic leak later. (note: if rectal CA, will need defunctioning anyway)
- Allow completion of staging before op.

2. Advanced metastatic cancer: emergency laparotomy will defer chemotherapy at least 1 month. Emergency surgery will not change prognosis but palliative chemo can prolong survival -- so it helps to get palliative chemo fast.

However even if I prepare to stent the patient, I will prepare for laparotomy (standby EOT, call for blood, resuscitate pt) in case stenting is unsuccessful (15%) or there is a complication.

Would you give him bowel prep for colonoscopy?

Sir, my patient is obstructed, I cannot give him bowel prep which will make the obstruction worse. I do not expect to be able to scope past the obstruction and hence I can make do with giving the patient fleet enema to clear the distal bowel.

How do you stage him?

- I will stage him according to the T, N, M system.
- CTAP + CT chest or CXR is the first line modality to determine clinical stage. I am looking at the primary tumor in terms of size and depth of invasion to determine T stage (T1 = mucosal, T2 = muscularis, T3 = serosa, T4 = locally invasive) and for nodes to determine N stage (may not be obvious on scan, only confirmed post resection). The presence of hepatic or lung metastases signifies M1 i.e. stage 4 disease
- I am aware that stage 4 colorectal cancer is potentially still curative, e.g. if there is a single small live metastasis which can be resected.
- After operation pathological examination of the resection specimen allows determination of pathological stage, at which point decision on chemotherapy can be undertaken by a multidisciplinary tumor board.

Would you do CEA levels?

- Sir, I am aware of the utility of CEA levels as a tumor marker, particularly to monitor for relapse.
- But I would not do CEA for diagnosis.

How would you manage this patient if CT shows -

(a) Descending colon tumor, no obvious nodes, no mets?

- Sir I would want to achieve oncologic resection of the tumour with 5cm margins proximally and distally
- Likely op is left hemicolectomy
- with lymph node dissection (at least 20 lymph nodes to complete histopathological N staging)
- and primary anastomosis
- KIV defunctioning ileostomy
- If nodes are positive, patient will require adjuvant chemotherapy

(b) Rectal tumor 8cm from anal verge, no nodes, no mets?

- Neoadjuvant chemoRT (survival benefit - do for all tumors below peritoneal reflection i.e. <12cm)
- Oncologic resection of the tumour with 5cm proximally and 2cm distally. If the distal 2cm margin does not hit the anal canal (i.e. where the anal sphincters are) → sphincter saving surgery. If the distal 2cm hits the anal canal, then have to sacrifice sphincters and do APR. Length of anal canal can range 2-5cm from anal verge.
- Sphincter sparing surgery: anterior resection (anastomosis above peritoneal reflection >12cm) or low anterior resection (anastomosis below peritoneal reflection, 7-12cm), ultra-low (<7cm)
- With Total Mesorectal Excision (survival benefit)
- Followed by primary anastomosis
- And creation of a defunctioning ileostomy (if low AR)
- Adjuvant chemotherapy if nodes +ve

(c) Rectal tumor 3cm from anal verge, no nodes, no mets?

- Sir, as the tumour is too low to save the sphincter (minimum 2cm distal and 5cm proximal margins)
- Neoadjuvant chemoradiotherapy
- Abdominal perineal resection with creation of an end-stoma.
- Total mesorectal resection
- If nodes are positive, patient will require adjuvant chemotherapy

(d) Descending colon tumor with solitary liver mets?

- Intent of treatment can still be curative if solitary liver met is resectable.
- E.g. left hemicolectomy with lymph node dissection and primary anastomosis (as in the case without liver mets)
- Plus resection of liver met.

(e) Multiple liver mets

- Palliative chemotherapy
- If obstructed, endoscopic stenting
- Palliative symptom management

What is the purpose of loop ileostomy?

This is a defunctioning stoma to protect a distal anastomosis, especially for low anterior resection where blood supply is poorer and anastomotic leak rates are higher. It does not decrease the incidence of anastomotic leak however it reduces the morbidity of a leak.

Can you please counsel the patient about stoma?

- Indication: Mr Mok, you will be going for an operation to cut out a part of your intestine and join the other two ends. This joint can sometimes leak and to reduce the severity of complications, we intend to do a stoma for you
- Procedure: A stoma is when we take out a part of the intestine and connect it to the skin.
- What it means for patient: You will go home with a bag into which fecal material will drain. The bag is airtight so there is no smell. You will need to change the bag once it becomes half full, about 3-4 times a day. This is temporary, about 1-2 months later we will do a contrast X-ray to make sure the joint has no leak, and then we will put the intestine we took out back inside your tummy after.
- Site marking: We will choose convenient place to put the stoma, away from your belt line, belly button, or bone (ASIS); not too near the operation site.
- Risks:
 - In the first few days the output may be quite high and you may need top-up with intravenous fluids, but this usually gets better within the week.
 - Sometimes the skin can get a bit irritated but we try to minimise this by designing the stoma accordingly.
 - With time the opening can become a bit weak and the stoma can either sink inside or drop out more; if it doesn't interfere with the appliance we do not need to do anything, if it does then you will need a small operation to adjust the stoma

IO: Crohn's Disease

PRESENTATION

Sir, my patient Mr Ai Oh is a 60 year old Chinese man with Crohn's disease with recurrent admissions for intestinal obstruction and 2 previous small bowel resections in 2002 and 2010, presenting now with 2 days abdominal pain, abdominal distension and vomiting.

His abdominal pain began in the night after CNY reunion dinner where he had an enormous helping of mushrooms. It is generalized, colicky, non-radiating. He had 6 episodes of bilious non-bloody vomiting, and also has abdominal distension. He was still passing small amounts of stool and flatus at the time.

[Ddx for abdo pain] Otherwise, he has no fever, diarrhoea, jaundice, dysuria/pyuria, polyuria/polydipsia.

[Ddx for cause of IO] He has no change in bowel habits, decrease in stool calibre, PR bleed, tenesmus, LOW or LOA in the preceding months.

[Complications of IO] He has not had any sudden worsening of pain and extreme pain on movement to suggest perforation and peritonitis.

More on his past medical history of Crohn's:

- He first presented in his 30s with mucous-y diarrhea 20x day, with bloatedness relieved on passing motion. He was initially labeled as irritable bowel syndrome, however persistent symptoms prompted an OGD and colonoscopy, and Crohn's was diagnosed after biopsy found caseating granulomas.
- Since diagnosis till now he has only had 2 flares, each usually with fever, melena, intestinal symptoms like his first presentation and raised CRP.
- In between those flares he has grappled with recurrent small bowel IO due to strictures, averaging about 1-2 admissions a year mostly managed conservatively.
- Thankfully he does not have the other complications of the disease, such as:
 - Fistula formation - there have been no enterocutaneous or anal fistulas, nor any recurrent UTIs, pneumaturia, or fecaluria to suggest rectovesical fistula
 - PR bleeding
 - And colorectal cancer - last scope 3 months ago did not have any suspicious lesions or polyps

- I do not note any extra-intestinal manifestations - no joint pain (spondyloarthritis), tendon pain (enthesitis), eye problems (uveitis), skin rash or ulcers pyoderma gangrenosum, erythema nodosum), and no oral ulcers.
- In terms of medical management, he is steroid dependant (the two previous flares happening when attempts were made to wean him off), with steroid induced hyperglycemia. He also takes daily sulfasalazine and monthly infliximab injections which he is tolerating well.
- In terms of surgical management, he has had 2 small bowel resections in 2002 and 2010 for IO, complicated by steatorrhea and malnutrition (short gut syndrome). He is on vitamin supplementation.

He has no family history of inflammatory bowel disease.

And in terms of current functional, social and financial situation, he is single and stays alone, works as an administrator and has been able to support himself financially. He has a good understanding of his disease and generally feels he is coping well. He sometimes gets frustrated about the recurrent IOs but is still grateful that he is ADL-I and community ambulant.

On examination, Mr Ai Oh is alert and comfortable. He has being kept NBM, and has an NG tube on active suction, and on IV drip. He is thin but not cachectic, no temporalis muscle wasting. He has no conjunctival pallor or jaundice. He is not clubbed. I note a midline laparotomy scar and a paramedian scar in keeping with his previous surgeries. His abdomen is distended, soft and non-tender on palpation, no RIF mass or organomegaly, tympanic on on percussion, bowel sounds are sluggish but not tinkling. No incisional hernias or inguinal hernias. No enterocutaneous or perianal fistulas.

In the peripheries, I do not note any joint swelling, red eye, skin ulcers. I do not note any signs of cushing syndrome

I would like to complete my examination with a digital rectal examination looking for masses or PR bleed or fistulae.

In summary, Mr Ai Oh is a 60 year old Chinese gentleman with refractory, steroid-dependant Crohn's disease who has had recurrent admissions for IO and 2 small bowel resections in 2002 and 2010, presenting once again and symptoms and signs of intestinal obstruction.

QUESTIONS

Why was he initially still passing stool if this is IO?

Sir, in a proximal IO, vomiting occurs early and constipation occurs late; so this presentation is not inconsistent with distal IO.

What are the possible causes of IO in this patient?

Sir, causes of IO in this patient can be disease related and non-disease related.

- Related to Crohn's, he could have inflammatory strictures or development of a colorectal tumour as well as post-op complications such as anastomotic strictures, adhesions and incisional hernias.
- Unrelated to Crohn's, he could have bezoars from high fibre food such as Chinese mushrooms. I looked for but did not find any inguinal hernia.

You are HO on call. What are you going to do?

- FBC UECr PT/PTT GXM
- Erect CXR looking for free air under diaphragm
- Supine AXR looking for intestinal obstruction, and what type
- Barium meal and follow through looking for site of obstruction
- Drip and suck, keep NBM, NGT, IDC in interim.

Please interpret this patient's AXR.

- Sir, I see dilated bowel with plicae circularis and 'stack of coins' appearance located centrally. This is small bowel obstruction.
- OR Sir, I see dilated large bowel loops located peripherally. There are no dilated small bowel loops, therefore ileocecal valve is competent. There is no rectal gas. I am worried about a closed loop obstruction of the large bowel.

What are the indications for surgical management of IO in such a patient?

It is important to aim to avoid surgery until absolutely necessary, as repeated small bowel resections can lead to short gut syndrome (like what Mr Ai Oh has) which in turn causes problems like steatorrhea and malnutrition. The indications Mr Ai Oh might have had for bowel resection include failure of response to medical therapy, closed loop obstruction or impending perforation, perforation of bowel, long segments of strictures >12cm (be it due to Crohn's itself or from the surgical anastomosis). Mr Ai Oh has not had other indications such as fistula or abscess formation and malignancy.

Let's say this patient came in with a flare. How would you investigate?

Sir, I would investigate with the aim of (1) confirming diagnosis and ruling out differentials (2) looking for complications (3) guide management

I will start with basic investigations such as:

- FBC looking at raised total whites and Hb for anemia
- RP looking at hypokalemia and dehydration from prolonged diarrhea
- ESR, CRP for disease activity
- LFT for hypoalbuminemia due to poor nutritional intake and also as baseline before initiation of therapy
- Stool cultures and ova cysts parasites
- Fecal elastase, fecal calprotectin

Then I will bowel prep patient and prepare for colonoscopy and OGD

Lastly, can do TB spot and hepatitis screen (TRO TB, hepatitis before initiation of steroids or immunotherapy)

How else would you manage the patient?

Refer to gastroenterologist for optimization of his medical therapy. Our patient Mr Ai Oh has refractory disease, as he is unable to be weaned off corticosteroids. This is the reason why he is on monthly infliximab injections, in addition to sulfasalazine and corticosteroids at the moment. (Refer to medical case > IBD)

OK how about surgically, how would you follow up Mr Ai Oh when he sees you in clinic?

Mr Ai Oh is at elevated risk of colorectal malignancy and therefore needs surveillance colonoscopy. Recommendations differ but MOH recommends for pancolitis, to begin scopes 8 years after onset of symptoms and thereafter every 1-2 years.

How does Ulcerative Colitis differ from Crohn's disease?

In ulcerative colitis, there is superficial inflammation and ulceration limited to mucosa of the large bowel, It always involves the rectum but may extend in a proximal and continuous fashion to involve other portions of the colon and occasionally even the terminal ileum (backwash ileitis). In Crohn's, there is transmural inflammation with deep ulceration, fistula and abscess formation. It occurs throughout the GIT from mouth to anus but in skip lesions with normal mucosa in between, and typically spares the rectum.

On endoscopy, Crohn's has a cobblestone appearance with deep ulcers and fissures whereas ulcerative colitis has shallow ulcers, pseudo-polyps and mucosal bridges.

Medical therapy for UC, like Crohns, consist of 5-ASA, glucocorticoids, immunomodulators like azathioprine and biologic/immunotherapy like infliximab.

Surgical Management of UC:

- Emergent Indications
 - Acute fulminant colitis with acute abdomen Toxic megacolon (colon > 5.5cm)
 - Impending Perforation (i.e. dilatation with thumb-printing or pneumatosis) or
 - Free/walled off perforation
 - Acute fulminant colitis without acute abdomen unremitting bloody diarrhoea→ Total colectomy + end ileostomy
- Elective Indications
 - Disease refractory to medical therapy with severe & extensive colitis (most common)
 - Serious complications of medical therapy
 - Malignancy – precancerous lesions or prophylactic risk reduction
 - Debilitating extra-intestinal manifestation – i.e. thromboembolic complications→ Proctocolectomy + ileal pouch anal anastomosis

Jaundice, Painful: Cholangitis

PRESENTATION

Sir, my patient Mdm Liew is a 50 year old Chinese lady with past medical history of DM, HTN, HLD. She now presents with 2 day hx of fever, jaundice and right hypochondrium pain.

This started 2 days ago with sudden onset right hypochondrium pain, sharp and constant, not radiating to back or shoulder and not better sitting forward. She also had jaundice, tea coloured urine, and lightening of stool colour. Her fever started the next day with Tmax of 38.0, chills and rigors. She has also had 4 episodes of non-bilious non bloody vomiting.

In terms of etiology,

- [Gallstone disease] I note that she has longstanding post-meal dyspepsia but has never been investigated with ultrasound or OGD.
- [Malignant] There is no loss of weight...
- [Stricture] ... and no past ERCP (past - stricture, recent - iatrogenic).
- [Metabolic cause of gallstone] She does not have any chronic hemolytic anaemia (e.g. thalassaemia) or mechanical heart valve replacement.

In terms of differentials, I note that she has had raw oysters last week and has not been vaccinated against viral hepatitis. She is in a stable monogamous relationship, has no hx of IV drug abuse, and has never received blood products from outside singapore.

Other than DM HTN and HLD, all of which are well controlled on medications, she does not have any other past medical or surgical history. She has no known drug allergies.

Socially, she stays with her husband and children in a HDB flat, works as a business executive and is financially well to do.

On examination, Mdm Liew is alert, comfortable. She is currently receiving IV fluids as well as IV ceftriaxone and metronidazole. She has scleral icterus but no conjunctival pallor. Her urine bag is draining tea coloured urine. Abdomen was not distended. There is tenderness and guarding over the right hypochondrium. Otherwise the rest of the abdomen is soft and non-tender, no signs of rebound or guarding. Murphy's was negative. There was no organomegaly. Bowel sounds were present. Cough impulse over bilateral groins were negative. Breath sounds were clear and equal bilaterally. Heart sounds were dual, no murmurs, pulse was regular at 95. I would like to complete my examination by doing a digital rectal examination looking for PR bleed or melena.

In summary, this is a 50 year old Chinese lady presenting with 2 days of fever, jaundice and right hypochondrial pain on a background of dyspepsia. My top differential is acute cholangitis, but I would like to consider hepatic abscess, acute cholecystitis, acute hepatitis, and acute pancreatitis.

QUESTIONS

How would you investigate?

Sir, I need to investigate to confirm my diagnosis, rule out differentials and find the etiology.

Initial bloods include

- FBC for raised TW
- LFT for cholestatic picture of raised LFT in cholangitis; hepatocellular picture if hepatitis.
- UECr for prerenal AKI since patient has been vomiting / to guide fluid therapy
- Blood cultures
- CRP, procalcitonin

For differentials, I will do

- Amylase, lipase to look for concomitant pancreatitis (if present, to do full glasgow bloods).
- Hepatitis viral serologies for viral hepatitis

Imaging modalities include

- US HBs for dilated CBD, evidence of gallstone disease
- CT abdomen for the same especially overnight where US cannot be easily obtained.

You are the HO on call. How would you manage?

Sir, cholangitis is a surgical emergency. I would see the patient immediately and escalate to my senior. My goals are to (1) Resuscitate the patient, (2) Initial investigations to confirm dx, rule out ddx, look for etiology, (3) Initiate medical therapy, (4) Send patient for emergent decompression, (5) Treat underlying condition.

(1) Resus

- A Assess airway
- B Ensure breathing, saturation
- C Take vitals
 - 2 large bore IV plugs
 - Aggressive fluids as I expect the patient to turn septic - 2 pint normal saline fast
 - Keep NBM
 - Insert IDC
 - Transfer HD/ICU
 - Chart: strict I/O, Q1h paras + SpO2

(2) Confirm diagnosis and rule out differentials

As discussed above.

(3) Initiate treatment

Empiric IV antibiotics with gram negative and anaerobic coverage - 3rd gen cephalosporin like ceftriaxone, and metronidazole (alternative: augmentin plus gentamicin/amikacin, plus metronidazole).

(4) Send patient for emergent decompression of the biliary system

Either by ERCP and stenting (or stone retrieval with sphincterotomy) or PTC

(5) Treat underlying cause (definitive treatment)

If ERCP → Early cholecystectomy

If PTC → ERCP → Cholecystectomy OR

If PTC → cholecystectomy with intra-op cholangiogram, CBD exploration and T tube

What is a normal ERCP?

- Normal intrahepatic ducts
- Smooth CBD
- No narrowing of the CBD
- No filling defects
- Free flow of contrast into duodenum

What are the contraindications to ERCP and what is the alternative?

- Patient is too sick for ERCP (cannot be put in prone position, not fit for anesthesia)
- Abnormal anatomy (previous gastrectomy, bilroth, roux-en-y, etc)
- Coagulopathic → can opt for stenting, forgo sphincterotomy
- Previously failed ERCP (relative)

The alternative is percutaneous transhepatic biliary drainage.

In the context of definitive stone removal after PTC, what are the indications for operative removal of stone (instead of ERCP)?

- Stone >25mm
- Intrahepatic stone
- Large number of stones
- Impacted stone
- Dual pathology
- Tortuous duct
- Previous Billroth II (unsuitable anatomy for ERCP)

Please counsel Mdm Liew on ERCP.

Mdm Liew, I am Dr Tsang. I'm going to explain to you (1) your current condition (indication) (2) What is a ERCP and why we recommend it (3) risks and complications of ERCP (4) risk of not going for ERCP and alternative. Do you have any other questions or concerns you would like me to address along the way? If not, still feel free to stop me any time along the way to clarify anything you don't understand.

*I recommend drawing the HBS system in this case!

(1) Your current condition

You have stones in your gallbladder, and one has dropped out into the bile duct. This stone is blocking flow of bile from the liver to the intestine. The blockage has caused infection → this is called cholangitis. Cholangitis is not something we can take lightly, because the bacteria can spread everywhere in your body and you can become very sick very quickly. Therefore it is very important that we take out the stone and relieve the obstruction, as well as treat the infection with antibiotics.

(2) ERCP

Removal of the stone is commonly done through ERCP, which stands for a very long name - "Endoscopic retrograde cholangiopancreatogram". First we give you sedation so that you don't feel any pain. Then we put a camera through your mouth into your intestine. From there, we either remove the stone, or put a stent so that bile can flow.

(3) Risks and complications of cholecystectomy

(a) Risk from sedation

Can get heart attack and stroke, but there is a lower risk of this as you have no previous heart or blood vessel problems

You have to be very closely monitored during the procedure.

(b) Risk from procedure itself

Perforation of bowel

Bleeding, esp from sphincterotomy

Infection

Pancreatitis

Failure of ERCP

(4) Risk of not going for cholecystectomy and alternatives

The alternative method to relieve the obstruction is percutaneous transhepatic cholangiography. This is where we insert a tube through the skin and through the liver into the bile duct to allow the infected bile to drain out. However this is just a temporary measure as it cannot remove the stone. This still has to be followed up by an ERCP or an operation to remove clear the stone from the bile duct + remove the gallbladder at the same time.

The last option is to not do anything, but the risk of that is the infection will spread to your whole body and can lead to death.

Please counsel Mdm Liew on cholecystectomy.

Refer to Acute Pancreatitis

Mdm Liew is very worried about whether this could be pancreatic cancer. She heard that you can do a blood test tumor marker to tell her if it is so. What do you say?

CA19-9 is a tumor marker for pancreatic cancer but it is also elevated in other diseases of the biliary tree such as cholangitis, gallstones. It is not specific and can be elevated in other cancers (gallbladder, cholangioCA); so it is generally unwise to do CA 19-9 in the setting of acute cholangitis.

Suppose Mdm Liew goes for the cholecystectomy but comes back again 3 months later with cholangitis once. US HBS shows stones in the CBD. What are you suspecting?

Recurrent pyogenic cholangitis

Jaundice, Painless: Pancreas CA

PRESENTATION

Sir, my patient Mr Huang is a 68 year old Chinese gentleman who presents with 1 month of painless progressive jaundice and significant loss of weight.

In terms of his jaundice, his relatives first noticed that he was yellow 1 month ago, during Chinese New Year visitations. This was not associated with any abdominal pain. He has noticed tea-coloured urine and lightening of stool colour, and also complains of pruritus. There is clinically significant weight loss of 10kg over 3 months. [Cx] There is no steatorrhoea or new onset polyuria/polydipsia (DM)

[Ddx / etio]

- He does not have fever or right hypochondrial pain to suggest cholangitis.
- He has never had post-meal dyspepsia or known gallstone disease.
- He has no previous instrumentation to the biliary tree, and no stay overseas in a rural country to suggest strictures.
- I also considered hepatic causes of jaundice, however he does not have any known chronic liver disease, chronic hepatitis, no IV drug use, alcoholism, or high risk sexual exposure.

In terms of family history, he has no family history of periampullary, biliary tree, or gastric cancer.

His other past medical history includes well-controlled DM, HLD, and IHD on aspirin. He also has L4/5 disk disease and BPH. He has no past surgeries and no known drug allergies.

Socially, he is a retiree who stays with wife and children. He is comfortable financially. He does not smoke or drink and is ADL-independent and community ambulant.

On examination, Mr Huang was alert and comfortable. He appears slightly cachectic with wasting of temporalis muscle and intrinsic hand muscles. I note that he is jaundiced and has scleral icterus. There are no stigmata of chronic liver disease such as clubbing, palmar erythema, asterixes, loss of axillary hair, or telangiectasia. On examination of the abdomen, there were no surgical scars, distension, or herniae. It was soft non tender. There was a palpable gallbladder. I say that it is a gallbladder because it is a globular shaped right hypochondrial mass, dull to percussion, moves inferiorly with respiration, and I cannot get above it. There was no other organomegaly and no shifting dullness. I looked for but did not

find cervical or supraclavicular lymphadenopathy. I would like to complete my examination by performing digital rectal examination to look for stool colour.

In summary, Mr Huang is a 68 year old Chinese gentleman who presents with painless progressive jaundice, palpable gallbladder, and significant loss of weight.

QUESTIONS

What are your differential diagnoses?

- My first diagnosis is a periampullary cancer e.g. head of pancreas, ampulla of Vater, or distal CBD.
- Differential diagnosis is gallstone disease but this is less likely given lack of pain or fatty dyspepsia.
- I also considered hepatic causes of jaundice such as decompensated cirrhosis or hepatitis however these were less likely.
- Other causes of obstructive jaundice include strictures and extrinsic compression by porta hepatis lymph node

What is Courvoisier's law? What are the exceptions?

Courvoisier's law states that jaundice in the presence of a palpable gallbladder is unlikely to be due to stones. This is because the gallbladder with stones is likely to be chronically fibrosed and shrunken. Exceptions include Mirizzi's syndrome (stones impacted in Hartmann's pouch of cystic duct compress on the common hepatic duct), primary ductal stone formation (recurrent pyogenic cholangitis), mucocele formation.

How would you investigate this patient?

For this patient, my goals are to (1) establish the diagnosis, (2) consider ddx.

I will start with blood investigations including

- LFT - for cholestatic pattern of derangement, albumin for nutrition state
- FBC - Hb (anaemia), Plt (thrombocytopenia), TW (cholangitis)
- UECr - as contrast studies will be done
- PT/PTT - measure hepatic function
- Glucose - measure hepatic function
- Tumor markers i.e. CA 19-9

I understand is that US HBS is the usual first line investigation for obstructive jaundice, but as my suspicion for CA is high, I would like to go straight for a contrasted CT abdomen looking for dilated biliary ducts, double duct sign, causes of obstruction, local invasion of the superior mesenteric vein or artery, regional spread to para-aortic lymph nodes or distant spread. This is usually followed up by a more detailed imaging scan such as CT or MRI pancreas to further delineate anatomy. Patient would also need a CT thorax for staging.

Whether to biopsy the lesion for histological confirmation depends on the index of suspicion for malignancy, surgeon and patient preference (*generally no need unless uncertain of dx, patient unwilling to accept risk of negative whipples - if do, use endoscopic ultrasound guided*).

Would you relieve the obstructive jaundice in this patient?

Sir, I would not relieve the obstruction in this patient. He is not cholangitic and does not have severe symptoms of coagulopathy, encephalopathy, or pruritus. I am aware that tumor obstruction is usually sterile and stenting may cause contamination and make subsequent tissue dissection more difficult. However if he develops cholangitis then definitely he requires a drain.

Can you read this CTAP

Sir there is a large mass in the head of pancreas not involving SMA or SMV. I see a double duct sign with enlarged gallbladder. I do not note any lesions in the liver and there is no ascites. I do not note obvious para aortic nodes. The liver does not appear cirrhotic. There are no other metastasis to the liver, lung or peritoneum.

OK so given his CTAP how will you manage?

Sir, my patient is fit and his tumor is resectable. I want to offer curative therapy yet knowing that 5-year survival after Whipple's is around 10%. I will discuss him at a multidisciplinary tumor board with a view towards Whipple's procedure and adjuvant chemotherapy. Prior to surgery I will optimise his nutritional state.

General principles

- Resectable = no SMA invasion, no paraaortic lymph nodes involvement, no mets. (SMV is resectable but difficult). 80% of patients are not suitable for curative resection.
- If resectable, Whipple's then adjuvant chemotherapy
- Unresectable or distant mets or unfit:
 - Relieve GOO: stenting
 - Relieve obstructive jaundice: stenting vs feeding jejunostomy
 - Nutrition support, enzyme replacement for steatorrhea
 - Surgical triple bypass (gastrojejunostomy, hepatojejunostomy, jejunojejunostomy) is an option to relieve both GOO and obstructive jaundice, but is now not commonly done due to the advancement of endoscopic stenting technique.

OK a Whipple's performed and the tissue is sent for histology. What are the possible types of periampullary cancer?

Sir, the periampullary cancers include carcinoma of head of pancreas, ampulla of vater, and distal cholangiocarcinoma. All can present as obstructive jaundice.

How about a body or tail of pancreas tumor? How will this present?

Sir, a body or tail of pancreas tumor does not cause obstructive jaundice. It often presents late as back pain, mass, loss of weight, incidental finding on CT scan, new-onset diabetes, or paraneoplastic manifestations (e.g. migratory thrombophlebitis).

Tell me about cholangiocarcinoma.

Cholangiocarcinoma arise from the bile duct and may be intrahepatic, Klatskin, or distal CBD. Distal tumors may cause obstructive jaundice but intrahepatic tumors do not, presenting instead with vague symptoms of RHC discomfort, hepatomegaly, and weight loss. ALP +/- bilirubin may be raised. Patients with primary sclerosing cholangitis are at particular risk. Diagnosis is via MRCP for proximal lesions and EUS for distal lesions, and the task in intrahepatic lesions is to distinguish from HCC.

Leg Pain: Claudication, Critical Limb

PRESENTATION

Mdm Tan is a 66 year old known vasculopath with claudication status post arterial bypass. She now presents with rest pain and tissue loss of her left lower limb for 2 weeks duration.

With regards to her known peripheral vascular disease, she was first seen by a vascular surgeon 2 years ago. At that time she had a 2-year history of claudication symptoms, worse on exertion, worse walking uphill than downhill, with no back pain or change with spine flexion or extension. Her function then was good and claudication was not disabling. She was managed conservatively for a year with risk factor control and exercise.

Over that year, however, her claudication distance decreased from 2 bus stops to barely 100 meters which severely affected function. Pain was worse on the right than on the left. She underwent balloon angioplasty followed by arterial bypass to the right lower limb, with significant symptomatic improvement and good function.

In the past 2 weeks, however, her claudication distance has fallen from 1.5 bus stops to 0.5 bus stops and she has experienced symptoms of critical limb ischaemia. She complains of rest pain over the dorsum of bilateral feet and lateral aspect of calf, severe enough to wake her up, however pain was not better when leg put in a dependent position. She also noted a new ulcer over the back of her left heel, which was dry, no discharge, no fever. She also has paresthesia over L LL over the past 3-4 months, with no weakness.

Since admission she has undergone balloon angioplasty to her left lower limb, with significant improvement to pain.

In terms of vascular risk factors, I note that she has a longstanding history of diabetes mellitus, hypertension, and hyperlipidaemia. She is maintained on increasing doses of oral medications which she appears to be unfamiliar with and poorly compliant to. She does not know her HbA1c or home glucose and blood pressure readings. In terms of vascular complications, she has had a myocardial infarction 3 years ago, status post percutaneous coronary intervention. She does not know her 2DE findings. I do not note any renal disease or cerebrovascular disease. Her medications include aspirin, oral hypoglycaemics, antihypertensives, and statins which she is unable to provide me the names of. She does not smoke.

In terms of function, she stopped working due to her vascular claudication symptoms a few years ago. She is however still ADL independent and community ambulant with a walking stick, until these two weeks.

Social wise, she is a widower who stays with the oldest of four children. Social and financial support is good from 4 children.

On examination, Mdm Tan was comfortable and alert. On inspection I noted a 4cm x 4cm round ulcer with gangrenous edges and visible extensor tendon over the back of her left heel. This was clean with no discharge and no surrounding erythema. Bilaterally there were multiple healed scars and arterial skin changes such as shiny hairless skin in both lower limbs. On palpation, the R LL was colder than L LL below the knee. Capillary refill time in R LL was 3s, and <2s in the L LL. DP, PT, popliteal, femoral pulses all felt bilaterally. Buerger's test was negative. There were no varicose veins and no loss of pinprick sensation bilaterally.

Dressing over the right femoral access site was dry, no hematoma. I looked for but did not find any AAA. Cardiovascular examination found first and second heart sounds with ejection systolic murmur over aortic region radiating to the carotids. I would like to complete my examination by measuring her blood pressure and ankle brachial pressure index.

In summary, Madam Tan is a 66 year old vasculopath currently admitted for L LL critical limb ischemia status post balloon angioplasty.

QUESTIONS

What are your initial investigations?

Sir, I need to investigate to (1) confirm arterial disease, (2) look for complications, and (3) plan for surgical intervention

I would start off with the following basic blood investigations:

- Full blood count, looking at whether her total white count is raised which may suggest infection of her heel wound, and also at her platelet count as she is going for an invasive intervention
- Renal panel, with an eye on the Cr especially knowing she is likely going to need contrast
- Pre-operative investigations: PT/PTT, CXR, ECG, 2DE
- Risk factors: HbA1c, fasting glucose, fasting lipids, if none were done recently

To confirm arterial disease, I will like to do

- Ankle brachial pressure index as very basic assessment to confirm critical limb ischaemia and also a reference point to compare post-op and quantify improvement.
- Ultrasound duplex of her lower limb arterial system to trace the arterial anatomy, look for stenosis, look at flow velocity and pattern i.e. monophasic/biphasic/triphasic, as well as to plan for angiography.

Her ulcer is clinically not infected at present and therefore I will withhold workup for infection i.e. wound swab and culture, foot X ray (to exclude OM), CRP and blood cultures.

How do you interpret the ABPI?

- Normal ABI is > 0.9 (can be > 1.0 as ankle pressures tend to be higher than brachial)
- ABI between $0.5 - 0.9$ – occlusion, often associated with claudication
- ABI < 0.5 : Critical limb ischaemia
- If > 1.40 , suggests non-compressible calcified vessel esp. seen in DM patients
→ Do Toe pressures index (TPI) instead (an abnormal TBI is < 0.70)

How would you manage Mdm Tan?

Sir Mdm Tan has critical limb ischaemia. My goals are to (1) revascularize, (2) modify risk factors, and (3) rehabilitate.

I would counsel for surgical management to her left lower limb (her previous bypass was to the right limb). *Look at the scans (inflow i.e. aortoiliac, outflow i.e. femoral, and runoff i.e. below knee) and decide, for example reasonable answers are-*

- She has a short focal segment of stenosis which is amenable to endovascular intervention such as angioplasty +/- stenting. However I think her life expectancy likely exceeds 2 years and hence I will suggest bypass surgery as the initial treatment because of better long-term outcomes in spite of higher immediate morbidity (BASIL study).
- She has extensive disease with long segment stenosis but has good runoff vessels, I would opt for femoral-popliteal bypass grafting.
- She has inflow (aortoiliac) disease and hence my first task will be to do aortobifemoral bypass.

In terms of risk factor control I believe that this patient needs multidisciplinary care with medical colleagues such as a cardiologist and endocrinologist, as well as allied health professionals like a dietitian. It is important to optimise blood pressure, lipids, and glucose control, as well as to treat her comorbidities.

After surgery I will send her for rehabilitation with supervised exercise to maximise function. I will also maintain her on an antiplatelet e.g. aspirin 100mg or clopidogrel 75mg (*? role for medications such as cilastazol, a type III PDE inhibitor which inhibits platelet aggregation and causes vasodilatation*). I will emphasize compliance to therapy otherwise she is sure to restenose and get critical limb ischaemia again!

While awaiting surgery what will you do?

- Give analgesia
- Optimise diabetes and blood pressure control in the ward
- Give high dose statin if not already on.
- Start aspirin - recommend to continue during perioperative period.

If she refuses surgery what is her prognosis?

At one year, there is a 50% chance she is alive with two legs, 25% chance of one leg amputated, and 25% chance of death due to cardiovascular disease.

When will you amputate?

Sir I will amputate if the limb is

- Dead (ischemic): peripheral vascular disease (80-90% of all cases)
- Damaged (trauma): unsalvageable limb, burns
- Dangerous: Gangrene, ascending sepsis, malignancy (soft tissue / bone)

Leg Pain: Venous Insufficiency

PRESENTATION

Mdm Latifah is a 49 year old Malay lady with bilateral leg swelling and discomfort at the end of each day, and a nonhealing right leg ulcer which has become infected in the past week. If I may discuss each of her complaints in turn -

She gives a chronic history of bilateral leg swelling and discomfort of two years duration. This is not present when she wakes up each morning, occurring only at the end of each day, especially on work days where she stands for long hours as a McDonalds counter staff. Discomfort is mild and aching in nature; there is no pain radiating from the back to her legs, and no exacerbation of discomfort on walking. Swelling and discomfort is relieved when she gets home and props her legs up to watch TV. She has also tried wearing stockings with mild relief.

Moving on to her ulcer, in the last 3 months she developed an ulcer just above the medial malleolus on her right leg, which has not healed since then. This is the first ulcer she has ever had. It was associated with bilateral varicosities which Mdm Latifah has noticed for a year. The ulcer was initially painless and did not bother my patient. She has attempted to apply some powder provided by a traditional medicine physician without success. However in the past week she has developed a low-grade fever, and has noted a foul smelling discharge from the ulcer which stains her stockings.

I looked for the presence of concomitant arterial disease and note that Mdm Latifah has no symptoms of vascular claudication or tissue loss. She is however a vasculopath with poorly controlled diabetes of HbA1c 8.8%, hypertension, and hyperlipidemia. She is also morbidly obese. I note that she is on glipizide, metformin, nifedipine, as well as a statin; she is however poorly compliant to her medications.

In terms of other contributory factors to bilateral lower limb edema, I note that she is on nifedipine which can contribute to pedal edema. Otherwise there was no history of cardiac or renal or liver disease, and systemic review found no shortness of breath on exertion, angina symptoms, frothy urine, and no heat or cold intolerance to suggest thyroid disease.

She also does not have any prior episodes of deep vein thrombosis. Apart from what I have mentioned there is no other past medical history or drugs or drug allergy. There is no family history of varicose veins.

Socially, she is married with five children, and stays in a HDB flat on a lift landing floor with her husband and children. She is still able to work in McDonalds but the discomfort at the end of the shift troubles her. She has attempted to lose weight unsuccessfully, and enjoys staff meal and unlimited coke provided by McDonalds at least 2 meals a day. She does not smoke or drink. She is coping alright financially.

Examination found a pleasant Malay lady, alert and comfortable at rest, quite obese. On inspection of the lower limb, there was a 4cm x 3cm ulcer on the gaiter region of the right leg which appeared shallow, with well defined edges, a base with slough, and a weeping yellow discharge. There are no heaped edges or fleshy appearance to suggest Marjolin's ulcer.

I note bilateral venous skin changes of hyperpigmentation and lipodermatosclerosis, as well as bilateral varicosities in the great saphenous vein distribution. Tourniquet test found that there is incompetence distal to the saphenofemoral junction. Lower limb pulses were well felt and capillary refill time was less than 2 seconds. On palpation of the abdomen there was no pelvic mass.

In summary, my patient is a 49 year old Malay lady with bilateral chronic venous insufficiency complicated by infected venous ulcer. I do not note any evidence of concomitant arterial disease.

QUESTIONS

What CEAP grade is she?

Sir, she has an active ulcer so she is CEAP 6.

What are this patient's risk factors for venous ulcer?

- Long periods of standing
- Obesity
- Multiparity

How do you distinguish venous vs an arterial ulcer?

See Approaches to Symptoms of Disease

What investigations will you order?

Sir, my goal will be to (1) guide treatment of infection, (2) diagnose venous disease, (3) exclude arterial disease.

(1) FBC, CRP, blood and wound c/s. (If can see bone, Xray)

(2-3) ABPI, arterial duplex and venous duplex ultrasound.

OK you confirm pure venous disease. how will you manage her?

Sir, in the acute setting my priority is to treat infection and to relieve venous hypertension. In terms of treating infection, I would start antibiotics, initially broad spectrum then culture guided. I will also give wound care and analgesia.

To relieve her venous hypertension and aid wound healing, I will give four layer compression bandage after excluding arterial disease (very important - if resting arterial pressure is systolic 75mmHg and the compression bandaging exerts 25-30mmHg, you are going to make the patient 45mmHg i.e. tip the patient over into critical limb ischemia).

I may also offer her surgery for underlying venous insufficiency, I know that this does not accelerate ulcer healing but reduces recurrence (ESCHAR study). Surgical options include endovenous radiofrequency ablation or traditional open surgery (high tie, stripping, and avulsion). Thereafter she will need stockings for life.

Say this patient was lost to follow up and came back to you 3 years later. The ulcer is now bigger and more fleshy. What would you do?

Sir, in a long standing non-healing ulcer I would be wary of Marjolin's ulcer i.e. malignant transformation into squamous cell carcinoma. I would need to take a biopsy of the edge of the ulcer.

A more detailed discussion is found under venous short case as veins appear more commonly as short cases.

LUTS: BPH & Prostate Cancer

PRESENTATION

Sir, my patient Mr Pang is a 63 year old Chinese gentleman who presents with 2 days of fever, dysuria, frequency, and later acute urinary retention, on a background of a few months of voiding type lower urinary tract symptoms.

In terms of his acute presentation, in the last 2 days he has been passing small amounts of urine q15minutes, with urgency and dysuria, even throughout the night. In the 6 hours before he came to the A&E, he was not able to pass urine and developed suprapubic tenderness. At A&E he was noted to be febrile at 38.5 degrees. There was no pyuria or hematuria, back or loin pain. There is no painful ejaculation to suggest prostatitis.

He has a background of voiding type of lower urinary tract symptoms for 5 months. This is mainly poor and intermittent flow but otherwise no hesitancy or sensation of incomplete voiding or double voiding, and no storage symptoms such as frequency, urgency and nocturia. GP started alpha blockers, to some effect. He has not had any side effects such as postural giddiness.

Other than BPH, he does not have other risk factors for urinary tract infection such as kidney or bladder stones, or known congenital abnormalities of the urinary system. *He is in a stable monogamous relationship and I do not note any high risk sexual exposure.*

I do not note any other possible precipitants of acute urinary retention such as constipation, new medications, neurological disease, painful ejaculation (prostatitis) or recent instrumentation to the urinary tract.

A second issue is that the GP found that he had high prostate specific antigen, but Mr Pang has not decided whether to biopsy. There are no red flags such as painless gross hematuria, loss of weight or appetite, shortness of breath, bone pain or back pain, or neurological symptoms. He does not have a family history of prostate cancer.

He has no other medical or surgical history, the only long term medications he is on are for his BPH, and he has no drug allergies.

In terms of social, functional and financial history,

- He stays with wife and children
- Is still actively working in company as engineer, no functional impairment
- Financially good
- Not smoker not drinker.

On examination, Mr Pang was alert and comfortable. Currently receiving IV antibiotics and on indwelling catheter draining small amounts of clear yellow urine. On examination of the abdomen, there are no scars, no hernias, no organomegaly, no palpable/percussible bladder. Renal punch was negative.

Digital rectal examination revealed an enlarged prostate of 3 finger breadths. This was smooth, symmetrical, firm and not nodular. I could feel the median sulcus and the rectal mucosa was smooth mobile over the prostate. Prostate was non-tender, no boggy areas felt. Stool in the rectum was soft and not impacted. Anal tone was intact.

There was no tenderness on percussion of the spine or any decreased breath sounds. There was no conjunctival pallor or pitting pedal edema to suggest chronic kidney impairment and there there was no cough impulse over both groins (hernia).

In summary, Mr Pang is a 63 year old Chinese gentleman. His issues are

1. Cystitis precipitating acute urinary retention, now stable post catheterization and receiving IV antibiotics
2. Background benign prostatic hyperplasia on alpha blockers
3. Elevated PSA for investigation.

QUESTIONS

What is the difference between voiding and storage symptoms, which does he have?

- His chronic history sounds like mainly voiding symptoms (hesitancy, slow stream, intermittency, straining to void, double micturition, terminal dribble) → this is characteristic of BPH.
- In the last two days he has developed new storage symptoms (urgency, frequency) with dysuria → due to UTI
- Most recently his ARU is the ultimate voiding symptom.

Tell me about his UTI. Is this upper or lower; what ddx do you have?

- More likely lower UTI due to the storage symptoms.
- No features of upper UTI such as loin pain, renal punch negative, or septic shock (note: fever does not equal upper UTI, severe cystitis can have fever too).
- Ddx is prostatitis - also dysuria + fever +/- ARU. Key points are pain on ejaculation, and on DRE, tender prostate +/- bogginess (abscess)

What are some other predisposing factors to UTI?

Sexually transmitted diseases, recent instrumentation of urethra, urolithiasis/cystolithiasis, structural abnormalities of the collecting system

What are the possible precipitants of acute urinary retention?

Causes can be divided into structural and functional.

Structural causes include:

- Intraluminal causes such as stones, blood clots and foreign bodies
- Luminal causes include tumour of bladder neck, urethritis (UTI), urethral strictures
- Extra luminal causes include BPH, prostate CA (less common as usually peripheral), constipation, pelvic masses, pregnancy, UV or rectal prolapse

Functional causes include

- Infection e.g. prostatitis
- Neurological causes, which can be further divided into
 - Central nervous systems pathologies like stroke, parkinson's, hydrocephalus, spinal cord injuries or lesions
 - Peripheral nervous system pathologies like DM autonomic neuropathy
- Medications such as anticholinergics, antihistamines, some analgesics esp opioids, anti-parkinsonian drugs, anti-depressants/anti-psychotics
- Post-op, post-anesthesia, pain and trauma

You are the HO on call, what will you do for this patient?

Sir, Mr Pang must be in a lot of discomfort from ARU; I will see to him as soon as possible.

- First I will assess his ABC and resuscitate accordingly.
- I will quickly insert a urinary catheter under aseptic technique which I expect to relieve his discomfort from acute urinary retention; I will also collect a urine sample and send for UFEME, gram stain, and urine culture - looking for pyuria and for microbiological diagnosis so that I can treat him better
- I will also set an IV plug and send off a septic workup which includes FBC for total white, CRP, blood cultures. I also want UECr looking for obstructive nephropathy
- I will do X ray KUB looking for stones.
- I will start empiric antibiotics for community acquired UTI, for example augmentin plus a dose of gentamicin, aiming for 7 days, to adjust based on culture and sensitivity.

His BPH will also need to be worked up later.

I would not do PSA in acute setting especially with ARU and catheterization (see later)

You have trouble catheterizing the patient. What do you do?

- Upsize the catheter
- Call urologist - bedside cystoscopy and insertion of catheter under direct vision
- Suprapubic catheterization (not really done anymore with the advent of bedside cystoscopy)

It is now the next morning; Mr Pang is now asking what to do about his BPH. What are some ddx you must consider before concluding it is BPH?

- Stricture → past hx of STD, urethral discharge, instrumentation
- Neurogenic bladder → any neurologic disease, lax anal tone, lower limb weakness or numbness
- CA prostate or bladder neck → ensure no painless hematuria, loss of weight, prostate normal on DRE; can do PSA to screen.
- Drugs

How would you manage his BPH?

I will like to confirm my diagnosis by doing uroflowmetry, expecting peak flow rate <15ml/s; and doing ultrasound bladder for increased post-void residual volume and intravesical prostatic protrusion. I will also do screening PSA (also do UFEME and Cr - already done in this case). I can also do IPSS (international prostate symptoms scale) scoring to find out how badly it is affecting him.

In view that he has had an episode of ARU despite being on alpha blockers, and knowing that such patients are high risk for recurrence of ARU, I am keen to offer surgery i.e. transurethral resection of prostate. An alternative is combination medical therapy with alpha blockers and 5-alpha reductase inhibitors, but the latter takes 3-6 months to work. I must also make sure that all drugs that predispose to ARU are taken off

Overview of treatment for BPH -

- Non-medical: avoid diuretics, caffeine, alcohol; reduce fluids after dinner
- Medical
 - For most patients, start with alpha blockers (tamsulosin, alfuzosin); counsel on hypotension, try to take at night.
 - Switch to 5-alpha reductase inhibitors (finasteride, dutasteride) if not tolerating (e.g. hypotension)
 - If not effective enough: combination alpha blocker + 5-alpha reductase inhibitor
- Surgical: transurethral resection of prostate
 - For patients who fail medical therapy, develop complications (repeated ARU, UTI, hydronephrosis, obstructive uropathy, bladder stones)
 - Risks: TURP syndrome (less with bipolar TURP, no need to use glycine for irrigation), retrograde ejaculation, incontinence, recurrence, structure.

Mr Pang prefers to continue with medical management for now. He asks about his high PSA - would you repeat his PSA now? What are the causes of high PSA?

- PSA can be falsely elevated as it is organ specific but not disease specific.
- Other than prostate cancer, it can also be elevated in BPH, prostatitis, UTI, ARU, and recent instrumentation of the urethra (including catheterization), recent TRUS biopsy, or even DRE!
- Therefore I would not repeat PSA this admission as it is likely to be falsely elevated.

How do you distinguish BPH vs Prostate CA?

- DRE: nodular, hard, asymmetrical prostate, obliteration of median sulcus, rectal mucosa fixed down to prostate
- PSA level (Prostate CA elevates PSA much more than BPH). Ranges: <4 normal, 4-10 abnormal (TRUS), >10 suspicious for CA (TRUS)
- If unsure > TRUS biopsy.

Mr Pang is discharged home well and came back for follow up in clinic, with repeat PSA on arrival. Repeat PSA comes back as 12. You proceed to do a TRUS biopsy. What are the complications of TRUS biopsy?

- Pain (cover with analgesia)
- Bleeding (counsel patient to expect PR bleed for a few days),
- Infection (cover with gentamicin)
- Lower urinary tract symptoms

Histology confirms prostate CA. Outline your management plan.

Sir, my patient's life expectancy is >10 years so I will like to treat actively.

- First I need to stage the patient; e.g. for high risk (Gleason 8-10, PSA >20) need MRI pelvis, CTAP for pelvic and paraaortic lymph nodes, and bone scan for bone mets. [Don't stage if don't intend to treat i.e. life expectancy <10y]
- Based on the risk stratification I will treat. For example if the patient is gleason 7, PSA 15, long expected life expectancy, I will offer radical prostatectomy vs radiotherapy.
- Thereafter need serial monitoring of PSA to look for recurrence.

General principles:

Risk class	If life expectancy >10y	If life expectancy <10y
Localized disease, low risk (Gleason ≤6, PSA <10)	Active surveillance: regular PSA, TRUS Individualize timing of resection: if short PSA doubling time, higher grade on biopsy → treat	Watchful waiting; no repeat TRUS or PSA (pt likely to die of comorbid, not prostate CA)
Localized disease, med risk (Gleason 7, PSA 10-20)	Radical prostatectomy + pelvic LN diss (SE: incontinence, impotence) Or Radiotherapy (SE: impotence, cystitis, proctitis; less incontinence) Or Brachytherapy	Active surveillance or watchful waiting.
Localized disease, high risk (Gleason ≥8, PSA >20)	As for mod risk, May need post-op androgen deprivation	
Locally advanced disease	RT with androgen deprivation therapy	
Metastatic disease	Androgen deprivation therapy - Castration (surgical vs medical - LHRH agonist) - Antiandrogen If hormone refractory → chemotherapy If bone mets, painful → palliative RT	

Patient is lost to follow up and now has unresectable /metastatic prostate CA with painful bone mets. How do you manage?

- Palliative RT
- Androgen deprivation
 - Castration: surgical vs medical (LHRH agonist)
 - Antiandrogen
- Analgesia.

Thyroid Cancer

PRESENTATION

Sir, my patient Mdm Tai is a 38 year old Chinese lady with no past medical history, who presents with a painless neck lump.

She first realized this lump two months ago when her relatives pointed it out during Chinese New Year visitations. Since Chinese New Year it has slowly increased in size. It is entirely asymptomatic with no pain, and she does not notice any other neck lumps.

[Ddx]

- [Thyroid cancer]: In terms of local symptoms, there is no change in voice, dysphagia, or dyspnoea (local invasion). She does not have constitutional symptoms of loss of weight or loss of appetite; also she does not have shortness of breath, bone pain, or jaundice.
- [Autoimmune thyroid disease]: In terms of thyroid status, she does not note heat intolerance, palpitations, diarrhoea, and oligomenorrhoea to suggest hyperthyroidism, neither does she notice cold intolerance, lethargy, and constipation to suggest hypothyroidism.
- [Parathyroid adenoma/carcinoma] No symptoms of hypercalcemia such as bone pain, loin to groin pain (kidney stones), colicky abdominal pain, confusion/lethargy
- [Lymph nodes]: She does not have any other symptoms of infection or malignancy in the head and neck region such as sinusitis or bloody nasal discharge.

[Risk fx] She has no known past medical or surgical history, is not on any long term medications, and has no known drug allergies. In particular, I do not note any history of MNG, radiation exposure, or autoimmune disease. She was born in Singapore and has stayed here all her life (MNG). Systemic review was unremarkable with no joint pain, rash

She has no family history of thyroid or parathyroid malignancy, no suggestion of colonic polyps (FAP) or other endocrine neoplasms (MEN2)

Socially, she does not smoke or drink. She is a full time housewife staying with her husband and 2 young children in a 5-room flat, and they are financially stable.

On examination, Mdm Tai is alert and comfortable at rest, not in respiratory distress, and not cachectic looking. On inspection I noted a central neck mass, just to the right of the midline, which ascends on swallowing but not tongue protrusion. There are no overlying scars or skin

changes. Palpation confirms a firm nodule, 4cm x 5cm, which has an irregular surface but is not fixed to overlying skin or underlying structures. It is not warm or tender. I can feel the lower border of mass. There were no other palpable nodules, and no cervical lymphadenopathy or retrosternal dullness. The trachea is central. Her voice is not hoarse. On examination of her thyroid status I find her euthyroid. There are no signs of thyroid eye disease.

In summary, Mdm Tai is a 38 year old Chinese lady with no past medical history presenting with a painless slow growing right thyroid nodule for investigation. There are no symptoms or signs of local invasion or distant spread, and she is euthyroid.

QUESTIONS

What are your differentials for a neck lump?

See short case thyroid and Approaches to Symptoms of Disease

How would you investigate this mass?

- TFT
- US thyroid
- Fine needle aspiration of the right lobe mass.

Can you please read this ultrasound?

Sir this is the ultrasound thyroid of my patient taken on _____. I note that there is a hypoechoic nodule (vs anechoic - cyst) measuring 3cm x 3cm, it appears to be taller than wide. Margins are irregular and I see microcalcifications. Doppler imaging confirms intranodular vascularity. Sir I am worried that this ultrasound shows features of malignancy.

FNAC was sent. Tell me about the Bethesda scoring system?

Sir the Bethesda system is a system for histopathological reporting of thyroid FNAC

- 1 : non-diagnostic, unsatisfactory → repeat FNA with US guidance
- 2 : benign → clinic follow-up
- 3 : atypia/follicular lesion of undetermined significance → repeat FNA
- 4 : follicular neoplasm → surgery
- 5 : suspicious for malignancy → surgery
- 6 : malignant → surgery

His FNAC came back as follicular neoplasm. What are your thoughts on this?

Sir a follicular lesion may be an adenoma or a carcinoma, and the only difference is capsular invasion which a FNAC cannot tell. I need to counsel the patient for hemithyroidectomy KIV completion thyroidectomy (removal of other lobe) if histology shows follicular carcinoma (capsular invasion).

Further questions

- How about frozen section? Previously the teaching was to do frozen section intra-op, but nowadays it has been found that frozen sections are inadequate for differentiating adenoma vs carcinoma... frozen sections only take a few cuts, high chance of missing the capsular invasion... therefore sending the whole hemithyroid for histology is currently the preferred option.
- Why is the completion thyroidectomy needed? Can do TSH suppression, can follow up with thyroglobulin levels, can do RAI ablation of any residual tissue

So patient goes for hemithyroidectomy, you review her in the ward post-op. What do you look for?

- Neck hematoma causing airway compression: airway, stridor, saturations, dyspnoea, visible hematoma, see drain amount and make sure not clogged > if present, need to undo stitches and return to OT
- Hoarseness of voice > injury to recurrent laryngeal nerve
- Check calcium levels (hypocalcemia), ensure no tingling and numbness of peri-oral region and hands, Chvostek and Trousseau sign are late.

Histology says follicular carcinoma. So how?

- Complete staging with CT neck thorax liver.
- Completion thyroidectomy with central compartment lymph node clearance
- KIV adjuvant radioactive iodine ablation depending on risk stratification, to be discussed at tumor board.

How do you prognosticate a thyroid cancer?

Sir differentiated thyroid cancers are generally of good prognosis especially since she is young (Age <40), has no metastases (M), extrathyroid invasion (E), and the size of her nodule was small (Size <4cm) - [AMES score].

After surgery how would you manage her in the long term?

- Give thyroxine at high doses for TSH suppression
- Monitor thyroglobulin levels (if RAI ablation done)
- RAI can be used to look for recurrence (lower doses than ablation)

Apart from thyroid cancer what are the other indications for thyroidectomy?

- Graves disease failed medical therapy and radioactive iodine (e.g. not compliant, not tolerated)
 - Beware thyroid storm: must ensure controlled before surgery, give Lugol's iodine pre emptively.
- Multinodular goitre causing compression symptoms, bad cosmesis

> *Can do subtotal thyroidectomy in these cases*

Hip: Osteoarthritis & AVN

HISTORY & EXAMINATION

Background: Age, PMHx, Drug Allergy (esp NSAIDs), Baseline function (ADL, Occupation - make sure not deep sea diving)

About the pain:

- Is this hip pain? → Please point to pain, any radiation? Differentiate hip vs back pain -
 - Hip: classically groin pain +/- radiation to anterior thigh (obturator nerve) but does not go past knee
 - Back: classically back pain radiating down side or back of leg past the knee
 - Other ddx: abdominal (retrocecal appendicitis, gynaecological disease, urolithiasis)
- Duration and course: How long has it been? Progressively worsening?
 - OA: elderly patient with longstanding progressive hip pain
 - AVN: beware in younger patient esp if risk factors present.
- Inflammatory vs mechanical: Worse on moving, better with rest? Worse in the morning + stiffness?

Etiology:

- History of fall / trauma?
- Rule out infection: fever?
- Rule out cancer: LOA/LOW?
- Could this be AVN?
 - Ortho hx: previous fracture, septic arthritis, congenital (Perthes, slipped capital femoral epiphysis)
 - Medical hx: steroid use, autoimmune disease
 - Lifestyle: e.g. deep sea diving, TCM (steroid)
 - Note that NOF# and septic arthritis are ddx of hip pain AND causes of avascular necrosis and secondary OA,

Function: ADLs? Ambulation requiring walking aid/orthosis? Are you still able to weight bear, walk? Clip toe nails? Able to continue work?

Examination: Sir, my patient Mr Tan is an 50 year old Chinese man who is alert and comfortable at rest. [Ensure adequate exposure and start with patient standing up]

On inspection, I do not note any scars, sinuses, swelling or deformities of the hip. I do not note any wasting of the quadriceps or hamstrings. **Sir can you walk there and back for me please?** I note that he has a right antalgic gait. There is no trendelenberg gait. I will now perform the Trendelenberg test. **Sir can you place your hands on top of mine and maintain your balance while standing on your right leg?** I note that the patient's left hip sags when she stands solely on her right lower limb, therefore this is a positive trendelenberg on the right. Trendelenberg test is negative on the left side.

Sir can you lie back down please? I am now palpating for bony landmarks, namely the ASIS, hip joint, greater trochanter. The right hip joint is tender on palpation, but not warm or swollen.

I will now move on to limb length measurement. Apparent limb length, measured from the xiphisternum to the medial malleolus, is ___ on the right and ___ on the left. True limb length, measured with the pelvis squared from the ASIS to the medial malleolus, is ___ on the right and ___ on the left. As there is true limb length discrepancy, I will now perform the Galeazzi test. **Sir can you flex your knees to 90 degrees?** Looking from the side, there is shortening of the femur but no shortening of the tibia. The patient's footwear does not have a heel raise.

I am now going to assess the ROM of the hip joints. All movements are limited (state the degree as you do each movement) and painful in the right hip, but normal in the left hip.

N.B. normal ROMs of the hip joint:

- Hip flexion: 110 to 120 degrees
- Hip abduction: 30 to 50 degrees
- Hip adduction: 20-30 degrees
- Hip extension: 10 to 15 degrees
- Hip external rotation: 40 to 60 degrees
- Hip internal rotation: 30 to 40 degrees

I am now doing the Thomas' test looking for fixed flexion deformities of the hip. **Ma'am I'm going to place one hand below your lower back. Bring your left knee up to your chest for me.** There is fixed flexion deformity of about 20 degrees in the right hip. **Ok put your left leg down. Bring your right knee up to your chest (or as high as you can) for me.** Patient is limited by pain, unable to assess if there is fixed flexion deformity in the left hip. There is crepitus felt over the right hip.

I would like to complete my examination by examining the lumbar spine and the neurovascular status of the lower limbs.

Summary: My patient is a 50 year old Chinese gentleman who fell on his hip 2 year ago and never saw a doctor. Since then he has had progressively worsening mechanical right hip pain. Examination shows positive right trendelenburg, shortened right femur on galeazzi, reduced ROM of the right hip joint in all directions and right FFD. I am worried about avascular necrosis from occult neck of femur fracture causing secondary osteoarthritis.

QUESTIONS

What are the causes of AVN?

1. Apparent: trauma, dislocation, fracture, septic arthritis
2. Insidious:
 - Drugs (steroids, alcohol, smoking)
 - Metabolic (Sickle cell, Gaucher)
 - Autoimmune (SLE, RA)
 - Pro-thrombotic states (e.g. Factor V Leiden, Protein C or S deficiency, hyperhomocysteinemia, antithrombin III deficiency)
 - Occupational (diving)
3. Paediatric age group: SCFE, Perthes

What are the causes of OA?

- Primary (degenerative)
- Secondary
 - Infective: septic arthritis
 - Inflammatory: RA, gout
 - Traumatic: untreated neck of femur fracture
 - Avascular necrosis

How would you investigate?

- Pelvis XR AP. Weightbearing XR of the hip AP and lateral views, looking for crescent sign or collapsed head of femur (Ficat stage 3), OA changes secondary to AVN (Ficat stage 4), also to rule out any fractures
- MRI of the right hip. T1 weighted scan – fat is white. Look for decreased signal in the femoral head, represents edema. T2 scan -- may find a double line sign (low density and high density line, corresponds to dead bone and surrounding inflammation for repair)

What treatment would you offer this patient?

Sir this patient has advanced OA of the hip... I will first attempt trial of conservative management which includes nonpharmacological and pharmacological therapy

- Nonpharmacological
 - Weight loss
 - Exercise in water
 - Walking aids
 - Physiotherapy for muscle strengthening exercises.
- Pharmacological:
 - Analgesia: paracetamol, NSAIDs with PPI cover
 - Intraarticular injection of steroids (triamcinolone)
 - Intraarticular viscosupplementation with Synvisc injections (hyaluronic acid which helps to lubricate the joint)

I think he will eventually need total hip replacement.

What if he came to you with earlier AVN?

Precollapse Ficat 0-2

- Bisphosphonates
- Core decompression (relieve intraosseous hypertension)
- Rotational osteotomy for small lesions - rotate lesion away from a weight bearing surface
- Curettage and bone grafting.

Collapsed

- Remove necrotic area and replace with fibular strut

Knee: Osteoarthritis

HISTORY

contributions from Teo Ling Li

Mdm Teo is a 50 yo female who has been complaining of progressively worsening right knee pain over the past two years. This is worse on exercise, especially on going downstairs, and relieved with rest. There is stiffness of 10min in the morning and swelling especially after exercise. Paracetamol used to relieve the pain but does not completely do so now. There are no red flags (fever, weight loss, calf pain, trauma, etc) or suggestion of referred pain (hip/back pain)

Notably Mdm Teo has had a previous sports injury s/p ACL reconstruction and meniscal repair 20 years ago. Currently she still experiences some locking/instability/catching especially when turning sharp corners (suspicious of ACL graft failing). She has no other past medical history and does not smoke or drink.

Functionally, she is a housewife who stays with husband and son. She is coping okay without walking aids but it bothers her that she cannot do housework. At home they stay on a lift landing floor and have a sitting toilet.

Examination: My patient is a middle aged Chinese lady in her 50s who is alert and comfortable. Inspecting from the front, side, and back with the patient standing; there is a short midline scar* over the anterior knee with arthroscopic scars over the medial aspect, suggestive of previous cruciate ligament repair using the patella tendon. There is swelling of right knee (loss of parapatellar fossa), but no Baker's cyst (associated with OA) or other swellings. There are no genu varus or valgus deformities of her knees, but I note a fixed flexion deformity of the right knee from the side



Maam can you walk for me please? Her gait is antalgic on the right, and she requires a walking stick. **Can you squat?** I note that she is not able to squat

Maam can you lie down please. Measuring thigh circumference 10cm superior to the lateral femoral condyle, the right thigh is 35cm whereas the left thigh is 39cm. Both knees are not

warm. On palpation there appears to be positive patellar grind, medial and lateral joint tenderness. There is also a positive fluid shift test.

I will now like to demonstrate movements. **Ma'am can you straighten your knee? Ok let me try to help you.** There is a fixed flexion deformity of 30 degrees in the right knee. **Ma'am can you bend your right leg to touch your thigh? Ok ma'am I'm going to help you bend a bit more.** Active and passive flexion are both 160 degrees. Range of movement is 0 degrees to 160 degrees in the left knee. Internal rotation of both hips are full and not painful. There is crepitus in the right knee, but not in the left.

I will now perform the drawer tests. **[Position patient].** There is no posterior sag. Anterior drawer test on the right knee is positive with a spongy endpoint, but negative on the left knee with a good end point. I will now perform the Lachmann test. Again it is positive on the right knee with a good end point, but negative on the left with a good end point.

I will examine the other ligaments. I am now performing the valgus stress test in full extension, and again in 20 degree of flexion. The test is negative. I am now performing the varus stress test in full extension and again in 20 degrees of flexion. This is also negative.

I will now like to perform the McMurray test for the medial meniscus. I am flexing and externally rotating the knee, applying a valgus stress and now gradually extending the knee. There is no clunk and the test is negative.

I will like to complete my examination by examining the hip, lumbar spine, and doing a neurovascular examination of the lower limb.

Summary: Sir, my patient is a 50 year old Chinese lady who was an avid soccer player in her youth, presenting now with secondary osteoarthritis of the right knee on a background of ruptured right ACL repair. This is affecting her ability to do housework. There are no red flag symptoms such as fever, rest pain, LOW, LOA. Physical examination was consistent with osteoarthritis of the right knee, and a right ACL tear.

* *Note:* scars for ACL repair include arthroscopic port scars, plus a graft scar -

- Semitendinosus graft: Hockey shaped scar along the medial aspect of the tibia
- Patellar tendon graft: short anterior midline scar [patellar tendon should not be used in individuals who have high use for it: Malays (kneeling to pray) or high jumpers as much of the explosive power of the extensor mechanism hinges on an intact patella tendon)]
- Distinguish from TKR scar which is a long midline scar.

QUESTIONS

Can you tell me how you do fluid shift test?

- Milk the supra-patellar pouch.
- Milk the median sulcus from bottom to up, up and around the top border of the patella and down into the lateral sulcus.
- THEN milk the lateral sulcus from bottom to up and watch for re-filling of the median sulcus --> positive fluid shift test.

Is this primary or secondary OA? Why?

This is secondary OA

- Unilateral
- History of previous ligamentous injury.

What are the causes of secondary OA?

- Previous trauma and injuries (e.g. patella fracture, tibial plateau fracture, meniscal tears and resections, ligamentous tears)
- Inflammatory arthritis: gout, RA
- Septic arthritis

What are your differentials?

- Knee pain: Rheumatoid arthritis, gouty arthritis, psoriatic arthropathy
- Locking symptoms: medial meniscus injury
- Also offer spine and hip.

How would you investigate?

I would start with weightbearing AP, lateral and skyline X-rays of the knee. I am looking for osteoarthritic changes namely reduced joint space, subchondral sclerosis, subchondral cysts and osteophyte formation.

Can also do long leg film looking at the mechanical axis of the leg (line from femoral head to ankle joint) which in this patient with varus deformity may go through the medial joint compartment instead of centre of the knee. (Alternative: look at tibial-femoral angle)

I would also consider doing some basic blood investigations such as FBC RP ESR RF ANA, to rule out rheumatoid arthritis.

How would you manage?

I would like to attempt conservative management first

Nonpharmacological measures:

- Weight loss
- Physiotherapy: knee strengthening (esp vastus medialis for varus knee), aerobic exercises (improve function and reduce pain)
- Modalities
 - Aquatherapy (exercise in water)
 - Transcutaneous electrical nerve stimulation (TENS): short term pain relief, improve ROM
 - Thermal modalities
- Orthotics to ameliorate varus deformity:
 - Valgus knee brace
 - Lateral wedge insoles
- Acupuncture (adjunct)
- Occupational therapy: walking aids, activity modification

Pharmacological measures:

- Topical NSAIDs e.g. ketoprofen patch.
- Analgesia - know exactly what and what dose (ensure no contraindication)
 - Paracetamol 1mg QDS
 - Diclofenac 75mg BD
 - Arcoxia 60mg or 90mg OM
 - Tramadol 25mg or 50mg TDS
- Glucosamine: helps with inflammation, not with repair. Evidence shows >6 month regular use no benefit compared to placebo.

Intraarticular injections

- H&L: steroids (triamcinolone)
- Viscosupplementation with Synvisc injections (intraarticular hyaluronic acid which helps to lubricate the joint)

However as her osteoarthritis is quite severe and pain is significantly limiting her function, I think she may require total knee replacement in order to preserve her mobility (if she is fit for operation).

What are the risks of total knee replacement?

- Early: Injury to surrounding structures esp peroneal nerve → foot drop, numbness over dorsum of foot, complications from general anesthesia, deep vein thrombosis and pulmonary embolism, surgical site infection
- Late: stiffness, periprosthetic fracture, implant failure

How do you do a TKR?

1. Make incision, usually 8-10 inches longitudinally on anterior aspect of knee
2. Rotate the patella to the side, to gain access to the knee joint
3. Resurface femur
4. Attach metal femoral component (titanium) to the end of the femur and secure with bone cement (PMMA)
5. Resurface tibia
6. Attach bottom portion of the implant, called the tibial tray (titanium), to the tibia and secure with bone cement (PMMA)
7. Put a polyethylene (medical-grade plastic) insert to sit between the tibial tray and the femoral component. This will serve as a kind of buffer, reducing wear and tear of the articulating surfaces of the implant.
8. Patella will be flattened and fitted with an additional plastic component to ensure proper fit with the rest of the implant
9. Bend and flex the knee to ensure that the implant is working correctly, and that alignment, sizing, and positioning is suitable
10. Close the incision with stitches or staples

How long would a TKR last and why doesn't it last longer?

A typical TKR lasts about 15 years. It may not last as long if patient is very active and the implant wears out faster.

So how about a young patient, what can you offer them?

If the patient has unicompartmental OA, can offer -

- Medial compartment: tibial osteotomy to correct varus knee (good for 5 - 10 years), medial unicompartmental arthroplasty
- Lateral compartment: distal femoral osteotomy to correct valgus knee, lateral unicompartmental arthroplasty
- Isolated patellofemoral: Patellectomy, tibial tubercle elevation, lateral retinacular release (+/- partial facetectomy)

Others

- Arthroscopic debridement: especially for mechanical symptoms e.g. loose bodies/flaps of meniscus or cartilage that are causing mechanical symptoms esp locking, catching, giving way
- Cartilage regeneration techniques e.g. harvest from non weightbearing part of knee joint, culture in vitro, then autologous chondrocyte implantation

Spine: Cervical Myelopathy

HISTORY & EXAMINATION

History

- Background: Age, PMHx, Drug Allergy, Baseline function (ADL, Occupation)
- Elicit presenting symptoms & time course (progressive or stepwise)
 - Neck pain → Ensure that pain is mechanical. May have 'electric shock' sensation down spine on forward flexion (Lhermitte sign)
 - Gait disturbance: spastic, ataxic
 - LMN in UL: loss of fine motor control / clumsy, numbness
 - UMN in LL: weakness, spasticity
 - Bladder/bowel symptoms (develops late)
- Rule out red flags
 - Trauma
 - Inflammatory pain: at rest, at night, disturbs sleep
 - Loss of appetite/weight
 - Fever
- Consider ddx: aside from cervical radiculopathy the rest are non-orthopaedic and neck pain does not feature prominently
 - Cervical radiculopathy: main complaint will be pain shooting from neck to arm in a dermatomal distribution; no gait, bladder/bowel, or LL disturbance
 - Discogenic disk pain: neck pain only with no other symptoms
 - Transverse myelitis: e.g. multiple sclerosis, neuromyelitis optica
 - Syringomyelia
- Evaluate complications
 - Falls
 - Anterior cord syndrome / spinal cord injury after minor trauma.
- Evaluate function: able to work?
- Explore PMHx
- What has been done for the patient so far?

Examination: Sir, my patient is a middle-aged Chinese gentleman who is alert and comfortable at rest. He uses a walking stick.

On inspection there are no scars, sinuses, swellings or deformities of the neck. **Sir, can you walk there and back please.** I note that he has an unsteady, broad-based gait (if normal, stress patient with tandem gait).

On palpation, there is no step deformity of the spine, no warmth or tenderness. There is some paraspinal muscle spasm

Sir can you now follow my movements - bend your neck forward, do you feel anything? On forward flexion of the neck, the patient complains of pain shooting down his spine which is a positive Lhermitte's sign. Neck extension, lateral flexion and rotation are otherwise full and without pain.

I will now do a targeted neurological examination.

- **Tone: Sir I am going to move your hands and legs, I want to see how relaxed you can be.** Tone is normal in upper limbs and slightly hypertonic in lower limbs with no clonus.
- **Reflexes: Sir, this is a tendon hammer, I'm going to tap your arms and legs gently.** I note that bilateral upper limbs have an absent biceps jerk, inverted supinator jerk and an exaggerated triceps jerk. Ankle and knee jerk are slightly hyperreflexic and Babinski is equivocal
- **Power: Sir I'm going to test how strong you are now. Don't let me push you up/down.** Power is 5 in all limbs.
- **Sensory: Sir I want to see how well you can feel. This is a satay stick - I am now touching your forehead, this is 100%. Can you close your eyes and tell me how many % each time. I note that sensation is intact over all limbs**

Special tests:

- Hoffman's sign (flick down) is present in both upper limbs.
- **Sir, open and close your hands as quickly as you can in 10 seconds.** My patient has slow grip and release of less than 20 times in 10 seconds.
- **Sir, stretch out your hands as straight as you can.** There is ulnar drift in both hands.

Sir, can you write something for me / button your clothes / pick up this coin? In terms of function, his writing is impaired

I would like to complete my examination with a digital rectal examination

Summary: Sir, my patient is a 70/M/Chinese with no significant PMHx who presents with 6 months of progressively worsening mechanical neck pain, associated with loss of finger dexterity and gait disturbance. I do not note any red flags such as rest pain, night pain, loss of weight, or fever. Functionally he is disturbed by difficulty writing and using chopsticks. Physical examination is consistent with cervical myelopathy at the C6 level

QUESTIONS

How would you investigate this gentleman?

- XR cervical spine, AP lateral views
- MRI cervical spine looking for spinal canal AP diameter <10mm, level of disease, and ruling out differentials such as mitotic lesion
- If prompted - pre-op: FBC, RP, PT/PTT, GXM, ECG, CXR, 2DE

Can you please read this X-ray?

Have a systematic approach to reading!

- Introduce: Sir this is the lateral cervical spine X ray of my patient.
- Adequacy: This is an adequate radiograph showing up till C7/T1 junction. There is good penetrance.
- Alignment: The alignment of the vertebrae (anterior vertebral, posterior vertebral, spinolamellar line, posterior spinous line) is intact although I note loss of cervical lordosis. *There is no spondylolisthesis (always look).*
- Bone: Bone is intact, no fractures.
- Discs: There is decreased height of the C4/5, C5/6, C6/7 discs
- Conclusion: This is consistent with spondylosis changes although I will like an MRI to look at the spinal canal itself

Can you please read this MRI?

- Introduce: Sir this is the T2-weighted MRI of the patient's cervical spine in axial / lateral view
- Lateral view: Cervical spine has lost its physiological lordosis. There is disc protrusions at the C4/C5, C5/C6, C6/C7 level resulting in effacement of the CSF (no more CSF space) and indenting the spinal cord. There is high T2 signal noted in the cord (=marrow edema, sign of chronicity). The discs also appear dessicated (no white inside)
- Axial view: There is central disc protrusion indenting the spinal canal and reduced spinal diameter of <10mm (kidney bean shape).
 - In contrast - for radiculopathy, look for lateral disc prolapse, foraminal narrowing.

How would you manage this gentleman?

Sir as my patient's has progressive symptoms affecting function, and because the natural history of cervical myelopathy is not to spontaneously resolve, I will like to offer the patient surgical decompression. In view of multi-level disease I would opt for posterior approach

(1) When to offer surgery

- Natural hx of cervical myelopathy: will progress but may be slow
- Functional disturbance, progressive symptoms → surgical
- Mild symptoms, good premorbid, severe disease on MRI → surgical to minimise risk of deterioration
- Mild symptoms, poor premorbid → conservative (analgesia, neck strengthening exercise, collar, fall caution)
- Asymptomatic → do nothing.

(2) What surgery to offer

- 1-2 levels affected → Anterior cervical discectomy and fusion (less infection, blood loss than posterior approach)
- ≥3 levels affected → Laminectomy with posterior fusion (higher cx of dysphagia, difficulty breathing for anterior approach in multi-level disc)

	PID, cervical radiculopathy (no cauda equina)	Lumbar spinal stenosis	Cervical myelopathy
Ntl hx	80-90% recover in 6 weeks	1/3 improve 1/3 same 1/3 require surgery	Progressively worsen Acute decompensation after trauma
When to offer surgery	Symptoms > 8 weeks Severe weakness or cauda equina syndrome	Failed conservative Mx and function poor Progressive neurology	Prevent progression Offer to most, unless mild symptoms mild & normal function, or poor premorbid
Surgical option	<u>PID 1 level:</u> decompression e.g. microdiscectomy or open discectomy +/- laminectomy <u>PID multiple levels:</u> need fusion e.g. TLIF <u>C-spine:</u> ACDF	<u>No spondylolisthesis:</u> decompressive laminectomy <u>Spondylolisthesis or back pain > leg pain:</u> add fusion e.g. TLIF	<u>1-2 levels:</u> ACDF <u>≥3 levels:</u> posterior laminectomy and fusion

How do you counsel for surgery?

- Goal of surgery is to reduce risk of progression, may not have fully normal neurologic function after, and 5-30% may not feel better.
- Early complications:
 - Nerve root injury: C5, C6, recurrent laryngeal nerve → dyspnoea, hoarseness
 - Anaesthetic risk
 - Epidural hematoma
 - Wound infection
- Late complications: nonunion, pseudoarthrosis

Spine: PID & Cervical Radiculopathy

HISTORY & EXAMINATION

History:

- Background: Age, PMHx, Drug Allergy, Baseline function (ADL, Occupation)
- Back pain
 - Is this neck/back pain? → Consider ddx: hip (unable to squat), knee, shoulder.
 - Onset + surrounding circumstances (suddenly after heavy lifting?), duration, progressive or stable
 - Mechanical vs inflammatory - worse on movement, better on rest
 - Better on flexion (disc pathology) or extension (facet joint disease, pars interarticularis)?
- Neurologic symptoms
 - Sciatica i.e. shooting/radiation of pain from back to arm/leg. Usually a single dermatome in PID. Identify which
 - L2: to the groin
 - L3: anterior thigh
 - L4: medial thigh and calf
 - L5: lateral thigh and calf, dorsum of foot
 - S1: back of thigh and calf, sole/heel
 - C6: Neck, deltoid, radial forearm, thumb ± index finger.
 - C7: similar going to middle finger.
 - Weakness, numbness
- Red Flags
 - Loss of urinary or bladder bowel incontinence, saddle anesthesia
 - Fever
 - Rest pain, night pain that disturbs sleep
 - LOW, LOA → Cancer
 - Abdominal pain → AAA
- Previous treatment
- Function

Examination: Sir, my patient is alert and comfortable at rest. **inspect from front, side, back** There are no scars, skin changes or deformities such as kyphoscoliosis or scoliosis. He is listing to the right.

Sir I'm going to feel along your spine now. On palpation, there are no step deformities of the spine. There is tenderness on palpation of the vertebrae, associated with paraspinal muscle spasm on the left.

Sir I'm going to see how well you can move. I am now performing Schober's test by marking the level of the posterior iliac spines, and marking a point 10cm above and 5cm below. **Sir can you bend forward please?** Flexion of spine is restricted to 40 degrees by pain, lumbar excursion is only 2cm. **Sir can you follow my movements please?** Extension, bending sideways and rotation are also with some pain but able to demonstrate full range of movement. **Sir can you sit down please? (fix the pelvis)** Rotation is normal.

Ok please lie down now sir. I'm going to do raise your leg slowly, please tell me what do you feel? > Can you tell me where is the pain. Straight leg raise of right leg positive at 40 degrees flexion, with positive Lasegue's test. Left leg normal.

I will now do a targeted neurological examination.

- **Tone:** Sir I am going to move your hands and legs, I want to see how relaxed you can be. Tone is normal.
- **Reflexes:** Sir, this is a tendon hammer, I'm going to tap your arms and legs gently. Knee reflexes are present but ankle is absent on the right.
- **Power:** Sir I'm going to test how strong you are now. Don't let me push you up/down. Big toe and ankle dorsiflexion was MCR 3 on the right. Hip flexion, knee flexion, knee extension, and ankle plantarflexion were full power, and all movements on the left leg were normal.
- **Sensory:** Sir I want to see how well you can feel. This is a satay stick - I am now touching your forehead, this is 100%. Can you close your eyes and tell me how many % each time. There is decreased sensation over the lateral aspects of the right leg, top of feet including 1st dorsal webspace

I would like to complete my examination by doing

- Digital rectal examination to assess anal tone and saddle anesthesia.
- Abdominal examination to check for pulsatile mass and palpable bladder.

Summary: In summary, this is a middle aged Indian loading officer with mechanical back pain and sciatica radiating to L5 dermatome on right foot. This started acutely 3 months ago when he was lifting heavy objects, and has persisted since then with worsening foot drop rendering him unable to work. There is no signs and symptoms of cauda equina syndrome, rest pain, night pain, LOW, or fever. Physical examination is consistent with prolapsed intervertebral disk at L4/L5 position causing L5 nerve root symptoms.

Note: Differential in an older patient can be spondylolisthesis

QUESTIONS

How would you investigate this gentleman?

Sir, I would first do XRays of the lumbar/cervical spine, both AP and lateral views

- This is the lateral lumbar/cervical spine XR of this patient taken on _____. There is good penetrance. There is preservation of cervical/lumbar lordosis. Most notably there is loss of disk space between the _____ vertebrae (count from odontoid process of C2, or count from sacrum). I do not see any spondylolisthesis. There are no spondylotic changes such as end plate sclerosis, syndesmophytes formation or facet joint osteoarthritic changes.
 - In a lateral lumbar spine XR, can also always comment on hip joint
- This is the AP lumbar/cervical spine XR of this patient taken on _____. There is good penetrance. The psoas shadows preserved (therefore no paraspinal abscess). There is no winking owl sign (disappearance of pedicle). I do not note any vertebral fractures, scoliosis. SI joints look normal (sacroileitis: blurring of joint lines, sclerosis, widening of joint space).
- An MRI spine would then be needed, looking for lateral recess prolapse causing impingement of transversing nerve root

How would you manage this patient?

Sir as 80-90% of patients recover within 6 weeks I will first offer conservative management

- Nonpharmacological
 - Bed rest
 - Lifestyle modification: avoid heavy lifting, educate patient to lift properly (squat down instead of bending back), stop smoking and alcohol
 - Physiotherapy: strengthening of back muscles, heat therapy, ultrasound therapy
 - Orthosis: lumbar spine support
- Pharmacological
 - NSAIDs with PPI
 - Nerve root agents: gabapentin, pregabalin, amitriptyline.

If he is still symptomatic at 8 weeks I would offer him surgical decompression with

- As his disease affects a single level gold standard is microdiscectomy (old one: discectomy +/- laminectomy or laminotomy), or
- As his disease affects multiple levels I would offer decompression and fusion e.g. transforaminal lumbar Interbody fusion
- Post-op: rehab is very important, light work only after 1 month and heavy work only after 3 months

[Please refer to table comparing treatment of various spine disease; see Cervical Myelopathy]

If this patient also has urinary symptoms what would you do?

Sir I am worried about cauda equina syndrome. I would arrange urgent MRI scan. The patient needs decompression within 48 hours.

What are the complications of surgery?

- Early: paralysis, loss of urinary continence, infection (including meningitis), epidural hematoma, anaesthesia risk.
- Late: recurrence of slip disk (10%), adjacent disk develops slipped disk, implant failure (no fusion)

Spine: Lumbar Spinal Stenosis

HISTORY & EXAMINATION

History:

- Background: Age, PMHx, Drug Allergy, Baseline function (ADL, Occupation)
- Elicit presenting symptoms & time course (progressive or stepwise)
 - [Axial] Back pain → Ensure that pain is mechanical. Is it worse on flexion (discogenic) or extension (facet joint)?
 - [Appendicular] Neurogenic claudication: unilateral or bilateral cramping/tingling/discomfort in calf/thigh/butt on walking, variable claudication distance, relieved on flexion, worse walking downhill, can have numbness/weakness if more severe
 - Differentiate from sciatica, which would be sharp shooting pain radiating down from back down the leg, present both at rest and on movement (spinal stenosis can sometimes have radicular symptoms, albeit less commonly than in PID)
 - Is leg pain worse or back pain worse? (Affects management)
- Consider ddx:
 - Vascular claudication -- this has a fixed claudication distance (vs variable), worse walking uphill (vs downhill), relieved on rest (vs spine flexion), no numbness or weakness
 - Prolapsed intervertebral disc -- younger patient, radicular (shooting) rather than cramping pain, has neurology; spinal stenosis patients do not often have neurology, but osteophytes may sometimes press on exiting nerve roots and cause radicular symptoms
 - Hip pain: if able to squat, less likely hip.
- Rule out red flags
 - Trauma
 - Cauda equina syndrome: Bladder/bowel dysfunction
 - Inflammatory pain: at rest, at night, disturbs sleep
 - Loss of appetite/weight → Cancer
 - Fever → Infection
 - Abdominal pain → AAA.
- Evaluate function
- Explore PMHx
- What has been done for the patient so far?

Examination: Sir, Mr Ahmad is alert and comfortable at rest; I do not see any walking aids next to him. On inspection there are no scars, skin changes or deformities. I note that he is standing in slight flexion. **Can you please walk there and back?** The patient's gait appears normal, not broad based or antalgic.

Sir I'm going to feel along your spine now. On palpation, I do not note any step deformity, midline tenderness, or paraspinal muscle spasm.

Sir I'm going to see how well you can move. I am now performing Schober's test by marking the level of the posterior iliac spines, and marking a point 10cm above and 5cm below. **Sir can you bend forward please?** There is only an increase of 4cm indicating reduced forward flexion. **Sir can you follow my movements please?** Lumbar extension and lateral flexion is normal. **Sir can you sit down please? (fix the pelvis)** Rotation is normal.

Sir I will skip straight leg raise in this patient as he does not appear to have symptoms of sciatica (do if there are radicular symptoms)

I will now do neurological examination

- **Tone:** Sir I am going to move your legs, I want to see how relaxed you can be. Tone appears to be normal and there is no clonus.
- **Reflexes:** Sir, this is a tendon hammer, I'm going to tap your arms and legs gently. Sir I note depressed reflexes over bilateral ankles. Knee jerk is normal. Plantars are downgoing.
- **Power:** Sir I'm going to test how strong you are now. Don't let me push you up/down. Power is 5 in all myotomes.
- **Sensory:** Sir I want to see how well you can feel. This is a satay stick - I am now touching your forehead, this is 100%. Can you close your eyes and tell me how many % each time. I note that sensation is intact over all limbs

I would like to complete my examination by

- Palpating dorsalis pedis pulses to rule out vascular claudication
- Doing digital rectal examination assessing anal tone and perianal sensation
- Abdominal examination to check for palpable bladder

Summary: Mr Ahmed is a 70 year old Malay gentleman presenting with 3 years lower back pain and worsening neurogenic claudication. I do not note any red flags such as rest pain, weight loss, fever, or suggestion of cauda equina. Function wise, there is mild limitation of mobility but as a retiree with low functional demand he is not extremely bothered. His pre-morbid conditions are good with only well controlled diabetes, hypertension, hyperlipidemia. Examination findings are that of reduced lumbar spine flexion with slightly depressed LL reflexes but full power. There was no step deformity. In summary Mr Ahmed is a 70 year old Malay gentleman with symptoms of lumbar spinal stenosis.

QUESTIONS

How would you investigate?

- XR lumbar spine AP lateral, looking for degenerative changes
- MRI lumbar spine looking for reduced spinal canal anteroposterior diameter
- FBC - rule out myeloma (think of myeloma if anaemia), lymphoma, infection
- UECr - so that I can give NSAID
- Pre-op also: FBC, RP, PT/PTT, GXM, ECG, CXR, 2DE; add HbA1c and fasting glucose for this patient.

Can you read this X-ray please

- Introduce: Sir this is a lateral X-ray of my patient's lumbar spine
- Adequacy: There is good penetrance.
- Alignment: I note that there is grade 2 spondylolisthesis of the L4 upon L5 (grade based on ratio of overhanging part of superior vertebral body vs AP length of inferior vertebral body, grade 1 = 0-25%, grade 2 = 26-50%, grade 3 = 51-75%, grade 4 = 76-100%). I will also like a oblique view looking for spondylosis (pars interarticularis break)
- Disk: There are changes of spondylosis with reduced disk height of L3/L4, L4/L5, L5/S1, endplate sclerosis, osteophyte formation, +/- facet joint OA change.
- Bone: Bone is intact (all square shaped)
- Hip shows no OA changes.
- In summary this X-ray shows spondylotic changes with spondylolisthesis

What is the prognosis of lumbar spinal stenosis?

1/3 improve, 1/3 same, 1/3 require surgery

How would you manage this patient?

As Mr Ahmed's function is not too badly affected I will first offer a trial of conservative management with weight loss, analgesia, and physiotherapy to strengthen muscles and correct posture. If he does not improve and symptoms worsen to limit function I would offer surgery - in his case he has spondylolisthesis; decompression alone would worsen instability, so he also needs a fusion procedure e.g. transforaminal lumbar intervertebral body fusion (TLIF)

[Please refer to table comparing treatment of various spine disease; see Cervical Myelopathy]

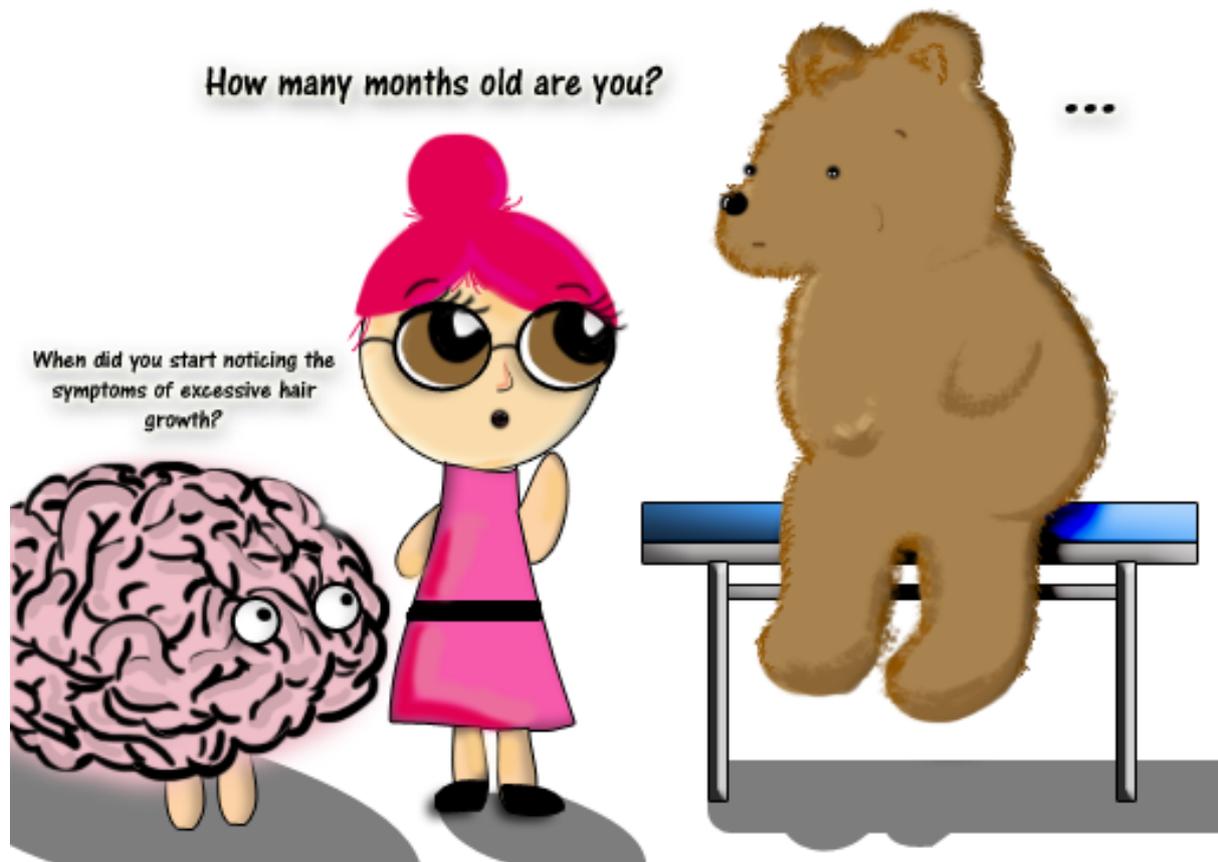
If for op what will you counsel the patient on?

- Risk that symptoms may not improve or may recur due to adjacent level disease (20%)
- Operative risk: e.g. infection, epidural hematoma, nerve injury, anaesthetic complications (depending on premorbid)

PREPARING FOR THE MBBS | CHAPTER FOUR

Medicine Long Cases

the clinical questions approach



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Strategy

What are medical long cases like? 25 minutes for history and examination, 2 minutes for consolidation, and 10 minutes for presentation and discussion. 2 examiners are present throughout. This seems to be designed to reflect an outpatient consultation of a chronic condition, although you can at times get acute conditions / inpatients. 80% of cases are adult and 20% are paediatric.

Examiners want you to:

- Identify the issues and formulate a problem list
- Assess each issue in terms of etiology – complications – management – control.
- If there is a new issue (diagnostic cases), demonstrate a sound approach to arrive at diagnosis and differentials.
- Have a holistic approach: not only deal with medical issues, but also how these have impacted the patient functionally and socially. You should be interested in the patient's life, delving deep into social, financial emotional, and psychological aspects.
- Consider interactions between issues: e.g. disease-disease, drug-drug, drug-disease interactions, how disease affects patient, and how patient / environment affects disease.
- Presentation: give problem list and your assessment of each problem, instead of presenting the entire history.
- Discuss your management plan, tailored to *this* patient's problems and unique circumstances. You should show awareness of community resources and strategies to deal with the social problems.

Your strategy: Medical patients often have multiple problems (some acute, some chronic), multiple interactions between issues, and a complex social history. Unlike the surgical long case, the focus is on managing *this* patient (and his/her multiple issues) *as a whole* as you would in a clinic consult, rather than going in depth into one specific acute problem. You will need to deal with whatever the patient throws at you – a new complaint (whether related or not to old issues), follow-up management of chronic issues (which may be very well controlled or a total wreck), complications of existing issues or medications, specific concerns and worries, and social issues, or a combination of the above. It should be apparent by now that the medical long case requires an organized thought process to sort out the issues, the mental flexibility to multitask, and a good deal of clinical acumen. Note:

- The twin exhortations discussed in the surgical long cases – to be comfortable with clerking real patients, and to think through cases you have seen – are just as important here. Please refer to the surgical long case strategy for that discussion.
- This chapter should be read with *Approaches to Symptoms of Disease*. *Approaches* takes you from presentation to diagnosis, while this chapter takes you from diagnosis to assessment and management.

A suggested toolbox to deal with the medicine long case is as follows:

1: CORRECTLY IDENTIFY THE CLINICAL PROBLEM

This is a necessary to proceed. Be aware that an ‘issue’ may reflect different clinical problems. For example, a patient with seizure might be (1) a first seizure, or a (2) known epileptic with breakthrough seizures. The management tasks are very different: in (1), the goal is to look hard for precipitating causes, in (2) the goal is antiepileptic drug titration.

2: FOR A NEW COMPLAINT, THINK THROUGH DIFFERENTIALS

Think in terms of ddx related to existing issues, or entirely unrelated. See the separate notes on *Approaches to Symptoms of Disease*. When you have shortlisted differentials, think through by filling in this table –

Differential	What is in favour?	What is against?	What is expected but absent?	What other info do I need?
Ddx #1				
Ddx #2				

3: FOR A CHRONIC ISSUE: APPLY A ‘CLINICAL QUESTIONS’ APPROACH

Aim: this is a tool to assess a chronic issue, identify management tasks (‘what do I have to do for this patient today’), and improve holistic management (‘how can we do better’).

Basic premise: each chronic issue can be broken down into a series of clinical questions (generally a variation of etiology – severity – complications – management tasks). For example, COPD can be considered in terms of –

1. Is the diagnosis COPD?
2. What is the COPD stage?
3. What is the risk and history of exacerbations, and how is this mitigated?
4. What are the baseline symptoms and how are these managed?
5. Are there any complications and how are these managed?
6. How is the patient’s general health and comorbids?
7. How is the patient coping in general?

Using the clinical questions approach: As you go through history / examination / investigations / management, think through each question in turn. For example,

- On history: (1) clarify the symptomology and rule out asthma, (3) explore previous admissions, (4) ask about baseline oxygenation, functional status, (5) screen for red flags of lung cancer, and so on.
- Investigations: spirometry for (1) diagnosis and (2) staging, rule out (5) pneumothorax in acute exacerbation
- Management: (2) consider GOLD guideline, (3) strategies to prevent exacerbations e.g. inhaled steroid, vaccinations, (4) symptomatic management including smoking cessation, bronchodilation, etc.

Benefit of this approach: We begin with the premise that every patient is very different and has to be carefully assessed (e.g. the well-controlled diabetic on OHGA is vastly different from the diabetic with ESRF and amputations). We have found this approach incredibly helpful in dissecting complex issues into simple questions, allowing comprehensive assessment of each patient without remembering checklists, making necessary investigations and management plans obvious, and even allowing identification of gaps in management so that they can be improved.

In this notes: This set of notes is organized along this 'clinical questions' approach, which will become clearer as you use these notes. Rather than learn separate adult and paediatric approaches, we have as far as possible integrated the adult and pediatric questions, and highlighted how they differ. The range of possible medicine/paediatric cases is wide and we cover perhaps 70-80% of the possible issues (but you can get combinations and variations of these issues).

4: SUMMARIZE ISSUES AND PRESENT – A WORKED EXAMPLE

The history you take:

Mr Akbar is a 49 year old Indian gentleman with a background of Child's C alcoholic cirrhosis, recently admitted for hematemesis and discharged 2 weeks ago.

He had been well for 1 week on discharge but now presents with a 1-week history of altered mental status, manifesting as increased lethargy and difficulty expressing himself. Given the background of Child C cirrhosis the first thought would be hepatic encephalopathy, but pure aphasia with alertness is not typical for hepatic encephalopathy. There are no neurological deficits on history or physical examination, and there are no infective symptoms. In terms of possible precipitants for hepatic encephalopathy, I do not note any malena, hematemesis, recent alcohol ingestion, constipation, or new medications.

In terms of his background of cirrhosis, this was diagnosed 1 year ago when he presented with lower limb edema. This is most likely alcoholic on the basis of significant alcohol consumption and negative tests or family history of hepatitis. He has never had a liver biopsy, which I would expect if other etiologies such as autoimmune hepatitis were suspected.

In terms of complications, he has had 2 admissions for symptomatic ascites requiring tapping, 1 admission for hematemesis requiring intubation, status post endoscopic variceal ligation. He does not have other bleeding symptoms.

Otherwise he has very good social support.

The examination you do:

Examination was remarkable for stigmata of chronic liver disease such as shifting dullness, loss of axillary hair, telangiectasia. There was also an umbilical hernia. Although distended, abdomen was non-tender; i expect splenomegaly but was not able to palpate for the spleen through the ascites. There was no asterixes and the patient was alert although he has expressive aphasia. I did not note any focal neurological deficit or lateralizing sign. There is no ataxia or ophthalmoplegia to suggest Wernicke's. The patient is eating french fries at the bedside in spite of a distended abdomen. I would like to complete my examination with digital rectal exam looking for malena or hard impacted faeces.

What you actually present:

Sir, presenting Mr Akbar, a 49 year old Indian gentleman with Child's C alcoholic cirrhosis and complications of variceal bleeding requiring ICU stay, as well as symptomatic ascites, who now presents a 1-week history of altered mental state. My main issues are:

1. Altered mental state - possibly decompensated cirrhosis but need to rule out structural lesion of left cortical region as it may be an aphasia.
2. Child C's cirrhosis cx ascites and hematemesis
3. Poor compliance to salt restriction as evidenced by eating french fries

Investigations include:

1. Rule out ddx for altered mental state (septic w/u for infection, CT or MRI brain for CVA, electrolytes, glucose for hypoglycaemia); LFT to trend, ammonia. If he later develops abdo pain to do ascitic tap for spontaneous bacterial peritonitis.
2. Also to look for precipitant for hepatic encephalopathy - ensure no drop in Hb to suggest BGIT, consider AFP and liver US to rule out interval development of HCC, unless these were very recently done. GGT & MCV for evidence of continued alcohol ingestion
3. Child's C alcoholic cirrhosis: hep B/C unless already done, look for other complications (platelets, creatinine, PT/PTT)

My management includes

1. For AMS: If no reversible ppt found, to manage as for hepatic encephalopathy, give lots of lactulose, ensure BO 2x a day. Consider thiamine and benzodiazepine if any suspicion that he is still drinking. Put on CIWA charting to watch for delirium tremens.
2. For Child C alcoholic cirrhosis, ascites and varices: need to ensure pt stopped alcohol, consider pharmacotherapy for alcohol use disorder if pt has not stopped alcohol, and vaccinate against hep A/B. Treat complications - may need re-scope to ensure all varices banded, propranolol to reduce portal pressures, spironolactone and furosemide for ascites. Consideration can be given to liver transplant.
3. Noncompliance to salt restriction: need to counsel, find out ideas and expectations. Good social support so need to explain the purpose of salt restriction especially when he has had two ascitic taps.

Cardio: Atrial Fibrillation

ADULTS

Atrial fibrillation is less commonly a standalone case (palpitations), than part of another case (e.g. stroke, valvular heart disease, ischaemic heart disease, cardiac failure, thyrotoxicosis)

Is this atrial fibrillation?

- In most cases quite a straightforward diagnosis once you palpate an irregularly irregular pulse or have the ECG
- To look for paroxysmal AF (not an innocent disease) → do 24h Holter
- For the patient complaining of palpitations → see approaches notes

What is the etiology of AF?

- Explore the cardiac status on hx, examination, and inx.
 - Ischaemic heart disease → symptoms, 2DE for SWMA
 - Valvular heart disease → Auscultate for murmurs, do 2DE. This changes management.
 - Cardiac failure → symptoms, 2DE, NTBNP
 - Recent cardiac surgery.
- Systemic disease: thyroid, sepsis, pulmonary problems
 - If recent onset, look for any precipitant.
 - Do CXR, TFT, septic workup

What is this AF causing?

- Symptoms: palpitations, poor effort tolerance
- Complications e.g. stroke

Do I do rate or rhythm control?

- Choice between rate and rhythm control: similar morbidity and mortality, rhythm control better exercise tolerance for young patients
- Rhythm control: if attempting cardioversion and AF >48h be sure not to cause a stroke -- need transesophageal echo to look for LA thrombus, or at least 3 weeks of anticoagulation before attempting.
 - Electrical: more effective but requires sedation
 - Pharmacological: e.g. flecainide, procainamide (amiodarone less effective)
 - Give maintenance Rx (don't cardiovert and send home - some recur)

- If rate control: is the patient in heart failure?
 - Heart failure: digoxin (less effective), amiodarone (risk cardioversion)
 - No heart failure: beta-blockade (beware asthma), verapamil/diltiazam
 - Please make sure there is no Wolff-Parkinson-White before using AV nodal blockers (beta blocker, CCB, digoxin; caution in amiodarone)
- If drugs fail and symptomatic → consider catheter ablation therapy.
- Complications:
 - E.g. for amiodarone: lung toxicity, thyroid status, liver toxicity, gynaecomastia, photosensitivity, GI side effect
 - All drugs: monitor ECG (brady, QTc prolongation, arrhythmia), electrolytes

Do I need to anticoagulate, and how?

- Anticoagulation is a cornerstone of stroke prevention
- Initiating: decide based on the clinical judgement of risk vs benefit
 - Stroke risk - CHADS-VASc scoring: CCF, HTN, Age (75+: 2 pts, 60-75: 1 pt), DM, past Stroke/TIA (2pt), Vascular disease (AMI, PVD), Sex Cat (female 1pt)
 - Bleed risk - HASBLED scoring: HTN, Abnormal renal/liver function (1pt each), Stroke, Bleeding, Labile INR, Elderly (>65), Drugs/alcohol (1pt each)
- What anticoagulation to use?
 - Aspirin usually not preferred, consider if CHADS-VASc 0-1 or refuse warfarin.
 - Warfarin is 1st line → discuss with patient
 - NOACs: apixaban, dabigatran, rivoxaban → more convenient and no need to monitor but has issues (*not for valvular AF*, more expensive, no reversal agent)
- Monitoring; how is the patient coping
 - Any bleeding
 - Is INR on target (2-3)? Is it labile?
 - Lifestyle: frequent blood test, food and drug interaction → explore patient knowledge of disease, awareness to tell any GP

What are the comorbidities

- AF is rarely the lone disease → Manage the comorbidities as well
- Consider drug drug and drug disease interactions esp if patient is on warfarin.

How is the patient coping?

- Function
- Social
- Financial

Sample summary: *see heart failure*

Cardio: Congenital Heart Disease

PAEDIATRICS

What congenital heart disease is this; how did the child present?

- May be picked up in the neonatal period e.g. cyanosis from birth in ToF.
- Worsening cyanosis and breathless at day 2 of life characterises a duct dependent circulation (e.g. TGA, tricuspid atresia without VSD, hypoplastic left heart). In these conditions, mixing of pulmonary and systemic circulations through the ductus arteriosus is critical to sustain life, physiological closure at day 2 of life causes worsening symptoms.
- Hypotension, poor feeding developing in first 2 weeks characterises defects like CoA (where defect is present at birth and exacerbated once ductus arteriosus closes).
- Symptoms of heart failure (dyspnoea, fluid overload) developing at 2 months characterises a large L > R shunt (e.g. VSD, PDA); at birth, high pulmonary pressures cause little shunting, it is only when pulmonary pressures fall that shunting occurs and symptoms develop.
- Mild disease (e.g. VSD, AS, PDA) may be asymptomatic and/or present with an incidental murmur.

Presentation	Anatomy	Examples
Cyanotic, not breathless > Cyanosis may not be visible, but SpO2 low.	R > L shunt	<ul style="list-style-type: none"> - <i>Tetralogy of Fallot</i>: Overriding aorta, PS, RV hypertrophy, VSD. - Pulmonary atresia with hypoplastic RV and VSD. - Hypoplastic left heart (RV supplies aorta)
Cyanotic, breathless	Common mixing	<ul style="list-style-type: none"> - Transposition of great arteries (TGA) - Complete AVSD / single ventricle
Acyanotic, not breathless > Severe: hypotension > Moderate: syncope > Mild: asymptomatic	Outflow obstruction	<ul style="list-style-type: none"> - AS - <i>Coarctation of aorta</i> - Totally anomalous pulmonary venous return with pulm outflow obstruction
Acyanotic, breathless > Severe: dyspnoea > Mild: asymptomatic	L > R shunt	<ul style="list-style-type: none"> - ASD - VSD - PDA

Please also refer to the table in Medical Short Case > Cardiovascular System (paediatric)

How was diagnosis confirmed?

- ECG: e.g. RVH in ToF
- CXR: e.g. boot shaped heart in ToF, evidence of CCF, LV hypertrophy
- 2DE
- Cardiac catheterization study

Is there any associated syndrome

- Down's
- VACTERL → ask if there are other Vertebral, Anorectal, (Cardiac), Tracheo-esophageal, Renal, and Limb abnormalities

Clinical course & management to date

- Infants with cyanotic heart disease receive extensive neonatal care and may have prolonged NICU care. Duct dependent circulation requires prostaglandin to maintain ductus arteriosus patency.
- Surgical repair may be performed in infancy e.g. TGA (arterial switch), severe CoA
- Surgical repair may also be performed later e.g. AS/PS, CoA, VSD/ASD, specific operations for right heart disease
- Surgery for right heart disease (pulmonary flow obstruction or hypoplastic RV) is often a staged procedure e.g.
 - Blalock-Taussig shunt: subclavian > pulmonary artery (can be done 2x, one on each side)
 - Hemi-Fontan: SVC to PA
 - Fontan completion: IVC to PA.
- Minimally invasive methods are sometimes used e.g. device closure (e.g. VSD, ASD), balloon valvuloplasty (AS, PS), coil embolization (PDA)

Current issues**(1) CCF**

- Are there still symptoms of CCF: shortness of breath, fluid overload.
- Management: fluid restrict, diuretics, digoxin -- how effective have these been?

(2) Is there pulmonary hypertension?

- Can contribute to SOB, lethargy
- If left to right shunt > is Eisenmenger's threatened? → Eisenmenger means that closure can no longer be attempted and prognosis is limited.

(3) Growth and development

- Infants: feeding difficulties in infancy are common and may result in failure to thrive.
- Children: may have poor growth, missed milestones.

Further issues in cyanotic heart diseases:

(4) Endocarditis & prophylaxis

- Incompletely repaired cyanotic heart disease may require Abx prophylaxis

(5) Cyanosis and hypoxia

- Tet spells in ToF: restless, agitated, squat. RVOT spasm worsens obstruction and worsens shunt, causing hypoxia.

(6) R > L embolism in R > L shunts

- Cerebral thromboembolism
- Cerebral abscesses.

Prognosis and support

- Children with cyanotic heart disease often have a stormy course with multiple surgeries, multiple hospitalizations. How is the family coping with this?
- Explore social setup and financial situation.
- If child is of childcare age or beyond, explore coping in school environment; daily activity.

Sample summary: Valentine is a 2 year old girl with congenital cyanotic heart disease, noted to be cyanosed and increasingly breathless on day 2-3 of life which improved with prostaglandin, status post bilateral blalock taussig shunt. She is currently admitted for decompensated congestive cardiac failure secondary to fever for investigation to rule out infective endocarditis. I note that she is failing to thrive, being at the 1st percentile for both height and weight, and that she has isolated gross motor delay. Her fever has responded to empiric broad spectrum antibiotics, and her dyspnea has improved with diuretics so that she was able to take feeds orally today. Plan now is to continue current management, arrange for 2DE and dietician review today, and await results of full septic workup.

Cardio: Heart Failure

ADULTS

Is this heart failure?

- Presentation of heart failure (forward failure: low output, backward failure: congestion) varies according to its etiology and time course:
 - Acute presentation: an acute cardiac event (e.g. AMI, acute valve regurgitation, arrhythmia, hypertensive emergency) may result in hypotension, pulmonary edema, fluid overload.
 - Subacute presentation: mainly exertional dyspnoea, paroxysmal nocturnal dyspnoea, orthopnoea.
 - Chronic presentation: fluid overload presentation, fatigue
- Each presentation requires that differentials are excluded-
 - Acute hypotension → consider other causes of shock (hypovolaemic, haemorrhagic, septic, anaphylactic, PE, etc).
 - Shortness of breath → consider respiratory disease (COPD, interstitial lung disease), anaemia
 - Fluid overload → consider renal disease (ESRF, nephrotic syndrome), cirrhosis
- There is no single diagnostic test; diagnosis is made on the cluster of symptoms (dyspnoea, fatigue), signs (fluid overload, hypoperfusion), and evidence of cardiac dysfunction (e.g. NT-proBNP, 2DE, CXR)

How bad is this patient's heart failure?

- NYHA class: what is the extent of the patient's symptoms?
 - Class 1: *no* SOB on ordinary activity
 - Class 2: SOB on ordinary activity (e.g. housework, walking to bus stop)
 - Class 3: SOB on less than ordinary activity (e.g. bathing, eating)
 - Class 4: SOB at rest
- Latest 2DE -- what is the ejection fraction?
 - Some patients may have heart failure with preserved ejection fraction ($\geq 50\%$) -- diastolic heart failure.
- Time course of exacerbations
 - How severe → requiring admission, ICU?
 - What was the exacerbation like? Conceptually think of the acute hemodynamic state as an intersection of cardiac output and fluid overload:

	Minimal fluid overload - <i>Well compensated</i>	Fluid overloaded - <i>Pulm congestion</i>
Good cardiac output > <i>Good BP</i> > <i>Peripheries perfused</i>	Warm and Dry > <i>Continue current Mx</i>	Warm and Wet > <i>Diuresis</i> > <i>Vasodilators e.g. nitrates</i>
Poor cardiac output > <i>Low BP</i> > <i>AKI, cold peripheries</i>	Cold and Dry > <i>Fluids (judiciously)</i> > <i>Inotropes</i>	Cold and Wet > <i>Diuresis +/- nitrates</i> > <i>Peripheries perfused</i> > <i>Inotropes eg dobutamine</i>

What are my clinical tasks:

(1) Identify etiology of heart failure → and treat.

- This may be obvious clinically or require workup; may be multifactorial.
- Ischaemic heart disease and its risk factors
 - This may be apparent from history (previous AMI, PCI, CABG) or symptoms (angina)
 - Nonetheless all patients should be worked up for IHD (ECG, echo, stress test), and screen for underlying risk factors (fasting glucose, lipids, BP)
 - Any IHD should be treated: antiplatelet, statin [see topic on IHD]
 - Modify risk factors aggressively: stop smoking, lose weight, control sugars, BP
- Valvular heart disease
 - Assessment: 2DE, cardiac MRI
 - Treatment specific to valve: heart failure is often an indication for definitive rx e.g. surgery [see topic on valve dysfunction]
- Arrhythmias
 - Assess ECG
 - Treat if any present e.g. atrial fibrillation → rate vs rhythm control + anticoagulation [see topic on AF]
- Hypertensive heart disease → hypertension increases afterload on the failing ventricle
 - Treat hypertension using evidence-based drugs: ACE-I, beta blocker, aldosterone antagonist (see (3))
 - Consider workup for secondary causes (especially young patient) [see topic on hypertension]
- Thyroid heart disease
 - Measure T4/TSH
 - Treat if present [see topic on Graves' disease]
- Others: cardiomyopathies, infiltrative disease (amyloidosis, hemochromatosis), drugs (e.g. doxorubicin), infection (myocarditis, HIV)

(2) Relieve symptoms and maximise function

- In addition to NYHA class, evaluate function -- of great importance to quality of life.
- Nonpharmacological
 - Dietary modification: salt and fluid restriction, plus as appropriate for risk factor (e.g. DM diet, low fat). Monitor weight
 - Exercise → Does the patient exercise? If no, explore why e.g. fear of cardiac event. Consider formal cardiac rehabilitation programmes
- Pharmacological - initiate carefully and titrate:
 - 1st line -- Furosemide → check K, postural BP, fall risk
 - Digoxin if (1) still symptomatic despite optimal diuresis, ACE-I, beta blocker, and aldosterone antagonist, or (2) AF with inadequate rate control despite beta blockade
- If the patient has exceptionally poor response → re-evaluate diagnosis of heart failure.

(3) Inhibit cardiac remodelling and reduce risk.

- Activated neurohormonal mechanisms initially maintain cardiac output by increasing filling pressures, however, in the chronic state, they cause detrimental remodelling
- Pharmacological therapy for heart failure with reduced EF (not evidence based for preserved EF) - initiate carefully and titrate:
 - ACE-inhibition or ARB: beware renal artery stenosis, monitor for hyperkalemia
 - Beta blocker: use an evidence based beta blocker e.g. carvedilol metoprolol bisoprolol, beware initial worsening of symptoms and asthma
 - Aldosterone antagonist e.g. spironolactone (evidence if NYHA 2 EF \leq 30%, NYHA 3-4 EF $<$ 35%, or previous STEMI EF \leq 40%), monitor for hyperkalemia
 - Additional drugs in certain situations: ivabradine
- Device therapy:
 - Cardiac resynchronization therapy (biventricular pacing): improves survival in patients who are still NYHA class 3-4 despite maximal medical rx
 - Automated implanted cardioverter-defibrillator: as secondary prevention (survivors of sudden cardiac arrest) and as primary prevention in certain cases (very poor EF, hx of dangerous arrhythmia).

(4) Prevent exacerbations

- Explore the precipitants for previous exacerbations
 - New cardiac insult: ischaemia, arrhythmia
 - Non-cardiac disease: renal disease, infection
 - Noncompliance to fluid restriction or medication: hypertension, fluid overload.
- Address precipitant accordingly e.g.
 - Immunizations
- Avoid drugs that worsen heart failure: NSAIDs, thiazolidinediones, metformin if eGFR \leq 30

(5) Treat comorbid and medication issues

- Heart failure patients are often older, with multiple medical problems and polypharmacy; a holistic multidisciplinary approach is particularly crucial
- Explore medication compliance and motivation → if not compliant, why?
- Explore medication side effects e.g. postural hypotension, falls
- Be wary of disease-disease, drug-disease, drug-drug interactions. Red flags:
 - Interactions with warfarin
 - Drugs acting on potassium levels
 - Nitrates and sildenafil

How is this affecting the patient?

- Ability to stay employed or engage in leisure activity
- Social interaction
- Psychological: medications, uncertainty of prognosis, activity and diet restrictions often take a toll
- Financial

Sample summary: Mdm Poh Xin Men is a 85 year old lady with NYHA II congestive cardiac failure secondary to severe mitral stenosis and atrial fibrillation on a background of rheumatic heart disease. She has had innumerable admissions for decompensated CCF over past 15-20 years, usually precipitated by intercurrent illnesses or non-adherence to salt and fluid restriction. Her latest ejection fraction is 25%. She is already on best medical therapy (furosemide for symptoms; ACE-I, beta blockers and spironolactone to prevent disease remodelling; beta blockers for rate control of AF and warfarin for anticoagulation) and has already previously decided she does not want to go for mitral valve replacement. She is ADL-independent and able to take her own medicines, but is mostly home bound. She stays with her daughter and son-in-law, both of whom dote on her and support her financially. My issues for her are:

- (1) Decompensated CCF
- (2) Severe mitral stenosis complicated by AF
- (3) B/g rheumatic heart disease
- (4) Non compliance to salt and water restriction

Cardio: Ischaemic Heart Disease

ADULTS

Where in the spectrum of IHD is this patient?

- IHD rears its head in many ways - it is crucial to accurately understand *this patient's* disease because management tasks are drastically different in each:
- Which chest pain syndrome does the patient have?
 - Stable Angina: episodic chest pain triggered by exertion, relieved with 3-5min of rest or sublingual nitrates → Continue chronic Mx.
 - Unstable Angina: increasingly severe angina (more severe, more frequent, takes less to precipitate), angina at rest, or new-onset angina → This portends a coming AMI so Investigate and treat intensely.
 - Acute coronary syndrome: acute chest pain, worse than usual angina, lasting >30min, with dyspnoea, diaphoresis, vomiting. This includes unstable angina, NSTEMI, and STEMI → See acute management.
- What is this patient's cardiac history?
 - Previous episodes of AMI
 - Previous coronary intervention → Stent or bypass can still thrombose!
 - Previous episodes of dangerous arrhythmias, collapse (e.g. VT, VF)

What is the patient's cardiac status and complications?

- Functional status and change over time.
- Heart failure: any symptoms (dyspnoea, LL swelling), what is latest EF? (see heart failure section)
- Arrhythmias: e.g. atrial fibrillation
- What are the latest investigations
 - Imaging: echocardiography, MRI/MIBI
 - Stress testing: treadmill ECG vs stress echo (exercise or pharmacological)

How can I optimise this patient's cardiac status?

- Goal of therapy in stable IHD: alleviate symptoms, prevent progression, decrease the risk of adverse outcomes (death, AMI, heart failure)

(1) Symptomatic relief

- 1st line -- Beta blockers, preferring cardioselective agents (metoprolol, atenolol)
 - Evidence-based to improve survival post-MI
 - Cautions: asthma, heart failure, bradycardia or heart block.
 - Safe in DM, amiodarone use
- 2nd line -- calcium channel blockers, if beta blocker unsuccessful, or contraindicated, or not tolerated (diltiazem, verapamil, amlodipine; avoid nifedipine)
- Nitrates -- 1st line for acute angina (GTN), 2nd line as add on chronic therapy to beta blockers (isosorbide dinitrate, transdermal nitroglycerin)
 - Avoid in right ventricular MI
 - Please make sure patient does not take sildenafil

(2) Risk factor modification

- Assess for and modify cardiac risk factors
- Low-hanging fruits:
 - Smoking → Stop smoking, refer to cessation clinics
 - Obesity → Lose weight
 - Diet → Reduce salt, reduce fat, reduce sugar.
 - Sedentary lifestyle → Exercise prescription, refer cardiac rehab
 - Hyperlipidemia → Invasive statins regardless of LDL levels
- DM → Screen and treat; see DM text
- Hypertension
 - Look for secondary cause, especially if young patient: renal artery stenosis (renal artery doppler), GN or CKD (UECr), Conn's syndrome (UECr for hypoK), Cushing syndrome (look), obstructive sleep apnoea (ask), coarctation of aorta (measure 4 limb BP).
 - Watch for fragile patients → those prone to postural hypotension and falls, as well as those who tend to present with hypertensive urgency (or emergency e.g. CVA, dissection, AKI, retinopathy)
 - Patients who have had an MI should receive ACE-I and beta blocker regardless of BP control

(3) Secondary prevention

- 1st line -- Aspirin 100mg OM + omeprazole cover
- Clopidogrel if allergic to aspirin.

(4) Revascularization

- May be performed in acute setting -- NSTEMI, STEMI
- Elective revascularization in -
 - Activity limiting angina despite best medical therapy
 - High risk lesion (likely survival benefit from revascularization): left main disease, triple vessel disease, two vessel disease with severe LAD stenosis
 - Active patients who prefer revascularization for improved quality of life compared to medical therapy

- Options: PCI vs CABG
 - PCI: single vessel disease involving the right or circumflex coronary arteries, short segment, anatomically feasible for PCI.
 - CABG: triple vessel disease, left main disease, two vessel disease with severe proximal LAD stenosis
- After revascularization give dual antiplatelet therapy: e.g. 1 year (drug eluting stent) or 3 months (bare metal stent), thereafter single antiplatelet. (although latest evidence suggests DAPT is for life).

(5) Treat comorbidities and complications

- Treat heart failure (see heart failure) → very often the same drugs above
- Treat arrhythmias e.g. AF (see AF)
- Other comorbidities

How is the patient affecting the disease?

- Worth exploring patient perceptions about disease
- Consider compliance to lifestyle and pharmacological measures.

How is the disease affecting the patient?

- ADL, ambulation status - has it been deteriorating? Is this affecting his function?
- Psychologically, how is the patient dealing?
 - Patient's understanding and attitude towards condition is important as this influences patient motivation to participate in rehabilitation AND control cardiovascular risk factors
- Social support?
- Financial support?

Sample summary: Ee Li Fen is a 70 year old retiree who presents with episodic chest pain which worsened in frequency and severity over 2 months. She was diagnosed to have unstable angina and received percutaneous coronary intervention a week ago, after which she has been symptom-free. She had no prior past medical history but on workup was found to be hypertensive and diabetic; otherwise she has no peripheral vascular disease, strokes; her renal function is normal and she is not proteinuric; she does not have peripheral neuropathy; she also has no symptoms of exertional dyspnoea or pedal edema. She is on long-term aspirin, bisoprolol, losartan, metformin, glipizide, and atorvastatin. Functionally she is now symptom free and has no functional limitation. She has good social support.

Appendix: acute management of acute coronary syndrome

Immediate Management

- Assess ABC, attach cardiac and oxygen saturation monitors, give supplemental oxygen as needed to keep SpO₂ >90%, ensure IV access
- Obtain 12 lead ECG within 10min of arrival, repeat 10-15min if initial ECG nondiagnostic but clinical suspicion high
 - Treat sustained ventricular arrhythmias according to ACLS
- Send bloods: cardiac enzymes, RP, FBC, PT/PTT
- Load aspirin 300mg, chewed and swallowed (unless aortic dissection is being considered)
- Give sublingual GTN tablets (0.4mg tablet every 5 min x3) or spray (one spray every 5 min x3) for relief of acute angina
 - If patient has persistent chest discomfort, hypertension, signs of heart failure
 - And if NO right ventricular infarction on ECG, no hemodynamic compromise
- STAT dose of atorvastatin 80mg preferably before PCI
- Give cardioselective beta blocker (PO metoprolol 25mg)
 - If no signs of heart failure, no bradycardia, no hemodynamic compromise, or severe reactive airway disease
- Give morphine for persistent pain (2-4mg slow IV push every 5-15min)

Definitive Management

(A) STEMI

- Decide if patient is for revascularization
 - Percutaneous coronary intervention within 90 minutes (door to balloon time)
 - IV thrombolysis within 12 hours of symptoms
- Oral antiplatelet therapy for all patients
 - Prasugrel 60mg stat, 10mg OM continue for 3/12
 - Age <75, BW60kg, no prior CVA, TIA, ICH
 - Clopidogrel 600mg stat, 75mg om continue for 1 year
 - Age >75, BW <60, prior CVA, TIA, ICH
- Anticoagulation therapy for all patients

(B) NSTEMI/Unstable angina

- Decide if patient is for early revascularization (cath within 48 hours)
- Oral antiplatelet therapy for all patients
 - If patient is for early cath: Ticagrelor 180mg stat, 90mg BD continue ticagrelor 90mg BD x 1 year (for raised CE (high risk), no prior CVA TIA or ICH)
 - If patient is NOT for early cath: Clopidogrel 600mg stat, 75mg om continue for 1 year (for Negative CEs (low risk), no prior CVA TIA ICH)
- Anticoagulation therapy for all patients: SC clexane 1.5mg/kg

Derm: Psoriasis

ADULTS & PAEDIATRICS

Is this psoriasis?

- Describe and delineate the extent & distribution of skin lesions
- Classic description: erythematous papules or plaques with silvery scales, classically affecting scalp, elbows, knees, back, usually in a rather symmetrical distribution. Positive auspitz sign (removal of scales reveals small bleeding points) and demonstrates Koebner phenomenon (skin trauma aggravates lesions).
- Identify the subtype and consider ddx.
 - Chronic plaque psoriasis: ddx discoid eczema, tinea corporis (offer to scrape)
 - Erythrodermic psoriasis: ddx eczemas, drug eruption, cutaneous lymphoma
 - Guttate psoriasis: ddx pityriasis rosea, secondary syphilis
 - Palms and soles psoriasis: ddx hand/feet eczema
 - Scalp psoriasis: ddx seborrheic dermatitis
 - Pustular psoriasis: ddx AGEP (drug reaction)

Are there any other manifestations?

- Psoriatic onychodystrophy: pitting, onycholysis, discolouration
- Psoriatic arthropathy (5 subtypes):
 - Asymmetrical oligoarticular
 - RA-type
 - OA-type
 - Spondyloarthritic/AS-type
 - Arthritis mutilans

What has the course and management so far been like?

- Has the patient required admission before? Any flares?
 - Triggers: infections, withdrawal of systemic steroids (any TCM?), post-partum deterioration, trauma, HIV infection
- What treatment has the patient received so far? Have the skin lesions/arthropathy responded?

How can I optimize treatment?

(a) Nonpharmacological

- Patient education & correct misconceptions. Disease is noncontagious, unpredictable, not scarring, treatable, incurable, not influenced by diet.
- Control/screening for comorbidities (DM, HTN, HLD, metabolic syndrome)
- Avoid smoking and alcohol
- Avoid precipitants of flares

(b) Specific therapy

- Topicals: Coal tar, Dithranol, Topical steroids, Calcipotriol/calcitriol, Topical calcineurin inhibitors (and as always, emollients)
- Phototherapy: 1st line for moderate-severe psoriasis (>10% BSA) or poor response to topicals or severe impact on quality of life.
 - Usually narrow band ultraviolet B, alternative psoralen + ultraviolet A (PUVA).
 - 2-3x/week, slowly build up from 1 min → 1.5min → 5 min per session to overcome skin's natural resistance to light
- Systemic: methotrexate (esp if joint involvement), retinoids, cyclosporine A, biologics (e.g. infliximab, adalimumab)

Have there been any complications of treatment?

- Phototherapy: SCC
- Liver toxicity on methotrexate or retinoid
 - Monitor LFT
- Agranulocytosis on methotrexate
 - Monitor FBC
 - Give folic acid
 - Check - does patient know what to do if fever?
- Complications of immunosuppression
 - Are there recurrent infections?
 - Prophylaxis: vaccinate
- If young female on retinoids or methotrexate - is the patient planning pregnancy? Both are teratogenic so if considering pregnancy need to switch; if not considering, check that contraception is adequate

How is the patient coping overall?

- Patient's understanding: of disease -- what are the concerns and fears? and of treatment -- competent?
- Psychological: (esp in young female patients) how affected is the patient by the skin lesions? does the patient resort to hiding the lesions?
- Social: does the patient face discrimination from friends, in school or at work? does being mindful of the skin lesions limit the clothes/activities the patient is willing to try?
- Functional: Able to work? Able to take care of self?
- Financial

Endocrine: Cushing's Syndrome

ADULTS & PAEDIATRICS

What are the visible features of Cushing's Syndrome?

- Central obesity
- Facial adiposity, supraclavicular adiposity, dorsal-cervical adiposity
- Thinned skin, easy bruising, abdominal striae, acne, female balding
- Hirsutism (rapid development of deepening of voice, frontal balding, male pattern hair, acne, increased muscle bulk, oligomenorrhoea, clitoromegaly, etc) should prompt suspicion of an adrenal tumor.
- Hyperpigmentation indicates high ACTH.

What is the cause of Cushing's?

(a) Are there any exogenous sources of corticosteroids?

- *****Always rule out exogenous causes first*****
- Any co-morbid conditions that require long term use of steroids?
 - Autoimmune (RA, SLE, IBD, MS), Renal (Nephrotic, RPGN), Respi: Asthma, Haem (ITP), Transplant patients
 - Is there any way to use steroid sparing agents instead?
- Does the patient take TCM or other supplements?

(b) If no exogenous source, likely endogenous source - proceed with workup for Cushing's syndrome.

- Endogenous causes include pituitary adenoma, ectopic ACTH secreting tumour, adrenal tumour.
- *Confirm Cushing's*: high 24h urinary free cortisol, or failure to suppress cortisol secretion with low dose dexamethasone.
- *Localize source of hypercortisolism*: serum ACTH levels (also looking for hyperpigmentation) and high dose dexamethasone test or CRH stimulation.
 - Adrenal tumor: Low ACTH (suppressed by high cortisol levels) -- may be adrenal adenoma or carcinoma > CT adrenal.
 - Pituitary adenoma: High ACTH, but suppressible with high dose dexamethasone (since pituitary is still responsive to high dose dex) > MRI pituitary, test other pituitary hormones (may be low)
 - Ectopic ACTH: High ACTH, not suppressed by high dose dexamethasone > often a lung primary, CT thorax.

How shall we proceed with management?

- If exogenous source, but patient is steroid dependant:
 - Attempt steroid sparing agents
 - If need to continue steroids, deal with complications of Cushing's syndrome as they come
 - Give patient stress doses of steroids during any procedure to avoid adrenal insufficiency
- If exogenous source which can be tailed down (e.g. steroid sparing agents, can stop TCM): gently tail down to prevent adrenal insufficiency.
- If endogenous source: surgery

What are the other complications of Cushing's Syndrome? --- often additional issues that need to be addressed.

- Metabolic: *Hypertension, hyperglycemia* --- screen and treat.
- MSK: *Osteoporosis*, avascular necrosis of hip, proximal myopathy --- should be on vit D / calcium, screen for osteoporosis, treat if osteoporotic.
- CNS: agitated *depression*, anxiety, irritability, emotional lability, psychosis, impaired cognition
- GI – steroid induced ulcers and BGIT
- Immune: *immunocompromised* state --- vaccinations (influenza, pneumococcal), screen hep B/C/HIV, TB, if high dose steroid need to know what to do if sick.
- Menstrual irregularity, increased libido
- Eye: *glaucoma, cataracts* -- may need ophthalmology screening

Sample summary: Yue Liang is a 32 year old lady with immune mediated thrombocytopenia which first presented 10 years ago with non-palpable purpura, menorrhagia, and gum bleeding. In terms of ITP she has been very stable with a baseline platelet count maintained around 50-60s, on prednisolone 20mg/day. However she is very steroid dependant; each time her haematologist attempts to tail down steroids her platelets plunge below 10 and she bleeds. Indeed she has become Cushingoid, and she is particularly concerned about her large body habitus, acne, and gradual hirsutism has had negative ramifications on her dating life. The other issue is that she has had symptoms of polyuria/polydipsia recently which I wonder could be steroid-induced hyperglycemia.

Endocrine: Diabetes

ADULTS & PAEDIATRICS

What is the patient's clinical course to date?

- When was the patient diagnosed?
 - Fulminant presentation with DKA/HHS, stupor → type 1
 - Indolent presentation: polyuria, polydipsia, +/- lethargy → often type 2
 - On screening or on admission for a comorbidity.
 - Pregnancy: gestational DM.
- Diagnostic criteria: 1 test if symptomatic or 2 tests if asymptomatic -- Random glucose >11.1 mmol/L, OGTT >11.1 mmol/L, fasting glucose >7.0 mmol/L
- Distinguish T1DM vs T2DM
 - Body habitus, age of onset, insulin requirement, acanthosis nigricans (T1DM)
 - Review diagnostic workup: e.g. if suspecting T1DM, anti-islet cell antibody, anti glutamic acid decarboxylase antibody, insulin levels (low in T1DM) C-peptide levels (low in T1DM)
- Any contributory factors to hyperglycaemia e.g. chronic steroid use, TCM?
- Have there been any admissions for diabetic emergencies?
 - If so, why? Non-compliance? Triggered by intercurrent illness?
 - DKA = BG >15mmol/L, pH<7.3 or HCO₃ <18, ketones>0.6
 - HHS = BG >33, serum osmolality >320 (2Na + glucose)

How has the control been?

- Lifestyle: has there been lifestyle modification: diet, exercise, weight loss
- What pharmacological therapy is the patient currently on
 - OHGAs: start with metformin, then a 2nd drug
 - Insulin: basal-bolus vs mixtard vs OHGA + topup regimen (mixtard not appropriate for T1DM)
 - Does this fit well into lifestyle?
 - Is patient competent in taking medicine?
 - Is patient compliant to medicine and home glucose monitoring?
- What is the daily glucose trend and HbA1c?
- For T1DM: is patient aware of what to do if he/she is sick and unable to eat? (NOT stop insulin -- give basal doses only and monitor with glucometer, smarter patients can be taught to titrate accordingly)

Do I need to adjust therapy?

- Adjustment of pharmacological therapy proceeds concomitant to lifestyle measures
- First address the 'hypos'
 - Does have hypo episodes e.g. giddy, diaphoresis?
 - If yes, why? > Irregular meals?
 - If no, true no or hypoglycaemic unawareness? → do home glucose monitoring.
 - Is the patient aware of what to do in hypoglycaemic episode?
 - Any hypos, change regimen (e.g. early morning hypo -- decrease night dose)
- Then look at the 'hypers'
 - Why hyper? Missed meds dose, patient gave himself a sweet treat?
 - If post-meal hyper, there is insufficient pre-meal insulin
- Look at overall glucose control: HbA1c (target <7% in most, <8% in old)
- Do we need to step-up therapy?
 - If on OHGA, is patient amenable to insulin?
 - If on mixtard BD, this is often difficult to titrate with post-lunch hypers and early morning hypos > is that acceptable or change to basal-bolus?
 - Consider convenient options e.g. insulin pens, pumps esp younger well-educated pts (but costly)

What DM complications are present and how are they managed?

- Microvascular: regular screening starting 5 yr after diagnosis (T1DM) or immediately (T2DM) is important because intervention can be performed
 - Retinopathy: has DM eye screen been done, if not, do.
 - Neuropathy: yearly DM foot screen; any ulcers?
 - Nephropathy: is there established CKD? If no, screen albumin-creatinine ratio for microalbuminaemia (abnormal: >3 mg albumin per mmol creatinine) → inhibit proteinuria with ACE-I
- Macrovascular: strokes, ischemic heart disease, peripheral vascular disease
- Immunopathy e.g. recurrent abscesses, DM foot infections

What comorbidities are present?

- [Adult] Other cardiovascular risk factors: HTN, HLD and the medications/control for those --- treat everything.
- [Child] Other autoimmune conditions: myasthenia gravis, grave's/hashimoto's, pernicious anemia (and family history for those)

How is the overall condition of the patient?

- [Child] Growth and development, participation in school
- Interference with work and function: e.g. having to inject insulin, complications (amputation, strokes)
- Social setup
- Financial situation

- Young ladies -- issue of pregnancy
 - Existing children -- any issues?
 - Is the patient sexually active and what contraception is used?
 - Is patient aware that pregnancy may be hazardous to mother and baby If DM control is poor?
 - Refer to high risk pregnancy specialist.
- Patient understanding of the condition and treatment

Appendix: Worked examples in titrating diabetes meds based on patient’s trend.

Notes:

- These are for outpatient use -- inpatient DM goals are different -- simply to avoid DM emergencies, not to stabilize outpatient management.
- Don’t titrate based on a single day’s reading, look at several readings and the HbA1c (beware: patients often control their diet very well when it’s monitoring day, and cut some slack otherwise!)
- Assume that diet factors are taken out → e.g. patient did not miss a meal, treat himself to ice cream, etc.

Examples:

Current Rx	Breakfast		Lunch		Dinner		10pm
	Pre	Post	Pre	Post	Pre	Post	
1. Metformin (max dose)	10	14	10	15	11	16	10
2. Metformin + Glipizide BD (max dose)	10	14	10	15	11	16	10
3. Metformin + Glipizide BD (max dose)	3	9	5	10	4	8	5
4. Mixtard BD	3.5	9	11	17	11	13	9
5. Mixtard BD	10	14	10	15	11	16	10
6. Lispro TDS premeal + Detemir ON	10	14	10	15	11	16	10
7. Lispro TDS premeal + Detemir ON	6	12	7	16	6	17	6
8. Lispro TDS premeal + Detemir ON	4	7	3.5	8	4	9	6

1 & 2: overall control is poor, glucose is too high throughout. Check diet, remove steroids, and consider step-up therapy e.g. add glipizide to (1), add a 3rd OHGA or basal long-acting insulin to (2), switch (2) to insulin regimen.

3: Control is too tight. Loosen up a bit! Reduce glipizide especially > metformin does not cause hypo.

4: This is the usual difficulty with mixtard: post-lunch hyperglycaemia and early morning hypo. This is because each mixtard dose is a calculated trade-off to avoid a 2nd injection, e.g. for the morning dose, the actrapid covers the breakfast glucose, while the insulatard covers the lunchtime sugar intake; for the evening dose, the actrapid covers dinner and insulatard tides through the night. Hence, for example, an insufficient evening mixtard dose results in post-dinner hyperglycaemia, while too high a evening mixtard causes nocturnal / early morning hypos. In this situation the principle is to tackle the hypos first -- reduce the evening dose! However this will cause post-dinner glucose to go too high. If acceptable control cannot be achieved without significant hypoglycaemia risk, it may be better to switch to basal bolus regimen.

5: All readings too high throughout the day > increase both am and pm mixtard dose (if only high post-breakfast till pre-dinner, increase morning dose only; if only high post-dinner till pre-breakfast, increase evening dose only)

6: All readings too high > Increase basal insulin. Increasing basal insulin will result in a global decrease in all readings (since basal acts all the time), while increasing bolus insulin will only decrease post-meal readings (since this is short acting).

7: Post-meal readings too high but pre-meal acceptable > Increase bolus i.e. pre-meal insulin.

8: All readings too low > decrease basal insulin. This often arises as CKD progresses and insulin excretion falls.

Endocrine: Graves' Disease

ADULTS & PAEDIATRICS

How did this patient present?

- Classic Graves' disease in a young lady who presents with an insidious onset of symptoms of hyperthyroidism (heat intolerance, sweating palpitations, anxiety, weight loss, diarrhoea, oligomenorrhoea; tremors, tachycardia), a soft diffuse goitre, and Graves' eye signs.
- Older patients present less classically, at times simply with weight loss or atrial fibrillation
- The occasional unfortunate patient presents with a complication e.g. thyroid storm or stroke due to atrial fibrillation.
- Some patients may have +ve family history, or personal history of other autoimmune disease.
- Initial workup → is the diagnosis of Graves confirmed?
 - Expect: high T4 low TSH with positive thyroid stimulating antibody
 - If initially hyperthyroid then hypothyroid, consider thyroiditis e.g. Hashimoto which has initial hyperthyroid phase.

What is the current thyroid status & can I optimise?

- What is the treatment to date
 - Options are: thioamides (carbimazole superior to PTU) vs radioactive iodine vs surgery
 - Explore patient preference, why was this chosen.
- What is the current thyroid status
 - Is the patient clinically euthyroid: are there symptoms & signs of hyper or hypothyroidism?
 - Is the patient biochemically euthyroid: what is latest TSH/T4?
 - Is there a large bothersome goitre?
- Any complications of treatment
 - Agranulocytosis
 - Liver toxicity
 - Does patient know what to do if fever?

How can I optimise management?

- If patient on medical Rx is not euthyroid --
 - Due to noncompliance? > Explore compliance, if not, why?
 - Due to dosing? Titrate thioamide + add beta blocker if hyperthyroid
 - Difficult to control → Consider RAI.
- If maintained on medical Rx or new diagnosis:
 - If long-term medical Rx > can I try stopping? (50% will not relapse)
 - Is there a role for radioactive iodine? > Especially if difficult to titrate medical therapy, patient bothered by burden of compliance, and pt non pregnant.
 - Is there a role for surgery? > Especially if fail RAI, bothersome goitre.
 - Discuss patient preference, esp understanding that RAI may result in hypothyroidism later → requiring replacement thyroxine.

Are there complications:**(1) Eye disease:**

- Assessment: Is there exophthalmos, ophthalmoplegia? Is the patient troubled by cosmesis, diplopia?
- Are there serious complications e.g. corneal ulceration due to proptosis, threatened loss of vision?
- Management: eye disease may take a separate course from systemic thyroid status. Consider ophthalmology referral.
 - Stop smoking
 - Artificial teardrops
 - Avoid radioactive iodine: worsen eye disease.
 - Prednisolone
 - If necessary - rituximab, orbital decompression surgery

(2) Atrial fibrillation or cardiac disease

- Is this AF in un- or under-treated disease (often resolves once T4 normalise), or persistent AF in euthyroid state (may not resolve)?
- Any complications e.g. stroke; any impairment of function
- Manage as per non-valvular AF in other patients → rate vs rhythm control + anticoagulation.

(3) Any past episodes of thyroid storm?

- What was the precipitant? → noncompliance, infection
- Any complications -- congestive cardiac failure, requiring ICU stay, etc
- Acute management: thioamide, hydrocortisone, beta blocker (controversial), lugol's iodine (1h after thioamide)

Is patient considering pregnancy?

- If yes → thioamides may be teratogenic
 - Consider radioactive iodine before pregnancy, or use propylthiouracil over carbimazole in 1st trimester.
 - Refer to O&G who specializes in high risk pregnancy.
- If no → counsel on teratogenicity of thioamides, use of contraception

How is the patient coping?

- Function: ability to work
- Social interactions: any issues due to anxiety, irritability?
- Financial status

Sample summary: Mr Kan Chiong is a 44 year old Chinese driver who was diagnosed 3 months ago with Grave's disease when he presented with fever, diarrhea, heat intolerance, mood changes. He was initiated on carbimazole and sent home, but defaulted follow up and medications as he felt he could not afford them. He has currently re-presented with thyrotoxicosis complicated by congestive cardiac failure secondary to atrial fibrillation, likely precipitated by URTI and non-compliance. CCF has resolved and symptoms have improved with carbimazole and propranolol, but he is persistently hyperthyroid and in rate controlled atrial fibrillation. Warfarin was initiated. The team has give him the option of RAI to achieve better control, especially since he is not keen on lifelong carbimazole and warfarin, and he does not have thyroid ophthalmopathy. My issues for him are:

- (1) Thyrotoxicosis
- (2) Atrial fibrillation secondary to thyrotoxicosis, complicated by CCF
- (3) URTI (resolved)
- (4) Non-compliance to medications secondary to financial issues
- (5) Financial issues, a/w MSW input

Gastro: Biliary Atresia

PAEDIATRICS

How did this child present?

- Classic presentation is *prolonged* neonatal jaundice with *pale* stools, conjugated hyperbilirubinaemia.
- Is the diagnosis of biliary atresia confirmed -- if not, consider ddx (see end)
 - Ultrasound: CBD not visible, triangular cord sign, abnormal GB size/shape
 - Hepatobiliary scintigraphy (HIDA scan) - failed excretion of tracer from liver to bowel
 - Intra-op cholangiogram
- Differential diagnoses for biliary atresia: consider based on time of onset (early <24h vs prolonged >2 weeks) and whether conjugated (pale stools, >15% direct bilirubin)
 - Early onset: hemolysis (ABO, Rh, G6PD, spherocytosis), TORCH -- antenatal history and maternal blood types.
 - Prolonged & conjugated: Biliary atresia; neonatal hepatitis
 - 24h-2wk: Infection e.g. UTI, Hypothyroidism, hepatitis
 - Physiological jaundice, breast milk jaundice

Has the child gone for Kasai procedure?

- Kasai is a hepato portoenterostomy (create roux loup and anastomose to liver hilum)
- At which day is Kasai done? This is prognostically important, Kasai < 60 days has better outcome

Was Kasai successful?

- Success rate: 1 in 3 succeed, 1 in 3 progress to cirrhosis, 1 in 3 fail from outset -- require transplant.
- Is there residual jaundice? > Ursodeoxycholic acid may help (no evidence)

How has the patient's course been post-op

- Cirrhosis and its complications: Ask if the child has had variceal bleeding, ascites, coagulopathy, or is puritic. Examine for stigmata of chronic liver disease. Intervene as for cirrhosis (see cirrhosis approach)
- Cholangitis is common due to altered biliary tree anatomy and stasis in ascending limb of roux-en-Y. Ask how many episodes have occurred and how were they treated? Inquire whether parents know what to do

Is the child's growth and development otherwise alright?

- Poor nutrition is common due to cholestasis and liver inflammation.
 - How has the child's growth been?
 - What has been done -- concentrated feeds, fat soluble vitamin supplements, nasogastric feeds?
- Developmental milestones
- School performance
- Puberty

Is transplant necessary and has it been considered?

- Necessity of transplant depends on development of cirrhosis, complications, and poor growth
- If considered -- has transplant workup been done, any donor available?

How is the patient and family coping with this illness?

- Time off school/work
- Financial cost can be immense
- Psychological cost - a child who feeds poorly on good days and gets cholangitic on bad days is very draining.

Sample: Xiao Huang is a 6-month old Chinese boy who first presented with prolonged neonatal jaundice at 1 month of age. Workup revealed biliary atresia and he underwent Kasai procedure at 6 weeks with resolution of jaundice. Since then he has had two episodes of cholangitis. He has been growing well and meeting all milestones. He has a supportive social setup. My main issues are:

1. Recurrent cholangitis
2. Biliary atresia s/p Kasai

Gastro: Chronic Hepatitis

ADULTS

How did this patient present?

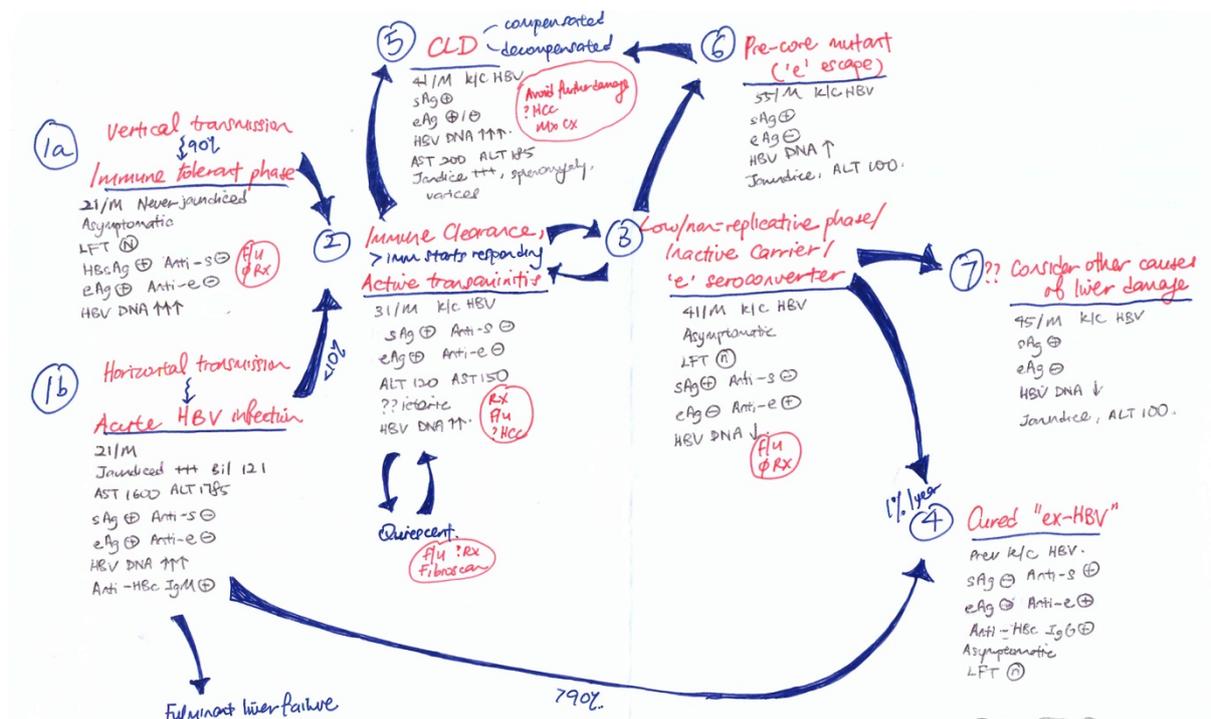
- Asymptomatic on screening
- Acute hepatitis
 - This may be a flare of previously immunotolerant disease (more common), or represent new infection
 - How severe? Usually mild, occasionally fulminant liver failure with encephalopathy, coagulopathy.
 - Consider ddx: obstructive jaundice, cholangitis, alcoholic, drugs, autoimmune
- Other manifestation: e.g. glomerulonephritis
- How was the diagnosis made?
- Is this hepatitis B or C?

Why does this patient have hepatitis?

- Vertical transmission most common unless young → ask for family history
- Acquired infection → not all present as icteric hepatitis (Hep B > Hep C); 5% of new Hep B and 60% of new Hep C develop chronic hepatitis. Look for:
 - High-risk sexual behaviour → take sexual hx
 - IV drug use
 - Blood transfusions e.g. overseas.
 - Occupational
- If there are risk factors, screen for other blood borne viruses
- For hep B: was the patient vaccinated?

Where is this patient in the course of disease?

- Hepatitis B: at each visit evaluate LFTs, serology and viral load to gauge where in the disease this patient is (see chart and text below)
- Hepatitis C: these patients do not usually get 'flares', disease progresses slowly to cirrhosis.



The natural history of Hepatitis B

- Immune-tolerant phase: young patient with perinatal infection and has never had flares (immune system does not recognize disease) -- LFTs normal, HBeAg +ve, high viral load (can be HBeAg negative if pre-core mutant)
- Immune clearance phase: immune system has started recognizing virus causing episodes of flares -- LFTs fluctuate, HBeAg +ve then seroconverts to HBeAg -ve, viral load falls (can be HBeAg negative if pre-core mutant)
- Nonreplicative / carrier phase: a patient who has had episodes of flares during which HBeAg seroconversion occurred, and is now flare-free -- LFTs normal, HBeAg -ve, viral load low. Some of these patients may go on to lose the HBsAg and are considered cured.
- End-stage with liver damage and cirrhosis: see approach to cirrhosis

What are the issues and how to manage?

(1) Flares

- Ensure that it is purely a hepatitis viral flare
 - Expect hepatocellular pattern of LFT derangement
 - Rule out alcohol use, autoimmune liver disease, non-hepatitis viruses (e.g. dengue), drug induced liver toxicity.
- Avoid precipitating flares
 - Vaccinations: hepatitis A vaccination, hepatitis B vaccination (if hep C)
 - Avoid hepatotoxic drugs.
 - Is the patient still drinking? → Stop! Get help if cannot stop.

(2) Complications and their management

- Cirrhosis
 - If no cirrhosis: monitor clinically, fibroscan, biopsy
 - If cirrhosis: manage complications (see cirrhosis document)
- Screening for hepatocellular carcinoma (various protocols) e.g. ultrasound, AFP
- Look for extra-hepatic manifestations: the hepatitis viruses can cause a wide range of extrahepatic manifestations, e.g.
 - Membranous glomerulonephritis
 - Palpable purpura / vasculitic rash.
 - Hep C is associated with other autoimmune diseases e.g. thyroiditis, ITP, etc.

(3) Direct viral control

- New breakthroughs in antiviral drugs have transformed the management of chronic hepatitis however cost is a big issue. Drugs include (will not discuss how to choose between the drugs)
 - Hep B: interferon, lamivudine, entecavir, tenofovir (check HIV first, don't inadvertently give HIV monotherapy and cause resistance!)
- Approach for hepatitis B: treatment is long-term, controls disease and reduces risk of progression to cirrhosis - but not a cure. Need to think carefully who to treat, and discuss with patient; once committed to treatment it can be for life.
 - If advanced fibrosis with high viral load, cirrhosis → Treat
 - Acute liver failure → Treat
 - Patient will need chemotherapy or immunosuppression → Treat (prevent flare)
 - Recurrent flares → Discuss with patient:
 - HBeAg positive: Rx reduces flares but is likely a long-term affair; without Rx such patients have the potential to spontaneously undergo 'e' seroconversion and move to immune carrier phase, but this is unlikely to happen on treatment
 - HBeAg negative: may be pre-core mutant likely -- body is unlikely able to control disease by 'e seroconversion'
 - Immune tolerant phase, inactive carrier phase → Do not treat
- Approach for hepatitis C: antiviral therapy can eradicate HCV RNA and lead to a cure
 - Need to know genotype → determines choice of direct acting antiviral
 - Direct acting antivirals are given for a fixed duration and result in excellent SVR rates 80-100%.
 - SVR: Test for HCV RNA at 12-24 weeks after stopping treatment, if none, this is sustained virologic response = cure (99% chance of being HCV RNA negative during long-term follow-up).
 - Treatment benefits almost all patients but high cost is a big issue

(4) Public health and transmission

- Vaccinate household members
- No blood donation → Explore awareness and counsel
- Is patient sexually active?
 - Committed partnership: for hep B, partner should be vaccinated and test antibody
 - Casual partners: counsel on use of protection (even if partner is immune, risks other STDs)
- Does patient intend pregnancy?
 - Neonate must be vaccinated with active immunization, plus passive immunization if 'e' antigen positive (i.e. transmissible)
 - Need to discuss with obstetrician.

How is the patient coping?

- This depends greatly on where the patient is in the disease course
- Occupation: time off, flare episodes, implications if healthcare staff
- Financial: important because of the high cost of antiviral drugs - is the patient able to afford it?

Sample summary: Gan Yan is a 30 year old Chinese lady with chronic liver cirrhosis secondary to hepatitis B infection, likely vertically transmitted. She was diagnosed during a recent admission for what turned out to be acute cholecystitis, and her liver function test was noted to be markedly deranged. She is otherwise asymptomatic, slightly icteric, She has never had complications of liver cirrhosis, and US HBS showed no lesions in the liver. She is currently Hbe positive with high viral load. She is still undecided as to whether to initiate antiviral treatment or allow spontaneous "e" seroconversion. Newly married, she is under great pressure to produce grandchildren by her mother-in-law. She is relieved that her husband is already vaccinated against Hep B with healthy antibody titres, and that it is possible to prevent vertical transmission to her children with active + passive vaccination perinatally. Otherwise she is able to work as per normal, no social or financial problems.

Gastro: Liver Cirrhosis

ADULTS

Does this patient have cirrhosis?

- Asymptomatic in the early stages: detected on LFT, fibroscan (e.g. monitoring in patients with known hepatitis)
- In later stages, our beloved stigmata of chronic liver disease: jaundice, clubbing, palmar erythema, scleral icterus, gynaecomastia, spider naevi, loss of axillary hair, testicular atrophy; with elevated LFTs.
- Some patients also receive imaging or liver biopsy.

What is the etiology of this patient's cirrhosis?

- Knowing the etiology is important because this can change management.
- Some etiologies may be obvious on history, examination, and confirmed on initial investigation
 - Alcohol: history of alcoholism → look at MCV, GGT.
 - Hepatitis: known status, immunization, family hx, high risk sexual behaviour, IV drug use, received blood products in another country → do HBsAg, HBsAb, HBcAg, HCV IgM
 - Congenital: e.g. biliary atresia.
- Other etiologies are less obvious although there may be clinical suspicion
 - Wilson's disease: neurologic symptoms, family hx, kayser-fleischer rings → measure serum copper, ceruloplasmin (low), urinary copper (high), may need biopsy.
 - Hemochromatosis: known transfusion-dependent hemolytic anaemia → do ferritin, TIBC,
 - Cardiac cirrhosis
 - Budd-Chiari
- Yet other etiologies are only investigated when none of the obvious ones fit -- often a biopsy is needed, but does a biopsy change management?
 - Non-alcoholic steatohepatitis (may be responsible for a lot of 'cryptogenic' cirrhosis)
 - Autoimmune hepatitis → IgG, ANA, liver biopsy.
 - Glycogen storage diseases
- Finally, in some patients, no obvious etiology is found and the cirrhosis is labelled 'cryptogenic'

What is the severity of this patient’s cirrhosis?

- Use Child’s-Pugh Scoring: Child’s A = 5-6, Child’s B = 7-9, Child’s C = 10-15

	1 point	2 points	3 points
Albumin g/L	>35	28-35	<28
Bilirubin μ mol/L	<34	34-51	>51
Coag: PT prolongation (INR)	<4 (<1.7)	4-6 (1.7-2.3)	>6 (>2.3)
Distension (ascites);	None	Controlled on diuretics	Refractory to diuretics
Encephalopathy	None	West Haven 1-2 1: Change in behaviour alone 2: Disorientation, drowsy, asterixes	West Haven 3-4 3: Marked confusion, mostly sleeping but arousable 4: Comatose

What complications has this patient had → how can I optimise management?

- Consider complications in terms of portal hypertension (variceal bleed, ascites), loss of catabolic function (encephalopathy, pruritus, jaundice), loss of synthetic function (coagulopathy, hypoglycaemia). Hepatocellular CA discussed below.
- Variceal bleeds
 - Any previous hematemesis, melena?
 - How severe, did the patient need TIPS, required ICU?
 - Emergent Mx: ABC, Abx, IV PPI, somatostatin, transfusion, endoscopic hemostasis.
 - Follow up: prophylaxis with propranolol, treat portal hypertension, interval variceal band ligation to eliminate all remaining varices
 - Treat portal hypertension: nonselective beta blockers (propranolol), transjugular intrahepatic portosystemic shunt insertion (TIPS) if recurrent.
- Ascites:
 - Symptomatic with abdominal distension, SOB on lying supine (diaphragmatic splinting), hernias? → Ddx cardiac, renal
 - If abdo pain → consider spontaneous bacterial peritonitis and treat
 - Low salt diet and fluid restriction
 - Pharmacological: furosemide and spironolactone in 5:2 ratio
 - Abdominal tap with IV albumin cover
- Hepatic encephalopathy: drowsiness/confusion, sleep wake reversal, asterixis
 - Low protein diet and nutritional supplementation
 - Lots of lactulose to ensure BO at least 2 times a day
- Others: pruritus, coagulopathy, hypoglycaemia (usually occurs late)

How often does this patient decompensate, why?

- Trace how many admissions - how often, what reason
 - How severe? > E.g. the symptomatic ascites requiring tap, vs the patient with variceal bleed and hepatorenal syndrome requiring ICU care!
 - What was response to treatment?
- Identify trigger of decompensation and treat
 - Non-compliance to low salt diet or fluid restriction
 - Acute liver injury e.g. alcohol, hepatotoxic drugs, acute hepatitis infection
 - Acute intercurrent illness e.g. URTI, GE
 - Newly developed hepatocellular carcinoma (refer to GS long case section on HCC)

Can I treat the underlying cause?

- Stop all further insults to the liver
 - No more alcohol → refer NAMS
 - Avoid hepatotoxic drugs, no TCM.
 - Vaccinate against hepatitis A and B
- Treat hepatitis B/C - indication to start treatment (see document on hepatitis)
- Non-alcoholic steatotic hepatitis → supportive mx, lose weight, treat other cardiac risk factors
- Autoimmune hepatitis → induction therapy with glucocorticoids alone, KIV tail after remission for 18 months or maintenance azathioprine
- Is the patient a transplant candidate; any donor available?

Is there hepatocellular carcinoma?

- Regular monitoring with ultrasound liver, alphafetoprotein needed
- Both hepatitis B and any cause of hepatocellular inflammation increases risk of HCC

How is the patient coping?

- Personal: Cirrhotics come in different sorts - the 'bo chup' still-drinking alcoholic, vs the young autoimmune or hemochromatosis patient. Explore the patient's disease understanding, motivation, and compliance (especially salt and water restriction)
- Functional: is the patient able to work, is he ADL independent?
- Social: often flavourful and worth some time delving into. Consider home setup, 'drinking buddies', sexual history (if hepatitis B/C - need to counsel)
- Financial: almost all will say they have financial issues.
- May need advanced care planning if end stage cirrhotic.

Sample Summary: *see worked example of 'How to summarise Issues' (Medical Long Cases > strategy)*

Gastro: Inflammatory Bowel Dx

ADULTS & PAEDIATRICS

How did this patient present & how was the diagnosis made?

- Presentation is usually inflammatory diarrhoea (bloody +/- systemic symptoms), abdo colic +/- weight loss or growth failure, +/- perianal skin tags (Crohns)
 - Exclude ddx: infections (travel history, stool OCP, C diff toxin), ischaemic (any AF?), neoplastic (scope), steatorrhoea.
- Initial workup: high TW, CRP, ESR; stool calprotectin +ve; ASCA (Crohn), pANCA (UC) not for routine diagnosis
- Colonoscopy and biopsy would have been done, this distinguished UC vs Crohn's.
 - Distribution: UC always starts from the rectum, proximal extension is continuous and may continue until the terminal ileum i.e. backwash ileitis (extent of proximal extension is prognostically important). Crohn's spares the rectum, has skip lesions, and can involve the entire GIT.
 - Appearance: Crohn's has a cobblestone appearance with deep ulcers and fissures; UC has shallow ulcers, pseudo-polyps and mucosal bridges.
 - Biopsy: UC shows shallow ulcers and crypt abscesses. Crohn's shows transmural inflammation with non-caseating granulomas.
- Small bowel workup may also be necessary for Crohns: e.g. CT or MR enterography.

Assess disease course and current issues / management tasks in terms of:

(1) Colonic flares

- History: Have there been any severe flares or emergencies?
 - UC: SIRS, anaemia <10.5, rapid weight loss → toxic megacolon (SIRS, bowel >5.5cm, pneumatosis intestinalis) → perforation
 - Crohns: IO → closed loop → perforation
 - Crohns: intra-abdominal abscess → peritonitis
- Patient in remission: The natural history is that of intermittent exacerbations.
 - Assess the tempo of flares and what maintenance drugs are required to sustain remission: none, 5-ASA alone, steroids (steroid-dependent), or other drugs?
 - Inquire compliance and motivation -- less of a problem with UC/CD than with many other chronic conditions because noncompliance quite easily leads to an unpleasant flare.

- Patient with flare: assess frequency and severity of flares (general guides below), induce remission then continue as per maintenance therapy.
 - Mild: tolerates orally, mild abdo pain, no SIRS
 - Moderate: prominent symptoms (fever, weight loss, abdo pain)
 - Severe / fulminant: SIRS or an emergency (toxic megacolon, perforation, severe IO) → perforation should manage according to surgical lines.
- Examine current management strategy and whether there is a need to step up or down - a sample approach is below (but evidence shifts quickly and exact choice is highly patient and consultant dependent)

	Ulcerative Colitis	Crohn's disease
Mild flare	[Paeds] Exclusive enteral nutrition Rectal 5-aminosalicylate (5-ASA) (sulfasalazine, mesalazine) Rectal steroid (budesonide)	[Paeds] Exclusive enteral nutrition PO 5-ASA - efficacy debated (not rectal, Crohns involves entire GI) PO Abx
Moderate flare	PO 5-ASA PO steroids (budesonide preferred due to high first-pass metabolism in liver, lower systemic effects) Steroid dependant or refractory: 6-MP or azathioprine.	PO steroids (budesonide preferred due to high first-pass metabolism in liver, lower systemic effects) Steroid dependant or refractory: 6-MP or azathioprine.
Severe flare	PO 5-ASA IV hydrocortisone / methylpred IV Abx No response: Cyclosporine or infliximab Fulminant or toxic megacolon: Panproctocolectomy & end ileostomy	IV hydrocortisone / methylpred IV Abx esp if suspecting abscess No response: Cyclosporine or infliximab
Regular maintenance	Stop smoking (reduces relapse) 5-ASA: sulfasalazine > mesalazine	Stop smoking (reduces relapse) 5-ASA: sulfasalazine, mesalazine +/- Abx
Difficult maintenance Relapse often Steroid dependant	Try azathioprine or 6MP Try biologics e.g. infliximab, adalimumab Medically refractory disease: proctocolectomy + ileal pouch anal anastomosis	Try azathioprine or 6MP Try biologics e.g. infliximab, adalimumab Minimise surgery: repeated SB resection can lead to short gut

(2) Chronic colonic complications

- Colorectal cancer
 - Patient should receive screening with biopsies
 - Recommendations for screening differ - e.g. American gastroenterological society recommendations for both UC and Crohns are to start 8 years (pancolitis) or 15 years (left colitis) after diagnosis, repeat every 1-2 years.
- Strictures causing IO
 - Rule out malignant strictures
 - Attempt to resolve with medical Rx and bowel rest, if not, surgical resection may be required
- [Crohn] Fistulation - enteroenteric (mass), enterovesical (pneumaturia, UTIs), enterovaginal (discharge), enterocutaneous (discharge), sinus tracts (intra-abdominal abscess, perianal disease (perianal abscess, fistula-in-ano)
 - Medical: immunosuppression esp infliximab, azathioprine, 6-MP
 - Surgical resection if medical Rx fails (attempt to minimise resected bowel)
- Malabsorption, malnutrition, vitamin ADEK deficiency.

(3) Extraintestinal manifestations & systemic complications

- Uveitis
- Joints: IBD associated arthritis
 - Use NSAIDs with caution
 - 5-ASA drugs, azathioprine, 6-MP, MTX, steroids, and biologics are helpful for both bowel and peripheral joints
 - Spine disease: manage like AS with physiotherapy +/- biologics.
- Skin: erythema nodosum, pyoderma gangrenosum.
 - Responds to immunosuppression.
- Liver: Primary sclerosing cholangitis → jaundice, RHC pain, raised ALP
 - Give ursodeoxycholic acid
 - Monitor for cancer
- Anaemia: due to BGIT and B12 deficiency
 - Periodically monitor Hb.
- [Crohn's] Oral ulcerations → topical steroids

(4) Complications of treatment

- Steroid side effects → see Cushing's approach
 - Osteoporosis monitoring (DEXA) and prophylaxis (replace calcium / vitamin D), KIV treatment
- Immunosuppression & myelosuppression
 - Check Hep B/C/HIV, TB status before starting potent immunosuppression
 - Vaccinations
 - Patient education - what to do if fever? (Come A&E and get FBC)
- Other side effects: liver toxicity, renal toxicity (ASA)

How is this affecting the patient?

- [Paeds] Growth & Development
- Able to work / school?
- Financial
- Social: esp if fistulating disease with enterocutaneous fistula, resection required, troublesome symptoms -- has social interaction been impaired?
- Psychological care: how does the patient feel about the illness?

Sample summary:

Lao Sai is a 12 year old boy with ulcerative colitis, who first presented at age 10 with bloody diarrhea of 2 months duration associated with low grade fever, fatigue, colicky abdominal pain and loss of weight, diagnosis confirmed on colonoscopy. He has been steroid-dependant since the time of diagnosis, developing moderately severe flares each of the 3 times his physician tails his steroids down. He is still currently in remission, and has just been started on infliximab in addition to azathioprine and steroids. Otherwise he also has joint pain in the wrists and ankles as well as skin lesions, both of which have improved with his medications. He has been in and out of school, and struggling to keep up with his studies. He is also self-conscious that he is smaller sized than all his peers (in spite of all his vitamin supplementation and efforts to tolerate exclusive enteral nutrition) and Cushingoid from the steroids. The family is barely keeping with the financial burden of his admissions and infliximab combined, but want to give him the best shot at regaining a normal quality of life. They are considering the option of an elective panproctocolectomy with ileal pouch anal anastomosis.

Please also refer to Surgical Long Case > Crohn's disease

Genetic: The Syndromic Child

PAEDIATRICS

A short short time ago in a galaxy next door ... a professor once said during an ill-fated MBBS long case, "This child has Goldenhar syndrome (??), please take a history (!!!)". Most students foamed. Are we expected to know all these rare syndromes? → You are probably expected to know something about the common ones (especially Down's), but for the rare ones, probably not. Regardless, you should have an overall approach to the syndromic child -- whatever the specific syndrome there are commonalities; the underlying cause cannot be treated so your task is to identify, in a systematic fashion, organ-based manifestations and how they have been managed, and what the current issues are.

How did this syndrome present and how was it diagnosed?

- Antenatal diagnosis may have been done: e.g. thickened nuchal fold (Down's), maternal serum screening (Down's), amniocentesis and karyotyping (Down's)
- The condition may be picked up in the neonatal period, as an abnormal neonatal examination or due to particular complications.
- It may have been picked up upon noting developmental delay.
- If features are mild, it may only be noted in adolescence or adulthood > e.g. short stature, delayed puberty (Turner's syndrome).
- The need to consider differentials depends on how obvious the presentation is, the more vague the presentation (e.g. microcephaly, vague dysmorphisms), the wider the net that should be cast – e.g. other syndromes, TORCH infection

Are there any visible features I can observe?

- The most important is to recognize that the child is 'dysmorphic'. If you recognize a specific syndrome and are confident, call it. If not, simply list the dysmorphic features.

Syndrome	Visible features	Diagnostic criteria
Down's	Eyes: upslanting palpebral fissures, prominent epicanthic folds, hypertelorism, flat nasal bridge. Ears: low-set Neck: short, excessive skin Hands: single palmar crease, incurved 5th finger Legs: widened sandal gap, hypotonia	Karyotype > trisomy 21

Syndrome	Visible features	Diagnostic criteria
Turner's	Short stature (short and fat) Neck: webbed Body: widely-spaced nipples, cubitus valgus	Karyotype > XO
Klinefelter	Tall stature Gonads: Small testes, gynaecomastia	Karyotype > XXY
Neurofibromatosis type 1	Skin: Cafe-au-lait, neurofibromas, axillary freckles	Two of the following: ≥6 cafe au lait >5mm (pre-) or >15mm (postpubertal) Neurofibromas, ≥2 or 1 plexiform Axillary / inguinal freckling Optic glioma, ≥2 Lisch nodule (iris hamartoma) Sphenoid dysplasia 1st degree relative with NF.
Sturge-Weber	Face: Port-wine stain	MRI brain.
Tuberous Sclerosis	Face: adenoma sebaceum (appears in adolescence) Skin: ash-leaf spot, shagreen patch,	Clinical diagnostic criteria, or Gene testing.
Prader-Willi	Neonate: hypotonia, feeding difficulty, failure to thrive Older: almond shaped eyes, obesity, hyperphagia, hypogonadism	Clinical criteria, or Molecular testing (complex); need to identify which is maternal or paternal chromosome.
Noonan	Face: hypertelorism, low-set ears Neck: short, webbed Short stature Chest: pectus excavatum	
William's	Elfin facies: periorbital fullness, hypertelorism, long philtrum, thick lips, wide mouth, small jaw	Karyotype > 7q deletion

Why is this child syndromic?

- Knowledge of the genetics of each condition is helpful but even if such knowledge is not available, following the broad principles of paediatric history taking will uncover much information.
- Broadly, take a deep interest in the family history. Ask if parents or relatives are affected. Also ask if the parents related to each other (consanguinity)
- When considering non-heritable conditions or de-novo mutations, delve into antenatal history and consider maternal risk factors e.g. advanced maternal age.

What are the complications and current (medical) issues?

- Approach: If you know the specific syndrome, look for its particular manifestation (see table); alternatively the patient/parent may volunteer the information.
- Regardless, some concerns are common to all syndromes (probably all children) so you should spend time looking into these.
- How is the child growing? → Plot height and weight on the percentile charts
 - Any feeding and nutrition issues? (Especially if unable to feed self)
- How is the child’s development? → Do a developmental assessment
 - Neurodevelopment, intellectual disability → If present, how has the family been coping? Special school, early intervention? Care needs? (see below)
 - Motor development → Any motor disability
 - Speech and language → Hearing aids? Early intervention?
 - Sensory impairment: blindness, deafness → Any special school or other intervention?
- Any seizures? (Neurocutaneous syndromes) → If so, what is the mx, any breakthroughs? (see approach to seizure)

Syndrome	Organ-based complication	Directed management
Down’s (Trisomy 21)	CNS: developmental milestones, behaviour CVS: VSD (most common), AVSD (most pathognomonic) GI: duodenal atresia, anorectal malformations Eye: nystagmus, strabismus, Hearing impairment Endocrine: hypothyroidism, DM Leukemia Immunodeficiency	Gauge severity ?surgery ? Surgical correction Hearing aids Replacement Monitoring
Turner’s	Endocrine: short stature, hypothyroid CVM: aortic valve disease, aortic dissection Renal: malformations → UTI, hypertension. Metabolic syndrome Gynae: amenorrhoea, infertility, osteoporosis Increased risk of autoimmune disease.	Screen TFT, KIV GH 2DE KIV MRI Do Cr, US, treat UTI, Monitor glucose, lipids Exogenous estrogen.

Syndrome	Organ-based complication	Directed management
Klinefelter	Endocrine: tall stature, infertility, hypogonadism Psych: usually normal IQ but may have behaviour problems.	
Neurofibromatosis	Malignant peripheral nerve sheath tumors Hypertension: coarctation of aorta, renal artery stenosis, pheochromocytoma. CNS: intellectual disability, seizures. Eye: optic glioma MSK: skeletal deformities (bowed legs, pseudoarthrosis, bone cysts)	Monitor neurofibromas. Measure BP KIV renal artery US. MRI brain if seizure- ?tumor Eye screen Examine MSK.
Sturge-Weber	CNS: Seizure, focal neuro deficits, low IQ Eye: glaucoma, choroidal haemangioma, visual field defect Endocrine: growth hormone deficiency	Refer eye, screen glaucoma Inx if not growing.
Tuberous Sclerosis	CNS: Brain tubers, seizures, behaviour problems CVS: rhabdomyoma Renal: angiomyolipoma, HTN, cancer	Treat seizures, MRI brain Usually asymptomatic, 2DE Measure BP, surveillance with MRI
Prader-Willi	CNS: low IQ, behaviour problems Hyperphagia, stealing food --> choking episodes Endocrine: short stature, hypogonadism Complications of obesity: DM, hyperlipid, OSA, IHD	Limitation of food Growth hormone. Monitor and manage accordingly
Noonan	CNS: low IQ CVM: PS, ASD, HOCM Bleeding diathesis	2DE
William's	CNS: moderate intellectual disability but good language, ADHD Hearing: loss CVS: AS, PS Renal: renal artery stenosis GIT: constipation	Audiology evaluation Renal artery ultrasound

How is this affecting the child & family?

- Special care arrangements
- Special school: MINDS, rainbow centre, special school
- Social: stigma, integration with society, what the relatives think
- Psychological: caregiver stress, lost hope (if know prognosis), guilt,
- Financial cost

Genetic implications

- Stance on future childbearing? → Genetic counselling?
- Generally it is necessary to first test parents for the mutation. NF and TS are AD, so if parents are unaffected, consider new mutation, mosaicism, or incomplete penetrance (if TS).
- Genetic counselling depends on the inheritance and penetrance and can be complex. For example,
 - NF: AD with high penetrance, low-risk if parents are not affected.
 - TS: AD with variable expression, need to test if parents are affected.
 - Sturge Weber: it is not heritable.
 - Down's syndrome: most are new mutations due to non-disjunction (risk of recurrence is 1 in 200 for <35 yr old mother and equal to age-specific risk in >35 yr old mother), however parents may also be balanced translocation carriers (risk of recurrence depends on what translocation)

Haem: Hemophilia

PAEDIATRICS (less likely adult)

How was the diagnosis made?

- At what age did this boy present, and how? E.g. known FHx, haemarthrosis, bruising, hematuria
 - Ddx for haemarthrosis: septic arthritis.
 - Often mistaken as non-accidental injury
- Laboratory confirmation is prolonged aPTT, correctable with mixing, reduced factor VIII (Hemophilia A) or IX (Hemophilia B)
- What is the factor activity level? > Defines severity
- There is usually a significant family history (X linked recessive inheritance): only boys are affected, apparently affected females either have lyonization or something else e.g. von willibrand disease.

Have there been any clinically serious bleeding & what is the sequelae?

- Intracranial bleeds
 - Any residual neurological impairment?
- Hemarthrosis
 - Any hemophilic arthropathy, what is current motor function
 - Any orthopaedic interventions necessary?

What is the treatment to date & any complications?

- How many active bleeds or injuries in the last year?
 - Mild bleed: Give desmopressin i.e. DDAVP (only for hemophilia A - releases factor VIII) and tranexamic acid (fibrinolytic)
 - Serious bleed: factor replacement to at least 40-50% of normal. All head injuries (including minor knocks) must be taken seriously.
- Has the patient needed surgery or faced any other hemostatic challenge, how were these dealt with?
- Is patient on baseline prophylaxis with factor replacement? -- if not on, why?
 - Primary prophylaxis indicated for severe hemophilia: reduces risk of arthropathy compared to on-demand treatment
 - Secondary prophylaxis if there is already hemophilic arthropathy
 - Options: Purified plasma concentrate or recombinant
- Complications:
 - Have factor inhibitors developed? -- Give bypass agent (very expensive!)
 - Any transmission of blood borne infectious disease? (Common in past, rare now)

How is the patient's life affected?

- What is the patient bothered by: any troublesome bruising, serious bleeding? Is the boy able to brush teeth?
- How is the patient's knowledge of disease: Know how to come in for on-demand factor replacement, know what to do if "minor" head injury?
- Functional limitation: is the boy able to exercise? Does he disregard restrictions in a bid to 'have fun' with friends?
- Social limitation: does inability to participate in some activity lead to social ostracization?
- Financial limitation: factor replacement is costly
- [Paeds] How is growth and development otherwise?

Genetic counselling

- Are other family members involved?
- Do parents understand implication for future pregnancies: X-linked recessive, therefore half of all male children are affected (i.e. 25% of all children), and half of all female children are carriers.
- Are patient's sisters aware that they *may* be carriers?
 - Ethical issue: should parent test the child before child is 21? What if the child does not want to know?
 - Implications for future relationships

Sample summary:

Humpty Dumpty is a 17 year old boy with hemophilia A, diagnosed at the age of 1.5 years old when he presented with painless right knee swelling with no history of trauma/fall. Since then he has never had any major bleeding episodes especially intracranially or from the GIT that required admission for desmopressin, and underwent an uneventful appendectomy last year with FFP supplementation. He is not on primary prophylaxis. In the past year however, he has been working out hard at the gym (against doctor's advice) to impress a girl he likes at school. He notices mild bilateral elbow swelling after every workout involving free weights and bench presses, but dismissed it as insignificant as they are painless and self-resolve. More recently though he has noticed stiffness and reduced range of movement in both elbows. Otherwise he is well-accepted in school and is thankful for the understanding he has from his friends when he sits out of PE class. Family is supportive and financially able to afford factor replacement treatment should he ever need it. His younger sisters (15, 16) are aware they that may be carriers of the gene, but only want to decide on testing when they turn 21.

Haem: ITP

ADULTS & PAEDIATRICS

What type of bleeding is this?

- Classic ITP presents with a thrombocytopenic bleeding pattern: non-palpable purpura (not blanchable) on bilateral lower limbs, with mucocutaneous bleeding (epistaxis, gum bleeding, ecchymosis on lips, menorrhagia).
- Distinguish from the vasculitic rash which is a palpable purpura.
- In contrast the hemophilias and other coagulopathies present as haemarthrosis, intramuscular hematomas hours after minor trauma

Is the diagnosis just ITP?

(a) Is the patient sick

- ITP patients are completely well. A sick patient does not have ITP.
- Consider: DIVC, meningitis, vasculitis, TTP-HUS, liver failure.

(b) Is there isolated thrombocytopenia, no other cell lines involved?

- The expected picture is isolated thrombocytopenia with normal RBC, normal WBC, normal coagulation profiles.
 - Patient should not have symptomatic anaemia, pallor, or recurrent infections.
 - Ask if patient is on warfarin or any NOACs.
- If other cell lines are involved → consider
 - All low: aplastic anaemia, megaloblastic anaemia, marrow infiltration, other causes of myelosuppression (e.g. drugs)
 - Low Hb, low platelets → B12 deficiency,
 - Look for the raised TW of leukaemia.
- If the clinical picture is that of ITP but Plt is 'normal' → likely von willibrand disease, do VWF antigen, VWF activity (ristocetin cofactor activity)

(c) Is there a secondary cause of thrombocytopenia?

- *Infection*: HIV, HCV can present as thrombocytopenia -- risk profile and order workup. Rule out dengue in acute setting. Sometimes no infection is identified but "ITP" remits spontaneously very soon > many of these are viral
- *Rheumato*: ask for symptoms of SLE, vasculitis -- if suspicious workup ANA, dsDNA.
- *Exclude drug induced*: heparin, antibiotics, antiepileptics -- take full drug hx, stop drugs, also inquire supplements and traditional meds.
- Liver disease -- ensure not jaundiced, LFTs normal.

ITP is a diagnosis of exclusion; exclusion of secondary causes is required for diagnosis.

Complications: Is there any clinically significant bleeding?

- Dangerous bleeds: Intracranial hemorrhage, PR bleed
- Past hemostatic challenges: surgeries, pregnancy & delivery.
- Patient education
 - Do patients know what to do if they get a bleed?
 - Are there dangerous occupational and leisure activities?

How has it been treated?

- *Goal of ITP treatment: to prevent clinically important bleeding rather than to normalize the platelet count. Treatment is not always required.*
- Acute admission for low Plt -- supportive mx:
 - Complete rest in bed, no IM injections, no brushing teeth
 - Daily platelet counts
 - FFP, platelet transfusions for patients with clinically imp't bleeding or Plt <20
 - IV fluid resuscitation
- 1st line therapy: glucocorticoids -- in the chronic setting the question is often titration to achieve an acceptable Plt count with no bleeding, and minimise side effects -
 - Cushings syndrome
 - Any immunosuppression -- vaccinate, counsel pt on what to do.
 - Osteoporosis: monitor DEXA, give Ca/VitD, treat if necessary
- 2nd line: IVIG -- raises platelet count more rapidly than glucocorticoids. Use for active bleeding, pts who need an urgent invasive procedure.
- Other therapies: splenectomy, rituximab, thrombopoietin receptor agonist, immunosuppressive therapy

What is the prognosis?

- Adults: ITP is generally a chronic disease with only 10% remitting spontaneously. The majority have stable disease with normal life expectancy.
- Children: up to 50% remit spontaneously, especially those <10 years old.

How is the patient's function, overall health, and quality of life?

- Child: Growth and development
- Is his/her function/participation in activities limited by the disease
- Social setup
- Financial

Sample case: Lolita is a 29 year old Filipino maid who presents with non-palpable purpura, mucocutaneous bleeding, and menorrhagia. She was otherwise very well and has no suggestion of autoimmune disease, chronic viral infection, liver disease, and is not on any long-term medications or supplements. She was diagnosed with ITP and required FFP. Her platelet counts have come up once she was started on prednisolone.

Haem: Thalassaemia

PAEDIATRICS (less likely adult)

How did thalassaemia present > what clinical phenotype?

- Thal major: well at birth, presents at 6-12 months (when HbF production switched to HbA) with pallor, irritability, growth retardation, hepatosplenomegaly, and jaundice. Thereafter they require 3-4 weekly transfusions. Genetically they are $\beta^0\beta^0$
- Thal intermedia: presents in late first decade often as lethargy or difficulty exercising. Unlike thal major, these kids do not require hypertransfusion, only require intermittent or no transfusion. The underlying genetic abnormality is heterogeneous e.g. abnormal beta globin chains which produce some but not zero beta-chains, HbE-beta thal, or HbH disease ($\alpha^{-/-}$).
- N.B. Thalassaemia minor is asymptomatic ($\alpha^{-/\alpha^{-}}$, $\alpha\alpha^{-/-}$ or $\alpha\alpha/\alpha^{-}$), Bart's hydrops ($^{-/-}$ with no alpha allele) dies in utero

How was the diagnosis confirmed?

- Blood: hypochromic microcytic anaemia with low RDW, evidence of hemolysis (unconjugated hyperbilirubinemia, raised LDH, decreased haptoglobin)
- Gel electrophoresis distinguishes the type of thalassaemia: HbH (β_4) in α -thalassaemia, HbF ($\alpha_2\gamma_2$) and HbA2 ($\alpha_2\delta_2$) in β -thalassaemia, HbE, and HbS.

How is the child's anaemia managed?

- Transfusion requirements: how often and how much?
- Is there residual anaemia?
 - What is the usual Hb?
 - Any symptoms: Pallor, lightheadedness, lethargy, poor exercise tolerance
- Is there residual extramedullary hematopoiesis? > look for chipmunk facies, hepatosplenomegaly.
- Are transfusion requirements increasing? (increasing frequency and volume) > is there hypersplenism?
 - Usual indication for splenectomy is hypersplenism causing accelerated RBC destruction and increasing transfusion requirements
 - If splenectomised, any infective complications, are the child's pneumococcal and meningococcal and HiB vaccinations up to date?
- Any complications from transfusion? > E.g. blood borne virus, antibody production making it hard to find suitable blood.

Is the child receiving adequate chelation?

- In the past thalassaemia kids used to die in their teenage years due to iron overload.
- What chelation is the child receiving?
 - SQ/IV slow infusion deferoxamine 8-24 hours every day
 - PO deferasirox (OD) or deferiprone (TDS) > more convenient but expensive.
- How is the compliance? > If poor, why, what are the issues? (Medical / financial / social / psychological)
 - Typically no problems while the child is young and parents administer the treatment. Compliance becomes a problem when the child becomes a teenager and the chelation regime clashes with hectic schedules packed with extra-curricular activities or late-night suppers with friends
 - PO deferasirox may be a more palatable option, but it is expensive so do also explore the family's financial situation when you offer that alternative

Is there iron overload?

- This is especially so for transfusion dependent thalassaemia but non transfusion dependence does not exclude iron overload because extramedullary hematopoiesis itself stimulates increased iron absorption from the gut.
- What are the latest monitoring parameters?
 - Target ferritin: 300-500 mcg/L (normal 200-300)
 - MRI T2* (cardiac, liver)
- Any end-organ damage?
 - Heart: any arrhythmias or heart failure?
 - Liver: any chronic liver disease?
 - Pancreas: any diabetes? Latest OGTT
 - Gonads: any delayed puberty? Latest serum FSH/LH, estrogen, testosterone
 - Thyroid: any symptoms of hypothyroidism? Last TFT
 - Pituitary: short stature/shorter than peers? Latest height and weight percentile

How is the overall coping of the child?

- Growth and development
- Is he/she able to participate fully in school?
- Social setup
- Financial

Genetic aspects: how about the siblings / family / future children?

- There is often a family history +/- other involved siblings
- Do parents want more children? > Have they gone for genetic counselling?
 - Beta-thal is autosomal recessive i.e. if parents are both $\beta^{++}\beta^0$, the risks are: beta thal major 25%, carrier 50%, normal 25%; genetic counselling is more tricky in alpha thalassaemia.
 - First need to do genetic testing to confirm they are carrying the alleles (most parents would have received Hb as baseline screen, then offered electrophoresis if Hb low)

- Can consider antenatal testing via chorionic villus sampling (10 to 12 weeks of gestation), or amniocentesis (>15 weeks of gestation); risk miscarriage. But consider parental values -- will they abort? If no, don't bother testing.

Sample summary: Alvin is a 15 year old Chinese boy who presented at 5 years old with lethargy and shortness of breath and was diagnosed with beta thalassaemia intermedia. He receives transfusions about q2-3 monthly and knows how to come in for transfusion should he feel unusually lethargic. His mum had all along ensured compliance to subcutaneous chelation therapy and I note that he has hit puberty last year, with no cardiac or liver complications. This year, however, his parents divorced with custody going to the dad - while he is a bright kid who understand his disease and is motivated to comply, he appears somewhat affected by the divorce and I worry whether he would keep up with regular chelation without his mum's watchful eye. I don't think they can afford oral chelation. Hence my issues are (1) parental divorce, and (2) thalassaemia intermedia requiring 2-3 monthly transfusion and no iron overload thus far.



ADDENUM: Hereditary Spherocytosis

Definition: HS is a hemolytic anemia due to a red cell membrane defect. It is a result of heterogeneous alterations in one of six genes (most often the ankyrin gene) that encode for proteins involved in vertical associations that tie the membrane skeleton to the lipid bilayer. Autosomal dominance in 75% patients, recessive inheritance for the rest.

When to suspect hereditary spherocytosis as a differential?

- Presents very similarly to thalassaemia → with anaemia, jaundice, splenomegaly, +/- positive family history of hemolytic anaemia. Blood work will also show anaemia with low MCV, high reticulocytes, and evidence of hemolysis (unconjugated bilirubinemia, high LDH, low haptoglobin)
- However unlike thalassaemia, there is
 - More hemolysis and less ineffective erythropoiesis, less extramedullary hematopoiesis → spleen is usually larger and liver smaller than thalassaemia
 - High RDW
 - Spherocytes on peripheral blood film with negative coomb's test (Ddx: alloimmune and autoimmune hemolytic anemia can also have spherocytes, but both are associated with a positive Coombs test)

How to confirm diagnosis?

- Classic: osmotic fragility test
- More accurate: eosin-5-maleimide binding test +/- a second test e.g. acidified glycerol lysis time or the cryohemolysis test.

What do I need to do for this patient?

- Asymptomatic or mild: nothing
- Symptomatic anaemia:
 - Blood transfusions
 - Consider splenectomy: recommended for symptomatic HS and moderate to severe hemolysis and anemia (reduces hemolysis, reduces gallstone formation)
- If splenectomized, risk of infection with encapsulated bacteria
 - Vaccinate: pneumococcal and meningococcal and HiB
 - If under 6 yrs old, consider partial splenectomy (temporary relief of hemolysis while still providing freedom from sepsis)
- Symptomatic gallstone disease: cholecystectomy

ID: Tuberculosis

ADULTS (less likely paediatrics)

contributions from David Ng

How did this patient present, is it TB?

- *TB can cause anything except pregnancy* → there is a million ways TB can present, and quite insidiously too, so always think of it as a ddx
- Pulmonary TB: cough +/- fever, loss of weight, pleural effusion
 - Always consider ddx e.g. CA lung
 - Start with CXR: classically infiltrates with upper lobe cavitation; other appearances possible e.g. collapse, consolidation, pleural effusion, cavitations, fibrosis, bronchiectatic changes, reticulonodular opacities (miliary TB)
 - Obtain at least 3 sputum samples (either coughed or induced with hypertonic saline nebuliser, at least one specimen obtained in early morning) for AFB smear, cultures, TB nuclear acid amplification test (NAAT) i.e. PCR (also has advantage of testing TB strain for drug resistance)*; if unable to obtain sputum consider bronchoalveolar lavage (also has added advantage of biopsy if there is a lesion favourably located)
 - If pleural effusion then tap; some institutions favour sending for ADA in addition to AFB and culture
 - Presumptive clinical diagnosis is sufficient to initiate therapy.
- Extrapulmonary TB e.g. lymphadenopathy +/- fever, loss of weight
 - Intracranial: CT/MRI. lumbar puncture
 - Lymph node: biopsy and AFB stain/culture
- Interferon gamma release assays (T-spot, quantiferon) are used to diagnose latent TB infection and should not be used to diagnose active TB (cannot distinguish latent vs active TB, in acute infection there is temporary anergy and negative T-spot)

**Interpretation of AFB / NAAT results:*

	NAAT +ve	NAAT -ve
AFB +ve	Diagnostic. treat with RHEZ	Check if inhibitors to NAAT are present. Repeat AFB. If 2nd AFB also +, consider non-tuberculous mycobacteria.
AFB -ve	Repeat NAAT (if 2x +ve, can be diagnostic) Start treatment if clinically suspecting TB	Cannot rule out TB Clinical judgment whether to treat.

Why does this patient have TB?

- Most often it is an elderly patient with reactivation disease
- But look for any immunocompromised state
 - HIV - in TB always look for HIV and in HIV always look for TB
 - Is patient on any immunosuppressive drugs (e.g. transplant, autoimmune conditions)
- Work out any contact history, foreign country of origin.

Is this patient receiving treatment?

- Drugs available:
 - 1st line drugs are R (rifampicin), H (isoniazid), E (ethambutol), Z (pyrazinamide)
- R and H are bacteriocidal
 - 2nd line include streptomycin, levofloxacin
 - Give H with pyridoxine.
- What is the patient on: for non-HIV patient a possible regimes is
 - Lung TB: 2 weeks of daily RHEZ, then twice weekly for 6 weeks, then RH twice weekly till 6 months
 - Extrapulmonary disease may require longer duration of treatment
 - Patient considered contagious until 2 weeks of treatment have been completed or sputum is negative → isolate until then
- How is the treatment response
 - Compliance to treatment? → Consider Direct Observed Therapy? Taking less than recommended is a recipe for MDR and treatment failure
 - Follow up sputum smears: repeat 2 months after starting therapy and again if still positive. Positive cultures after three months of effective treatment must be evaluated carefully to identify the cause of the delayed conversion. Positive cultures after 4 months define treatment failure
 - If treatment failure, don't just add single drug (will breed resistance); retreat using at least 3 new 2nd line drugs.

Are there any complications of treatment?

- Rifampicin: hepatitis/cholestasis, orange discolouration of urine/tears → monitor LFT, stop if bil rises
- Isoniazid: peripheral neuropathy → ensure pt is taking pyridoxine 10mg/day
- Ethambutol: optic neuritis → Monitor Ishihara chart
- Pyrazinamide: hepatitis, gout → monitor LFT
- GI side effects can be significant, may affect patient's ability to take retain meds and obtain adequate nutrition

Is there a need for contact tracing?

- Consider chemoprophylaxis for individuals with significant exposure to patient while pTB was active
 - Consider Mantoux testing (result interpretation depends on prior BCG vaccination)
 - Or do interferon gamma release assays.
- Isoniazid 300mg OD X 9/12 + pyridoxine 50mg/day

How has the patient been affected? How is the patient coping overall?

- Patient will be unable to work for a period of time (loss of income) and may be difficult to have to keep going back to polyclinic for DOT
- Function: ADL, ambulation
- Nutrition: important to be well nourished to respond to anti tuberculous therapy.
- Financial, social situation: TB treatment is free in Singapore.

Sample summary: Mdm Xiu is a 70 year old lady with non insulin dependant diabetes and diabetic nephropathy CKD3, admitted for 3 weeks of cough with fever and weight loss. Chest X ray showed upper lobe cavitation with a small right-sided pleural effusion, and sputum AFB cultures were positive. She was isolated and commenced on direct observed therapy with RHEZ. There was symptomatic improvement and weight gain, follow up chest X ray showed resolution of disease with no residual nodularity. However she lost her job due to her admission and having to attend direct observed therapy thereafter, and as a result has significant financial and emotional concerns.

ID: HIV

ADULTS

What is this patient's disease course / where is he in the natural hx of HIV?

- Antiretroviral therapy has dramatically altered the disease course of HIV, offering the potential of normal life expectancy. The majority of HIV patients are well outpatients, but some progress to end stage because of late presentation or failure to receive treatment
- When and how was this patient diagnosed with HIV?
 - Asymptomatic, incidental diagnosis: e.g. admission for unrelated illness, at NS screening, employment screening etc.
 - Primary HIV infection: an infectious mononucleosis like syndrome (ddx: EBV, CMV) with fever, lethargy, myalgia, rash, lymphadenopathy, pharyngitis, headache
 - Symptomatic HIV infection without AIDS: patients with persistent generalized lymphadenopathy, other organ-based complications (e.g. thrombocytopenia, glomerulonephritis) who were found to have HIV → Need to consider ddx.
 - Late-stage presentation with AIDS-defining illnesses e.g. pneumocystis pneumonia
- Where in the course of disease is this patient? The stage of HIV is of crucial importance because it determines the likely issues
 - What is the latest CD4? CDC definition: >500 (stage 1), 200-499 (stage 2), <200 (stage 3) during which AIDS defining illnesses appear.
 - Are there any AIDS-defining complications?

What are the issues and how to manage?

(1) Antiretroviral therapy

- Is the patient on ART?
 - ART is now indicated regardless of CD4 count: increases chance of immune reconstitution to near normal CD4 levels, reduces transmission
 - If not on, why? → Is the patient ready to start? Need compliance, commitment to lifelong Rx.
 - If initiating → beware immune reconstitution inflammatory syndrome.

- What ARTs has the patient had / what is he currently on?
 - Usually start with a backbone of 2 NRTIs (tenofovir, emtricitabine, abacavir - must test HLAB5701, lamivudine) plus an NNRTI (efavirenz, nevirapine)
 - NNRTIs have low genetic barrier to resistance → Once resistance develops, switch to a protease inhibitor which has a higher barrier (e.g. lopinavir, atazanavir, +/- boosting with ritonavir)
 - Choice often depends on comorbidities (specialist topic).
- Any issues with ART?
 - Side effects and other issues? > e.g. renal (tenofovir), liver (nevirapine)
 - Compliance is very important as drug-resistance mutations will invariably emerge if HIV replicates in the presence of antiretroviral drug concentrations insufficient to exert complete suppression → if noncompliant, explore why. Consider combination tablets for easier dosing
- What are the response to therapy
 - Any resistance to Rx?
 - Check viral load: prognostically important, goal is to reduce HIV replication below which the virus does not evolve. If rising → Check compliance, genotype resistance testing.

(2) Opportunistic infections

- What are the patient's baseline serologies? > hepatitis, toxo, CMV
- What is the patient's history of infections?
 - Any respiratory infections: PCP, MAC, TB
 - Any skin infections: HSV,
 - Any chronic diarrhoea: cryptosporidium
 - Any dysphagia: candidiasis
 - Any eye disease: e.g. CMV retinitis (need eye screen)
 - Intracranial infections: toxoplasmosis, cryptococcus, PML (JC virus)
 - Any septicaemia: any bacteria e.g. salmonella
- Prevention of infections
 - Vaccinations: an advanced discussion -- in general vaccinations are imppt due to immunocompromise, but response to vaccine may be poor, also avoid live vaccines if CD4 low. Consider vaccinating against influenza, tetanus, pneumococcus, hepatitis B.
 - Precautions if CD4 <200: beware undercooked food including soft boiled eggs (salmonella, toxoplasma), bartonella (cats)
 - Prophylaxis if CD4 <200: depending on CD4 level, prophylaxis against PCP (bactrim), toxoplasma (bactrim), MAC (azithromycin)

(3) Non-infectious disease manifestations

- HIV is itself an inflammatory syndrome and can cause other organ involvement
- Malignant: lymphoma, kaposi sarcoma
- Skin: psoriasis
- Cardiac: HIV cardiomyopathy, inflammatory state increasing risk of IHD
- Neurologic: gullian barre syndrome, peripheral neuropathy
- Haematologic: anemia, leukopenia, lymphopenia, or thrombocytopenia
- Renal: membranous glomerulonephritis
- Psychiatric: depression, HIV related neurocognitive disorders (ddx: intracranial infection)

(4) Infectivity and public health

- How did the patient acquire disease?
 - Sexual hx: no. of partners, type (homosexual vs heterosexual), casual vs committed
 - IVDU
 - Occupational
 - Overseas blood transfusion
- Is the patient currently sexually active
 - Must inform partner (by law)
 - If good adherence to ART, viral load <400 → low risk of transmission (Partner study). If formed partnership, no need condom (acceptable psychologically?). If casual sex, still need to protect against other STD
 - If viral load high and sexually active → need counselling
- Pregnancy: If female patient, any intentions?
 - Good adherence to ART and well control of viral load → pregnancy is usually safe
 - Discuss with physician and obgyn → Planned pregnancy

Are there any other comorbid conditions?

- With increasing life expectancy many patients are more likely to die of a comorbid condition than of AIDS
 - Address comorbid aggressively.
- Beware of disease-disease and drug-disease interactions
 - On ritonavir: statins contraindicated
 - On atazanavir: no PPIs (needs acidic pH for absorption)
 - Tenofovir causes renal side effects
 - Protease inhibitors cause hyperlipidemia, DM → must screen

How is this affecting the patient?

- Function and employment
- Social stigma is great → look into emotional and psychiatric issues.
- Does the family know and what do they think?
- Financial burden → if cannot afford drugs, join buyer's club to buy generics from Thailand.

Sample summary: Anonymous is a 68-year-old Chinese gentleman who was diagnosed 2 years ago with HIV when he first presented with pneumothorax secondary to pneumocystis pneumonia. CD4 count then was 100. He was started on antiretroviral therapy, to which he has been absolutely compliant, and his CD4 count has climbed up to 300. He has not had any other opportunistic infections, or any other non-infectious HIV complications. He has developed hyperlipidemia and diabetes mellitus secondary to the antiretroviral therapy, for which he is on medical treatment. Unfortunately, as he had acquired the infection from one of his many previous illicit affairs, his wife divorced him after he broke the news to her (she has thankfully tested negative). He is socially isolated, having been ostracized by family and too ashamed to tell his friends. He now stays alone in a rented room, supporting himself comfortably as he continues work as a security guard.

ID: Infective Endocarditis

ADULTS (less likely paediatrics)

contributions from David Ng

Approach to non-specific fever

The best thing to do is be systematic. A overview is repeated below, a more comprehensive approach can be found in Approaches to Symptoms of Disease > Fever

History	Physical Exam	Investigations
Fever Hx (duration, pattern, onset, frequency) Any localising symptoms? a) Infection - Cranial (AMS/Headache/BoV) - Ear (tinnitus/giddyness/LoH) - Pulmonary (Cough, SOB) - Cardiac (murmur/IE signs) - Intraabdominal (Pain/Vomit/Diarrhea) - Hepatic (Jaundice/RUQ pain) - OM/TB Spine (point tenderness) - Urine (pyuria/dysuria/flank pain) - GU (PV d/c, urethritis, low abd pain) - Ulcers (numbness/calluses/d/c) - vector-borne diseases (malaria) b) Malignancy - constitutional & B symptoms - system specific symptoms c) Connective tissue disorder - rash, ulcers, joint pains Risk profile patient: - Any risk of immunosuppression? - Travel history - Contact history (including animals) - sexual history - HIV/STIs - Drug History - Vaccination - FHx of cancer or CTD Social history and function	Go head to toe Head: - CN palsies, - Oral: ulcer, fungus Lymph Nodes (cervical, axillary, inguinal) Heart & Lungs - Murmurs - Consolidation, collapse - Effusion Abdomen: - Organomegaly / mass - Tenderness - Genitalia: sores? Bones/limbs: any point tenderness Skin: any infection, bites, ulcers	Routine: FBC, RP, LFT Cultures: - Blood: 3 sets 3 sites - Sputum Cultures CXR 2DEcho for I/E CT (TAP) Serologies - ANA - RF - Complement - ESR - CRP - IFN-γ assay Additional tests - CTD serologies - Bacterial serologies - KIV biopsies - Thick/thin film (malaria) - HIV serology

Could this be infective endocarditis?

- Determine onset as acute vs subacute vs chronic involve different organisms
- Clinical manifestations
 - Most commonly PUO, initial 1st line investigations come up empty and time to look closely for more occult sources
 - New murmur
 - Extra-cardiac manifestations
 - Complications: Heart failure, stroke
- Ddx
 - Common: sepsis (any source) complicated by AMI and/or CCF (esp if pt has cardiac disease)
 - Other occult infections: deep seated intra-abdominal/pelvic abscess, pocket site infection from permanent pacemaker or intra-cardiac device

How could this patient have gotten infective endocarditis? Is this a high risk individual?

- Previous episode of infective endocarditis | Ask for dentition,
- Congenital heart disease | recent dental op
- Prosthetic valves (entirely different ballgame altogether) | Abx prophylaxis
- History of cardiac lesion causing turbulent flow e.g. hx of rheumatic heart dx
- Known IV drug abuser
 - When was last IV use? Shared or re-used needles? Other friends also fever?
- Long-standing catheter e.g. perm cath in renal patient
 - Find out about perm cath care, hx of perm cath infections, MSSA/MRSA bacteremia

How do I confirm the diagnosis?

- Modified Duke's Criteria --- please look up
- Translation: what do I need to do on the ground?
 - Thorough clinical examination! Including fundoscopy for Roth spots and urine dipstick for microscopic hematuria
 - Take 3 blood cultures from different peripheral veins under aseptic technique over 30 minute intervals, BEFORE initiating IV Abx
 - Arrange for transthoracic echocardiogram (1st line) or transesophageal echocardiogram (if TTE negative but high index of suspicion, prosthetic valve or intra-cardiac device causing acoustic shadows)

What are the complications of IE I need to watch for/manage?

- Congestive cardiac failure
 - Secondary to sudden regurgitant lesion (chord rupture or dehision of prosthesis), valve obstruction by vegetation, or fistula creating a shunt
- Periannular extension
 - I.e. Formation of abscess, fistula, valvular dehiscence
 - Is there heart block (due to invasion involving conduction bundles)? → serial ECGs please!
 - Common in prosthetic valve IE, aortic valve most often affected
- Systemic embolization
 - CNS (stroke), kidneys (AKI), spleen (splenic infarct), spine (anterior cord syndrome)
 - Risk factors: left sided IE, vegetations that are mobile, large (>10mm) or increasing in size
 - Majority occur within 1-2 weeks of initiating antibiotic treatment → keep inpatient for at least 1st 2 weeks, before considering OPAT,
- Acute renal failure
 - Multifactorial: multiorgan failure from septic shock or cardiogenic shock, renal infarct from systemic embolization, immune complex glomerulonephritis, interstitial nephritis from gentamycin or vancomycin
 - Monitor urine output and renal panel closely, may need renal replacement therapy

How do I manage this patient?**(a) Medical**

- Management is multidisciplinary involving ID physician, cardiologist, +/- cardiothoracic surgeon
- Begin empiric IV antibiotics covering suspected organisms (e.g. viridans Streptococci, Streptococcus bovis, S. aureus, Enterococcus). Regimens e.g. benzylpenicillin + gentamicin, vancomycin if suspect MRSA.
- Antibiotics should thereafter be guided by culture results
- Fever should resolve within days of starting IV antibiotics
- Refer to dentist if suspecting dental source for IE.

(b) Surgical

- May be necessary for total removal of infected tissue and reconstruction of cardiac morphology
- Early surgery (during initial hospitalization before completion of full therapeutic course of antibiotics) may be required if
 - Heart failure from valve dysfunction
 - Periannular extension: Heart block, abscess, destructive lesion
 - Persistent infection: fever or bacteremia in spite of 5-7 days of IV appropriate Abx
 - Difficult to treat: Left sided IE caused by *S. aureus*, fungi or highly resistant organisms
 - Recurrent emboli and persistent vegetation in spite of appropriate antibiotics
- If not for early surgery, ensure fully treating extra-cardiac infection so that new implant does not get infected. Can consider doing bypass at same time if needed.

(c) Subsequent antibiotic prophylaxis

- Only for cardiac conditions highest risk for IE: prosthetic valve, previous IE, unrepaired cyanotic congenital heart disease
- Only for procedures high risk for IE:
 - Dental operation
 - Biopsy of respiratory mucosa (tonsillectomy, etc)
 - Surgery involving infected skin or tissue
 - NOT required for routine scopes
- Regimens: amoxicillin 2g PO once, ampicillin 2g IV once, clindamycin 600mg PO once (if penicilin allergic)

(d) Treatment of source

- *Streptococcus bovis* IE: associated with CA colon, do colonoscopy

Neuro: Acute Stroke

ADULTS

Patients may be at different timepoints of disease: acute stroke – post-stroke follow-up – old stroke + cardiovascular risk factors; in each case the clinical focus is different. This topic discusses the whole range but be sure to tailor it to ‘your’ patient.

Is this a stroke?

- Diagnosis is clinical *not* based on ‘scan’
- Typical presentation:
 - *Acute* onset (minutes) → characterizes a vascular lesion
 - Unilateral neurological deficit → e.g. weakness of face and/or limbs
 - UMN signs (hyperreflexia, hypertonia, Babinski): take hours-weeks to develop, in acute setting paralysis is flaccid
- If symptoms appear transient → consider transient ischaemic attack (see appendix)
- Do not forget to consider and exclude stroke mimics:

Stroke Mimic	Feature
Hypoglycemia	Patient on DM medications Always do hypocount → excellent mimic of stroke.
Epilepsy - partial seizure with Todd’s paralysis	Noted seizure: witnessed or past episodes Improves rapidly, no lasting neuro deficit.
Structural intracranial lesion - AVM - Subdural hematoma - Tumour	Can’t tell: need scan Hx of head trauma Chronic progressive hemiparesis and/or headache.
Infection - meningitis, encephalitis	Fever, photophobia, neck stiffness
Migraine	Hx of migraines (but beware of the headache of a different nature that could be a SAH)
Inflammatory - Multiple sclerosis	Younger females, more subacute in presentation

Where is the stroke?

- Anterior circulation
 - Cortical: suggestive in the presence of dense hemiplegia and cortical signs (aphasia, hemineglect, sensory/visual extinction, apraxia, agnosia, hemianopia, *drowsiness*)
 - Subcortical (lacunar strokes): *no* cortical signs, known lacunar stroke syndrome which involves at least 2 contiguous areas (FAL, FA or AL):
 - Pure sensory
 - Pure motor
 - Sensorimotor
 - Ataxic - weakness and ataxia on ipsilat side
 - Dysarthria clumsy-hand
- Posterior circulation: brainstem, cerebellar or isolated occipital lobe involvement, e.g.
 - Crossed hemiparesis
 - Disorder of conjugate eye movements
 - Ataxia without weakness on ipsilat side
 - Isolated hemianopia or cortical blindness
 - *Drowsiness*

What is the mechanism/underlying etiology?

- Workup - see individual sections.
- Clinical suspicion of likely stroke etiology is based on patient’s risk factors as well as the localization of stroke -

Ischemic			Hemorrhagic
Thrombotic	Embolic	Hypoperfusion	
CVM risk factors: DM, HTN, HLD, smoking Vasculopath: IHD, PVD, CKD	Cardioembolic: AF Artery-to-artery emboli: carotid stenosis	Systemic shock: haemorrhagic, cardiogenic, septic	Uncontrolled hypertension Coagulopathy Known AVM
Lacunar infarcts Unilateral well- defined territory e.g. MCA infarct	Large vessel territory (e.g. MCA infarct) Multifocal infarcts (spray of emboli)	Infarct seen in watershed area	Bleed visible on scan

Acute stroke - early management:**(1) Immediate resuscitation**

- ABC, Q1H paras & consciousness level, hypocount, supplemental oxygen, NBM and NG tube, IV drip 2 litres
- Is there any raised ICP: worsening headache and vomiting, drop in GCS, Cushing's reflex (late), papilloedema on fundoscopy, false localizing CN6
 - Supportive: elevate head 30 degrees, IV mannitol 20% 0.25-0.5g/kg over 20min Q6-8H up to x 3/7, hyperventilate PCO₂ 25-30mmHg,
 - KIV intubate esp patient drowsy and unable to maintain saturation,
 - KIV call neurosurgery for decompression

(2) Definitive treatment

- Send for CT brain or MRI stroke protocol early: to distinguish ischaemic stroke vs haemorrhagic stroke, not for diagnosis.

(a) Ischemic

- Does the patient qualify for IV thrombolysis? (recombinant tissue plasminogen activator)
 - Onset of symptoms within 4.5h - if unsure (e.g. woke up weak), take time patient last seen to be neurologically normal?
 - Measurable neurological deficit
 - Age > 18 yrs
 - No high-risk for bleed: no recent stroke, neurosurgery, brain tumor, AVM, bleeding diathesis, active internal bleed, massive infarct, platelet <100, INR >1.7
- If not for thrombolysis, are clot retrieval techniques e.g. Solitaire, TREVO, MERCI (subject to availability) possible?
- Early secondary prevention: antiplatelet therapy with either aspirin or clopidogrel
 - If aspirin: 300mg stat, 100mg OD thereafter. Add dipyridamole for high risk patients
 - If clopidogrel: 75mg stat and OD thereafter.
 - Give PPI cover
- Be wary of complications: cerebra edema, haemorrhagic conversion if thrombolysis given
 - If patient becomes more drowsy or neurologically deteriorates post thrombolysis → re-CT for haemorrhagic conversion.
 - Early decompression for malignant MCA infarct (50% MCA territory, NIHSS >15, drowsy) → within 48 hours of onset, 18-60 years old

(b) Hemorrhagic

- Rx is mostly supportive
- Monitoring of GCS and ICP, maintaining oxygenation
- Surgical decompression in selected cases
- Consider seizure prophylaxis.

(3) Manage BP

- Concept is to allow *permissive* hypertension so as to maintain cerebral perfusion.
- Targets:
 - Ischaemic stroke, *no* thrombolysis: keep <220/120 (lower 15% in 1st 24h)
 - Ischaemic stroke, for thrombolysis: keep <185/110
 - Haemorrhagic stroke: target 160/90 (if raised ICP, difficult situation -- monitor ICP and titrate BP according to ICP)
- Unusual situations
 - If hypotensive → find out why
 - If other indication for aggressive BP reduction e.g. aortic dissection → treat BP regardless of above targets

(4) Treat underlying cause

- Treatment depends on underlying mechanism of stroke. Identify the clinically likely stroke mechanism and workup as:
 - Neurovascular imaging: MRI/CT angiography, carotid ultrasound (anterior circulation stroke), MRI neck (posterior circulation stroke)
 - Cardiac evaluation: echocardiogram, Holter monitoring for AF.
- Thrombotic and embolic stroke: Control vascular risk factors (DM, HTN, HLD), start statins regardless of lipid levels
- Embolic stroke: search hard for AF (including paroxysmal), rate control
 - Anticoagulate → first ensure no haemorrhagic conversion; repeat CT before initiation, delay initiation for 2 weeks if hemorrhage is evident
- Haemorrhagic stroke: control hypertension, treat coagulopathy
- Young stroke: workup e.g. prothrombotic disease, vasculitis (out of scope of this text).

(5) Aggressive early rehabilitation

- Often neglected, but crucial to maximise functional recovery.
- If non-ambulant or other impairment: be wary of aspiration pneumonia (speech therapy assessment), decubitus ulcers.

Acute TIA - early management:

- Clinical significance of a TIA: portends a high risk of stroke
 - ABCD2 scoring to risk stratify: age, BP, clinical feature, duration, diabetes.
 - May require hospitalization for workup if moderate to high risk.
- Needs urgent workup to determine etiology of TIA - as for a stroke
- Treat identified etiologies urgently.

Post-stroke follow-up - issues & management:

- Has all the above been done? > Find and treat etiology and risk factors, prevent re-stroke
- Follow up on rehabilitation progress and function
- Lifestyle adjustments to cope with new functional status
 - Swallowing impairment → diet adjustment, NG tube or PEG
 - Mobility assistance devices
 - Nursing care

All patients - social issues**(1) How has this affected the patient?**

- Pre-morbid status (ADL, ambulation) vs current status: is there a big drop?
- Occupation: how badly will this impact patient's ability to work? E.g. taxi drivers cannot drive for a year post-stroke
- Hobbies: how badly will this prevent patient from enjoying them? QoL
- Psychologically, how is the patient dealing?
 - Reactive/post-stroke depression is common
 - Patient's understanding and attitude towards condition is important as this influences patient motivation to participate in rehabilitation AND control cardiovascular risk factors

(2) How is the patient's social and financial support?

- Who does the patient stay with?
- Will the patient be able to take care of himself on discharge home? If not, does he have competent caregivers?
- Does the patient have the financial reserve to put himself through this period of loss of income? If not, does he receive financial support from anyone else?

Sample summary: Mr Muthu is a 70 year old Indian gentleman with significant past medical history of poorly-controlled diabetes, hypertension, ischaemic heart disease. He presented two weeks ago with acute onset left hemiparesis and left facial droop. I understand he received thrombolysis for ischaemic stroke. Thereafter he underwent rehabilitation and has currently regained a good deal of left-sided motor function with power 4/5 in most areas. He is able to ambulate and has no swallowing impairment. In terms of the underlying cause, he was noted to have an irregularly irregular pulse, and has since been started on warfarin. My main issues for this gentleman are

1. Right cortical/subcortical ischaemic stroke, s/p thrombolysis
2. Left hemiparesis undergoing rehabilitation.
3. Newly diagnosed atrial fibrillation, on warfarin.
4. Poor patient understanding of disease, likely poor compliance if not addressed.
5. Social isolation: stays alone, divorced, and estranged from children.

Neuro: Cerebral Palsy

PAEDIATRICS

How did this present > is it cerebral palsy?

- Definition: Cerebral palsy is a heterogeneous group of permanent, non-progressive clinical syndromes characterized by motor and posture dysfunction, secondary to an insult to the developing brain before the age of 2 years.
- Implication: Trace the developmental hx of the child carefully > it is a *static* insult, *before 2 years* of age. Regression after 2 years is not CP
- Presentations include developmental delay, difficulty walking, etc.

Which type of cerebral palsy?

- Spastic: diplegic, quadriplegic, and less commonly hemiplegic. Elicit UMN signs.
- Dyskinetic: dystonic vs choreoathetotic (observe the involuntary movements).
- Ataxic

What is the etiology of CP in this child?

- Antenatal: TORCH infections, chorioamnionitis, congenital brain malformations, prematurity -- careful antenatal history.
- Perinatal: hypoxic ischemic encephalopathy
- Postnatal: stroke/intracranial hemorrhage, meningitis, severe drowning, kernicterus -- ask in hx, examine for any VP shunt

What are the issues - prioritise - and how are they managed?

- *Motherhood statement* (but particularly critical in CP): management requires a multidisciplinary team with a holistic approach - working with the family to maximize the child's social and emotional development, communication, education, nutrition, mobility, and ADL independence

(a) Intellectual & Communication

- The degree to which intellectual development is affected varies - spastic diplegics may be pretty smart, while others may not even communicate. Visual disorders, hearing impairment, and speech disorders are also common.
- Special schools: e.g. Cerebral Palsy Association and Rainbow Centre (Yishun, Margaret Drive) for patients up to 18 years old -- but what happens after the child turns 18?
- Technology has allowed development of augmentative communication strategies for nonverbal children or those with poor language skills - e.g. picture symbols and voice output device

(b) Motor: mobility and ADL independence

- Again, the range of mobility is quite wide -- get to know the child; assess function both in biological (can stand) and social terms (can walk in the house)
- The relatively mobile child may benefit from PT, OT, and motorized wheelchairs.
- Is spasticity bothersome? Try PO antispasmodics (baclofen, dantrolene), botulinum toxin injection, selective dorsal rhizotomy (interrupt afferent limb of reflex arc)
- Are there contractures? Try muscle tendon release surgery
- Hip dislocations: conservative or reconstruction
- The immobile: management of complications e.g. sacral sores.

(c) Nutrition

- Feeding problems are common in children with cerebral palsy
- Any aspiration pneumonias and how have they been managed?
- Is intake adequate? > Growth of child
- Is there a need for NG tube or PEG feeding? If so, have they been carried out? If not, why?

(d) Seizures

- A common comorbidity in CP > refer to discussion on seizure.

What are the care arrangements?

- Especially child is ADL dependent
- What are the concerns with the care arrangement?
- Is there caregiver stress? > Special schools provide some reprieve.
- Social and financial concerns.

Have others been able to accept the child?

- E.g. family members, peers
- Drooling can be quite disconcerting. Consider behavioural therapy (oral motor skills training to improve lip and jaw closure), anticholinergics (e.g. glycopyrrolate).....
- Urinary incontinence: toileting schedule, diapers, clean intermittent catheterization.

Sample summary: See P. is a 12 year old boy with spastic diplegic cerebral palsy. He was a 32-weeker, requiring prolonged neonatal ICU care, and was diagnosed with cerebral palsy when he failed to walk at 1.5 years of age. He also suffers from epilepsy with monthly breakthrough seizures. Functionally, he engages in simple communication, is able to assist with self-care, is able to get around on a motorized wheelchair. He attends a special school. He has a good social setup with a supportive mother, however she seems to be suffering from some caregiver stress. My problem list is

1. Epilepsy with breakthrough seizures - mother does not know how to give rectal diazepam and tries to stuff spoon in mouth when he seizes.
2. Caregiver stress
3. Spastic diplegic cerebral palsy

Neuro: Headache & Migraine

ADULTS & PAEDIATRICALS

What is my diagnosis?

- The key to diagnosis is characterizing the nature of pain (a classic use of the ‘SOCRATES’ mnemonic - not an exhaustive list of what to consider, but things you have to be able to answer by the time you finish the consult).
- Time course is important - distinguish acute vs chronic vs recurrent/episodic
- Note: Any new onset headache in a patient >50 years old → worry about tumor, giant cell arteritis

(1) Have I ruled out dangerous causes?

- Acute generalized headache:

Cause	Characteristic	Workup	Treatment
Subarachnoid haemorrhage	Thunderclap: sudden onset, max at onset Worst headache ever had Suspect in ADPKD pts	CT brain (best within 12h) LP if CT -ve > for xanthochromia	Coiling (radiology) Clipping (neurosurg)
Meningitis	Fever, photophobia, neck stiff Altered mental state Purpuric rash	LP Blood c/s	Ceftriaxone, vanco +/- ampicillin (listeria) Steroids
Haemorrhagic stroke	Neurological deficit	CT brain	See stroke
Hypertensive crisis	Uncontrolled BP	CT brain (ddx SAH)	BP management
Venous sinus thrombosis	Acute: like SAH Subacute: raised ICP, focal signs, seizure, encephalopathy CT and LP negative		Prothrombotic screen CNS/ENT cancer Autoimmune screen Anticoagulation (clexane then warfarin)

- Chronic progressive headache
 - Tumour: worse on supine position / coughing / sex, associated nausea & vomiting +/- progressive focal neurological deficits (e.g. CN6, hemiplegia, seizure). In pituitary tumours, look for bitemporal hemianopia and features of acromegaly

(2) Are there other possible secondary causes?

These can either be acute first episode localized (consider together with acute first episode generalized group), or recurrent episodic (consider together with primary headache group).

- Under this category, the next important thing to rule out is if there are any *vision threatening* causes: Giant cell arteritis, glaucoma
- Giant cell arteritis: >50yo, subacute throbbing headache over superficial occipital or temporal arteries, a/w jaw claudication on chewing
 - 75% raised CRP and ESR, diagnosis confirmed on biopsy (within 2 weeks),
 - Treat with prednisolone to prevent blindness
- Glaucoma: unilateral painful red eye with fixed mid-dilated pupil, diminished peripheral vision, hx of glaucoma
- Sinusitis: hx upper respiratory symptoms, pain behind browbone or cheekbones
- Trigeminal neuralgia
- Benign idiopathic intracranial hypertension: young fat female, headache worse on supine position/coughing/sex. Look for papilloedema.
 - Usually CT brain done first TRO intracranial lesion
 - Dx confirmed with high opening pressure on LP

(3) If likely primary headache, which type?

- There are three primary causes of episodic recurrent headaches (>5-10 episodes) with characteristic differences (see table: diagnostic criteria marked by *)
- Always re-explore the diagnosis to avoid missing ddx.
 - If any change in character or response to treatment of previous headaches → do not simply blame primary headache disorders, worry about other sinister causes

The primary headache disorders: characteristics and diagnostic criteria

	Tension headache	Migraine	Cluster headache
Duration & frequency *	Each 30min-7d	At least 5 attacks Each 4-72h	At least 5 attacks Each 15-180min Frequency: between EOD to 8x/day for >half of the time when the disorder is active
Characteristic of pain * Classic description	At least 2 of - Bilateral - Nonpulsating - Mild or moderate - Not worse on physical activity Pressing/tightening band around head	At least 2 of - Unilateral - Pulsating - Moderate or severe - Worse on physical activity	Unilateral Severe pain Sharp boring pain focused in an around the eye
Associated features*	None of - Photophobia - Nausea/vomiting	Either or both of - Photophobia - Nausea/vomiting	Sense of restlessness or ≥1 ipsilateral autonomic feature - Conjunctival injection or tearing - Eyelid edema - Nasal congestion - Facial sweating - Facial flushing - Fullness in ear - Miosis / ptosis
Some subtypes also have		May or may not have aura: visual, sensory, aphasia, hemiplegia, brainstem dysfunction (dysarthria, vertigo, ataxia, diplopia; exclude stroke)	May be chronic or episodic (lasts 7d-1y, separated by pain-free remissions of ≥1m)

* Diagnostic criteria

Management issues

(1) Trigger avoidance

- Most patients know their trigger e.g. work stress, certain foods (cheese, chocolate, alcohol), flashing lights → can this be avoided?
- Behavioural management strategies; lifestyle modification
 - Regular sleep, exercise, meals
 - Stress management
 - Cognitive behavioural therapy
- Rule out medication overuse headache: if headache is persistent or worsens during medication use, ≥ 15 days/month for at least 3 months
 - Establish pattern of medication use: consider medication overuse headache if using for ≥ 10 days a month (ergot, triptan, opioids) or ≥ 15 days (simple analgesics)
 - If medication overuse headache, need to withdraw overused drug + give pharmacological and nonpharmacological support through withdrawal headaches (antiemetics, antipsychotics, rescue analgesic other than the one overused)

(2) Pain control

- How much is this affecting the patient?
 - Patient satisfaction with current Rx
 - Migraine Disability Assessment Scale: guides stratified approach to pharmacological treatment (low, moderate and high need)
- Acute treatment: principle is to start Rx early and whack hard (better outcome than escalation if not improving).
 - Simple analgesics: paracetamol, NSAIDs +/- caffeine as adjuvant
 - Migraine: serotonin agonists (triptans > ergots), antiemetics (metoclopramide, chlorpromazine)
 - Cluster headache: oxygen (100% 10L for 20min), SC sumatriptan
 - Be wary of side effects and contraindications: e.g. renal disease and NSAIDs, ischaemic heart or cerebrovascular disease and triptans/ergots
 - Adjunctive dexamethasone reduces risk of recurrence.
 - Generally try to avoid opiates → high risk of early recurrence
- Prophylaxis
 - Start if recurrent attacks → cannot use acute treatment >10 days a month
 - Start low dose and titrate up
 - Tension headache: antidepressants e.g. amitriptyline, mirtazapine
 - Migraine: antiepileptics (e.g. valproate), antidepressants (e.g. amitriptyline), beta-blockers e.g. propranolol
 - Cluster headache: verapamil, steroids, lithium.

(3) Therapeutic relationship

- Headache diary: frequency, duration, disability, response, adverse effects to meds
- Establish partnership with patient:
 - Address patient expectations
- Education and collaborative management.
 - Educate on nature and mechanism of disorder
 - Strategies for identifying and avoiding triggers
 - Importance of limiting days of acute Rx to prevent medication overuse headache
 - Maximize compliance by discussing rationale for treatment, when/how to take medications, adverse effects

How is this affecting the patient?

- Personal: how is the patient's mood? Chronic pain predisposes to depression
- Function: is the patient able to work? how often does he need to take leave?
- Elicit any concerns, especially that of brain tumour

Sample summary: Jitter is a 35-year old lady with a 4 year history of episodic recurrent headache; this is severe, unilateral, throbbing, and associated with photophobia. She was diagnosed as migraine and given ergots plus propranolol prophylaxis. In the last half year, however, she complains of progressively worsening headache. She had been under significant work stress and has been taking ergots plus panadol almost daily. My main issues for her are:

1. Medication overuse headache - she is aware of medication overuse headache but feels crippled without medications, and unable to meet work deadlines.
2. Migraine

Neuro: Multiple Sclerosis

ADULTS

What is the clinical presentation?

- Begin by eliciting a history of previous episodes of CNS dysfunction
- Where is the lesion? For example common MS lesions include -
 - Optic neuritis: eye pain accentuated by eye movements, central visual loss, RAPD, disc edema +/- pallor
 - Brainstem disease: Internuclear ophthalmoplegia (bilateral - highly likely MS), hemiparesis.
 - Cerebellar disease: slurred speech, gait imbalance, dysmetria
 - Spine disease: long tract sensory symptoms, diplegia/paraplegia, bladder/bowel dysfunction
- What is the lesion: is it inflammatory?
 - Onset is characteristic: inflammatory diseases like MS have subacute onset over hours-days. In contrast vascular lesions have an acute onset over minutes, while chronic onset (days-weeks) characterises a neoplastic lesion
 - Ddx: infective lesions (meningitis, abscess), episodic lesions (seizure) - ensure no fever and no seizure symptoms.

Is this MS?

- Diagnosis of MS requires demonstrating dissemination in space (DIS) and dissemination in time (DIT), clinically or by MRI criteria (McDonald 2010), with exclusion of ddx
 - Clinical diagnosis: two attacks that localize to two locations
 - MRI evidence of DIS: T2 lesions in ≥ 2 of 4 typical regions (periventricular, juxtacortical, infratentorial, spinal cord)
 - MRI evidence of DIT: new lesion on follow-up scan, or simultaneous enhancing and non-enhancing lesions
 - Disease felt to be MS but not meeting criteria is called 'possible MS'.
- LP is not necessary but if done shows oligoclonal bands

- Be wary of differential diagnoses
 - Neuromyelitis optica (NMO): visual defect more likely bilateral, altitudinal (vs unilateral, central), myelitis more likely longitudinally extensive and affecting entire cross section (vs discrete, affecting part of cross section) → worse prognosis (60% blind and 30% die in 5 years) → do NMO-IgG (AQP4), look at Wingerchuk criteria
 - Acute disseminated encephalomyelitis (ADEM): post-infectious presentation, monophasic illness, lesions on MRI all same age.
 - Autoimmune disease e.g. SLE: joint pains, rash, seizures → do ANA, dsDNA, ANCA, etc.
 - HIV

What is the course of MS?

- MS has four defined clinical courses
- Clinically isolated syndrome: a single monosymptomatic attack compatible with MS is often the first presentation
 - Diagnose MS if there is MRI evidence of DIS/DIT
 - Else observe -- it may subsequently develop into MS
- Relapsing-remitting MS (85%): episodes of relapse (which may recover or have sequelae) and intervening periods without disease progression
- Secondary progressive MS: A longstanding relapsing-remitting MS may secondarily progress i.e. gradually worsen during intervening periods, with minor remissions between episodes
- Primary progressive MS (10%): progression of disability from the beginning, without significant remissions; there may be superimposed relapses/relapses

What are the issues and what treatment has the patient received?

(1) Flares

- What is the course of flares: how often, how troublesome, what is the response to treatment? → Rule out ddx.
- Acute Rx: first line glucocorticoids → no effect in disease activity or disability. Plasma exchange if no response.
- Disease modifying therapy for RRMS e.g. injection (interferon), infusion (natalizumab), and oral
 - If not on → why not
 - Consider starting -- reduces flare frequency although (effect on disability uncertain)

(2) Disability

- MS inevitably progresses to disability, but quite slowly (RRMS: median time to needing walking aid is 28 years; faster in PPMS)
- Elicit if there are gait problems or other disability
 - Supportive measures - physiotherapy, walking aids
 - Spasticity - e.g. baclofen
- Bladder dysfunction
 - Anticholinergics (oxybutynin), intermittent catheterization

How is the patient coping?

- Function: disability, fatigue, ability to work, disruption to life.
- Psychological: depression, anxiety at disease course
- Pregnancy: any intention? > MS gets better during pregnancy but worse post-partum.
- Financial issues

Sample summary: Ms Claire Rose is a 23 year old Caucasian lady with relapsing remitting multiple sclerosis. She was diagnosed 3 years ago when she presented with one-sided weakness/numbness and difficulty coordinating worsening over 3 weeks. MRI brain showed 2 lesions of different ages. She has had 2 relapses since, one of which had additional spine involvement. Her symptoms respond to high dose steroids in all 3 admissions, but her pre-morbid function takes a small dip with every relapse. She now needs a walking aid to ambulate independently and some assistance for her ADLs, but is still able to write and type. Otherwise her vision is unaffected and she is still bladder/bowel continent. She is still hopeful that she can complete her university education and hold a job that is clerical or that allows her to work remotely from home. Her family is extremely supportive - although they cannot afford interferon injections for her at the moment, they are working hard to save up so she has the best shot at preserving her function.

Neuro: Muscular Dystrophy

PAEDIATRICS

How did this boy present?

- Duchenne Muscular Dystrophy: weakness is first clinically noticeable ~ 2-3 years old and progresses thereafter, patients are usually wheelchair bound by 12 years.
- Becker Muscular Dystrophy: weakness starts later and is milder. Patients do not become wheelchair bound before 15 years old.
- Ddx:
 - Consider non X-linked etiologies e.g. limb-girdle muscular dystrophy.
 - Rule out fatigability in myasthenia gravis.
- There will often be a +ve family history.
- Clinical findings are LMN LL weakness affecting proximal before distal muscles, calf pseudohypertrophy, Gower's sign, waddling gait, lumbar hyperlordosis → findings should still be present.
- Initial investigations would have showed: elevated CK, abnormal ECG, myopathic EMG; diagnostic confirmation is via genetic testing (dystrophin) or muscle biopsy.

What are his current status?

- Lower limb motor function: is the patient still ambulant? > Usually wheelchair bound by 12 years old.
- Respiratory function: any respiratory failure, pneumonias?
- Cardiomyopathy: any heart failure, arrhythmias
- Cognitive development: often associated with mild intellectual disability
- Overall health: growth & development

How is he managed?

- Disease modifying therapy: Steroids prolong walking years and delay mortality. Start at 4-5 years old;
- Mobility aids: orthoses, braces, eventually wheelchair
- Vaccinations
- Management of heart failure.

How is the child & family coping?

- Physical: Function, ambulation, and self care.
- Emotional: Awareness of disability, social interactions, self esteem
- Financial issues

Have end of life issues been explored?

- Duchenne patients lose mobility by 12 years and die in late teens or 20s while Becker patients lose mobility after 16 years and can survive beyond 30s.
- Advanced care planning and palliative management is often helpful.
- Gently probe patient's and family's understanding of prognosis and if not adequate, should refer for ACP.

Genetic counselling

- Are other family members involved?
- Do parents understand implication for future pregnancies: X-linked recessive, therefore half of all male children are affected (i.e. 25% of all children), and half of all female children are carriers.
- Are patient's sisters aware that they *may* be carriers?
 - Ethical issue: should parent test the child before child is 21? What if the child does not want to know?
 - Implications for future relationships

Sample summary: Da Tui is a 12 year old boy with Duchenne's muscular dystrophy, diagnosed at the age of 3 when his parents brought him to a pediatrician for delayed gross motor milestones (he started to pull to stand at 1.5 years, walk at 2 years and run at 3 years). He was able to ambulate and participate fully at Rainbow Centre with his walking aids up til the age of 9, and became wheelchair bound by 11. He has never had any cardiac or respiratory complications, but is cushingoid from the steroids he has been taking since 5 years old. Da Tui is accepting of the fact that he will never be strong like the other children, but is still quite happy in school where there are other peers like himself. His parents plan to break the news about his prognosis soon and start advanced care planning. Da Tui has 2 other younger sisters who have not been tested for carrier status -- the parents intend to let them decide on whether to test for carrier status older in life.

Neuro: Myasthenia Gravis

ADULTS AND PAEDIATRICS

How did this patient present → is this MG?

- MG is characterized by a *fluctuating* degree and *variable* combination of *fatigable* weakness which may first affect --
 - Ocular involvement only (50%): non-conforming pattern of ophthalmoplegia (does not fit CN3, CN6, or INO pattern - but sometimes can mimic) + ptosis
 - Generalized involvement (see table)
- For each presentation consider differentials (see table). In particular,
 - Sensory involvement is *not* a feature of MG
 - Lambert-Eaton myasthenic syndrome (LEMS): important not to miss as it is paraneoplastic. In LEMS, weakness and reflexes *improve* on exercise (vs fatigue in MG); it tends not to first present ocular-only disease. If suspicious, test for antibodies to voltage-gated calcium channel (VGCC) and do electrophysiology. If LEMS is confirmed, look hard for a cancer e.g. lung.

Presentation	Classic for MG	Differentials & their features
Ocular	Non-conforming ophthalmoplegia Ptosis (uni or bilat) Pupils are spared	Brainstem & cranial nerve lesions: known pattern of ophthalmoplegia (CN3, CN6, INO), other CN palsy Horner's syn: Miosis, unilateral ptosis, anhidrosis. Thyroid ophthalmopathy: Proptosis, Exophthalmos, symptoms and signs of hyperthyroidism
Bulbar	Fatigable chewing Dysphagia Dysarthria	Brainstem & cranial nerve lesions: other CN palsy Cortical lesion (e.g. stroke): long tract signs Motor neuron disease: mixed UMN & LMN signs. Nasopharyngeal CA
Limb	Proximal weakness Fatigable Sensation spared	Lambert-Eaton myasthenic syndrome (see above) Motor neuron disease: mixed UMN & LMN signs Peripheral nerve: motor neuropathies e.g. CIDP Myopathies: muscle pain, percussion myotonia
Face & Neck	Expressionless face Myasthenic sneer Dropped head syn	Bilateral CN 7 palsy Brainstem disease
Respiratory	Type 2 respi failure Dyspnoea.	Motor neuron disease Myopathies

- Confirm diagnosis of MG via:
 - Bedside tests: Tensilon test (edrophonium), ice pack test
 - Electrophysiologic tests: Repetitive nerve stimulation, single fibre electromyography
 - Serum antibodies: anti-acetylcholine receptor antibody, anti-MuSK (muscle specific receptor tyrosine kinase) antibody

What is this patient's disease course?

- 50% of patients who present as ocular MG may develop generalized myasthenia by two years; no way to predict who will generalize
- Where in the 'classic' trajectory of disease is the patient? → But each case is unique.
 - Early MG: transient symptoms, may even remit spontaneously for weeks
 - Active phase: symptoms progress (in frequency, severity, distribution). This occurs 5-7 years after onset (peak at 3yr). Myasthenic crises may occur.
 - Second phase: symptoms stable but persist; occasional exacerbations precipitated by infection, medication taper, etc
 - Third phase: many patients eventually remit (on meds or off)
- Risk assessment - Hx of myasthenic crisis?
 - How severe → respiratory failure, intubation?
 - What was the trigger? (e.g. infection)
- Is this patient progressing: increasing drug requirements or symptoms?
- What is the functional status now?

What is the patient's treatment, can I improve it?

- What is current mx, is it optimal?
 - Acetylcholinesterase inhibitor: pyridostigmine → symptomatic relief
 - Chronic immunomodulators: steroids, azathioprine, mycophenolate, cyclosporine → indicated if symptomatic on pyridostigmine or respond only temporarily
 - Rapid immunomodulators: plasmapheresis, IVIg → for myasthenic crisis, as a bridge to initiating other immunotherapies, or pre-surgery for mod-severe MG
- Is there a need to offer thymectomy?
 - CT scan for a thymoma → If present, should resect
 - Even if no thymoma, recommend thymectomy in generalized MG: increases chance of becoming asymptomatic or achieving medication-free remission
 - Controversial in ocular MG
- Prevent mortality & morbidity from myasthenic crisis
 - Patient education -- when to seek help
 - Early recognition → generalized weakness can mask respiratory distress
 - Monitoring e.g. negative inspiratory force
 - Careful when stepping down medication

Are there any comorbidities?

- Other autoimmune disease: e.g. Grave's disease, RA, SLE
- Is the patient on other drugs?
 - A lot of drug-disease interactions, need to look up
 - Avoid fluoroquinolones, aminoglycosides, Mg sulfate.
- Is the patient going for surgery? > Beware of anaesthesia in the MG patient
 - Avoid neuromuscular blocking agents if possible: MG patients have unpredictable resistance to depolarizing NMBA (succinylcholine), and unpredictable sensitivity to nondepolarizing NMBAs (eg rocuronium, vecuronium). Anticholinesterases prolong the effect of succinylcholine and delay the onset of nondepolarizing NMBAs.
 - Use short-acting sedatives, hypnotics, and anesthetic agents

How is the patient coping?

- Functional status: able to work?
- Social support: if stay alone -- what happens if enters crisis?
- Financial

Sample summary: Floppy is a 45-year old housewife who presented to the ophthalmologist three years ago with ocular myasthenia. Six months ago her myasthenia generalized to involve her limbs, and she has found it increasingly difficult to do heavy housework. She presented one week ago with increasing weakness and shortness of breath, although on admission her negative inspiratory force and PCO₂ were within normal values. She was managed as for threatened myasthenic crisis with steroids and IVIg and has since improved, although she remains slightly symptomatic. Workup also revealed a thymoma for which she will undergo surgery next week. My issues for her are:

1. Generalized myasthenia gravis with threatened crisis → Need to monitor, educate patient, vaccinate
2. Thymoma in myasthenia gravis for resection

Neuro: Parkinsons' Disease

ADULTS

Is this parkinsonism?

Relatively easy to identify: Tremor, Rigidity, Bradykinesia, Postural instability

Is this idiopathic Parkinson's disease or something else?

- Features supporting / suggesting idiopathic Parkinson's disease (PD)
 - Unilateral onset, persistent asymmetry of signs
 - Resting tremor 4-6 Hz
 - A clear and dramatic beneficial response to dopaminergic therapy
 - Presence of levodopa-induced dyskinesia
 - Progressive clinical course of 10 years or more
- Are there any features to suggest "Parkinsons-Plus" syndromes?
 - Lack of response to levodopa or dopamine agonists in early stages of disease
 - Marked symmetry of signs in early stages of the disease
 - Rapid progression
 - Features specific to each "Parkinson's Plus" syndrome:

Multi-System Atrophy (MSA)	<p><u>Pyramidal</u> signs unexplained by previous stroke or spinal cord lesions</p> <p><u>Autonomic</u> symptoms e.g. postural hypotension, incontinence early in disease course (may occur late in PD)</p> <p><u>Cerebellar</u> involvement</p>
Progressive Supranuclear Palsy (PSP)	<p>Ocular: impaired vertical gaze overcome with doll's eye, eyelid freezing</p> <p>Limbs: Severe postural instability, rigidity trunk > limb, tremor is rare</p> <p>Early onset of dementia</p>
Corticobasal Degeneration	<p>Cortico: Pyramidal tract signs not explained by previous stroke or spinal cord lesions, progressive aphasia</p> <p>Basal: Prominent (myoclonus) apraxia, alien limb phenomenon</p>
Lewy-Body Dementia	<p>Early onset hallucinations or psychosis, precipitated by initiation of low-dose levodopa or dopamine agonists</p> <p>Early onset of dementia (occurs late in PD)</p>
Vascular Parkinsonism	<p>History of cardiovascular risk factors, multiple lacunar strokes</p> <p>Marked symmetry of signs</p>
Extrapyramidal side effect	<p>History of anti-psychotic use or psychiatric disorder</p>

Where is the patient in the course of disease?

- Identify where in the natural course is the patient: PD begins indolently and progresses over years.
 - Motor function is initially good but disability eventually sets in (25% severely disabled at 5 years, 67% at 5-9 years, 80% at 10-14 years).
 - Non-motor manifestations develop late in the course of disease
- Staging systems include Hoehn and Yahr (motor only) and unified Parkinson Disease Rating Scale (more complicated, looks at all aspects)

What are this patient's manifestations & management tasks?

- The main goal of Rx is to preserve the patient's mobility and function for as long as possible, and manage symptomatic non-motor manifestations as they come.
- Hence Rx is all about what is bothering the patient → find out!

(1) Maximise motor function

- Difficulty walking: a product of bradykinesia (shuffling gait, freezing, festination, turning in numbers), rigidity (stooped posture), and postural instability (falls)
- Difficulty communicating: hypomimia (mask-like facies), speech impairment (hypokinetic dysarthria, hypophonia, palilalia), micrographia
- Other features: decreased spontaneous eye blink rate, dysphagia and sialorrhea

(a) Treat dopamine deficiency

- Early PD: <65yo, milder motor symptoms → Try dopamine agonists (Bromocriptine, ropinirole) first
 - Less efficacious but postpone levodopa use and save it for later; long term levodopa inevitably leads to reduced response, motor fluctuations, dyskinesia.
 - Alternative: anticholinergics.
- Later PD: >65yo or any age with prominent motor symptoms: Levodopa-carbidopa (madopar).
 - Assess side effects: e.g. postural hypotension, nausea/vomiting (carbidopa is a peripheral decarboxylase inhibitor, reduces side effects by reducing peripheral levodopa to dopamine conversion)
 - Assess response: progression of motor symptoms, any reduced response or increasing requirements for madopar
 - Assess dyskinesias
 - Assess motor fluctuations: any difficulty titrating dosing to reduce dyskinesias when "on" drug and bradykinesia when "off" drug
- Later lines of therapy - other drugs available as add-on therapy, but no miracles.

(b) Adjuncts to mitigate reduced motor ability

- Walking aids
 - Walking sticks - built in lasers project lines on the floor to break freezing episodes
 - Walker with wheels (please do not give walking frame! PD patients cannot do the many steps to walk with a frame)
 - Wheelchair
- Manage fall risk
- Home modifications
- Social interactions: are these inhibited due to hypomimia, speech difficulties, and writing difficulties?

2. Assess & manage non-motor features**(a) Incontinence**

- Usually more due to inability to reach toilet in time, than true incontinence
- Options include use of diapers, bedside potty/pan, or caregiver to wheel patient to toilet every time

(b) Nutrition and swallowing

- Is the patient getting enough nutrition?
- Difficult issues in end stages → anorexia, dysphagia
 - Loss of interest in food
 - Dysphagia
 - Aspiration risk → pneumonias
- Get speech therapist input: thickened feeds, NG tube?
- Discuss goal of care!

(c) Psychiatric issues

- Psychiatric complications are the most challenging manifestation of PD to deal with and are the main culprits of caregiver stress
- Assess what specific psychiatric issues are present and how this affects patient and carers.
- Pharmacological therapy may be available -- but is this what you want?
 - Dementia: cholinesterase inhibitors (donepezil, memantine)
 - Sleep disturbances → benzodiazepines, non-benzo hypnotics (zolpidem, zolpidem)
 - Mood disorders → antidepressants, commonly SSRI
 - Visual hallucinations → reduce PD drugs, consider atypical antipsychotics

Advanced care planning

- Gently probe patient's and family's understanding of prognosis and wishes → if insufficient, need to gradually discuss.
- Do ACP if not done
 - Clarity on goals of care.
- Palliative management of symptoms.

How are the patient and family coping?

- Disease understanding → focus groups
- What is the patient's current function: ambulation, ADL, ? occupation
- What are the care arrangements?
 - If already has a caregiver → Any carer stress?
 - If not yet → Does he need one?
 - If still ambulant → What is the long-term plan?
- What are the patient and family's emotional and psychological needs: very often there is little understanding of the complex issues involved.
- Financial concerns

Sample summary: Mr Park is a 70 year old gentleman who first presented with unilateral tremor and rigidity upon retirement 7 years ago. He was diagnosed to have idiopathic parkinson's disease and treatment was initially withheld. With disease progression treatment was started 5 years ago, first with bromocriptine and subsequently madopar. Effectiveness has gradually decreased despite uptitration of dose and dosing frequency, with declining motor function, dosing-related dyskinesias and pre-dose freezing. Mr Park is no longer able to walk with a walking stick and spends his days mainly in a wheelchair. He no longer communicates much with his family. The current issues are:

1. Idiopathic parkinson's disease, failing madopar therapy
2. Behavioural symptoms - sleep-wake reversal and night-time agitation which disturbs his family's sleep
3. Care issues - his main caregiver (wife) suffered a stroke last year and is struggling
4. Advanced care planning - goals of care are not established and family does not understand prognosis.

Neuro: Seizure, First Seizure

ADULTS AND PAEDIATRICS

If the presenting complaint is 'seizure', be sure to distinguish whether this requires an approach to first seizure, or is an known epilepsy case.

Is this really a seizure?

- The usual task is to distinguish seizure vs syncope. Obtain history from a witness if available, if not then it has to be distinguished based on pre and post events.
- Rule out other mimics: hypoglycaemia, anaemia, cardiac events, stroke -- always check glucose.
- Seizure
 - Pre-ictal: happened in any position, may have preceding aura e.g. flashing lights, smell, sounds
 - Ictal: loss of consciousness, not responsive to call; witnesses will describe jerking of limbs, uprolling of eyes, clenching of jaw and arching of back. If tongue is bitten, usually at the lateral aspects of the tongue. Urinary incontinence is not a useful feature.
 - Post-ictal: drowsy for up to half an hour, can sometimes have Todd's paralysis
- Syncope:
 - Pre-syncope, happened when suddenly getting up, with a sensation of 'dark closing in' (like James Bond movies)
 - Syncope: witnesses will describe loss of tone and patient sliding to the floor, can have a jerk or two but otherwise quite motionless
 - Post-syncope: no drowsiness on regaining of consciousness
- Screen for causes of syncope anyway, esp when it may not be so clear whether seizure vs syncope
 - Cardiac: preceding chest pain, palpitations, SOB, heart murmurs (syncope is a poor prognostic feature of AS) -- exertional syncope is a bad sign.
 - Postural hypotension: DM, parkinsons, MSA -- do postural BP
 - Drugs: HTN meds (postural hypotension), antidepressants
 - Vasovagal: positional change, on micturition

If likely seizure > what pattern?

- Generalized: Petit mal (absence) vs Grand mal (tonic-clonic)
- Partial: simple partial (normal conscious level) vs complex partial (impaired conscious level) vs secondary generalization → look for a focal lesion!

What is the course of seizure thus far & what is response to treatment?

- Abort spontaneously without treatment
- Abort after rectal diazepam or IV benzo (lorazepam 4mg, diazepam 5mg)
- Status epilepticus = seizure ≥ 5 minutes or 2 seizures without recovery of consciousness in between \rightarrow phenytoin, barbiturate coma \rightarrow ICU.
- Any injury

What is the etiology? > look for any provoking factors present?

- Infection e.g. meningitis - fever, neck stiffness, photophobia
- Metabolic: look for background setup and measure the electrolytes
 - Hypoglycemia: diabetic?
 - Hypercalcaemia: urolithiasis, abdominal pain, hx of parathyroid problems
 - Hyponatremia: poor oral intake, hx liver/cardiac/renal failure, hx TURP
 - Hypokalemia: e.g. gastroenteritis, vomiting.
 - Drugs and alcohol: withdrawal, delirium tremens?
- Intracranial lesion \rightarrow consider CT brain.
 - Acute stroke, SAH, or cranial trauma
 - Previous strokes now with scar epilepsy
 - Brain tumour - preceding history of constant progressing headache, one-sided weakness/numbness or difficulty walking
- [Paeds] Is there an underlying syndrome or seizure disorder?
 - Developmental milestones: have they been on time? have they been regressing?
 - Any dysmorphism?
 - Any neurocutaneous stigmata?
 - Any family history of epilepsies?
 - Any abnormal neurologic findings
- [Paeds] Febrile seizure - Has the child been febrile from intercurrent illness with a rapid rise in temperature? Any family history of febrile seizures?
 - Simple febrile fit: typical epidemiology (6 months - 6 years), typical seizure (GTC, <15min, do not recur in 24h), otherwise normal child (normal development, no neuro findings)
 - Complex febrile fit: does not fit the above.
 - Diagnosis of exclusion so exclude the above causes first.

What is the management?

- Workup and treat precipitant
- If precipitant will persist (e.g. new stroke, trauma), consider antiseizure prophylaxis.
- If apparently idiopathic: do not diagnose epilepsy on a 1st seizure unless there is a high probability of further seizures (equal to recurrence risk after two unprovoked seizures i.e. 60% - e.g. if focal brain lesion), or a clear epilepsy syndrome \rightarrow if suspicious, consider EEG.

APPENDIX: Paeds febrile fit – Communication to parents

- What is this condition? Ma'am, what your child has had is called a febrile seizure. It occurs when temperatures rises rapidly during a fever. It tends to occur in children between 6 mth old to to 6 yrs old. It must have been very scary for you, but this happens quite commonly. It does not cause brain damage, or any delay in your child's development, as long as it is stopped as soon as possible.
- Can it happen again? Yes - the risk of your child having another febrile seizure is 1 in 3, and a further 1 in 3 will have ≥ 3 seizures. Recurrence is higher if onset before 1 yr old and if there is positive family history. But it has a benign course -- only 1% develop epilepsy, which is the same risk as the general population.
- What to do if it happens?
 - Stay calm, take note of the time it started
 - Clear a space on the floor and position child on side, keep sharp objects away. Do not restrain child or put objects into mouth.
 - Give rectal diazepam: twist top off, spread open your child's butt cheeks, insert the tube into the anus and squeeze in the contents. Then remove the tube and squeeze the butt cheeks together to prevent the diazepam from spilling out
 - Try to bring the fever down (after the fit has ceased) with paracetamol or sponging (do not feed any medication orally while your child is still drowsy).
 - Always bring your child to the doctor if in doubt
 - Bring the child to A&E (call ambulance) if: 1st episode, >5min, child unable to move one side of body, unusual drowsiness after fit, injury during fit.

Neuro: Seizure, Epilepsy

ADULTS AND PAEDIATRICS

If the presenting complaint is 'seizure', be sure to distinguish whether this requires an approach to first seizure, or is an known epilepsy case.

Elicit disease course:

- How was the diagnosis made (how does this patient fulfill the definition of epilepsy)?
 - When did the seizures start?
 - How many seizures and their temporal relationship to each other and to the current moment?
 - ILEA definition: (1) At least two unprovoked (or reflex) seizures occurring >24 h apart, or (2) one unprovoked seizure + probability of further seizures similar to the general recurrence risk (at least 60%) after two unprovoked seizures, or (3) Diagnosis of an epilepsy syndrome
- Usual seizure morphology / Seizure signature
- Etiology: idiopathic epilepsy or any known underlying predisposition to recurrent seizures?
 - Stroke
 - Structural brain malformation
 - Epilepsy syndrome, neurocutaneous syndrome

What has the management been so far?

- Was patient started on AED?
 - If so, what? How many agents, what is the dosage, has regime been stable?
- Have there been any side effects?
 - Bone marrow suppression
 - Liver toxicity
 - Teratogenicity: if female and childbearing age → ask about sexual history, use of contraception; if intending to get pregnant needs very careful discussion

How has control of epilepsy been?

- What is the frequency of breakthrough seizures?
- Explore precipitants
 - Drug compliance
 - Precipitants e.g. alcohol, stress, sleep deprivations, new drugs e.g. antibiotics (cipro), painkillers
 - Any other reversible causes? e.g. hypoglycemia, electrolyte disturbances, meningitis
- What is the nature of breakthrough seizures
 - Complicated e.g. status epilepticus?
 - Require admission for AED titration?
- Patient education: does the patient know when to go to the hospital?
 - Worrysome for non-epileptic seizure: high fever, localizing neurology, change in seizure character.
 - Complicated seizure: status epilepticus, prolonged drowsiness after seizure
 - Needs titration: increasing frequency of breakthroughs with no identifiable triggers.

How has this affected the patient?

- [Paeds] Milestones and development, participation in school, social set-up at home
- Occupation: ability to find a job?
- Social: ability to maintain friendships / relationships / stigma
 - In a lady, has being on AED kept her from childbearing?
- Function and precautions
 - Patients with epilepsy are legally prohibited from driving
 - Should not be allowed to swim
 - Discouraged from cycling, climbing or scuba diving
- Financial situation

Sample Summary: Mr Jerk King is a 30/Chinese/M with epilepsy, diagnosed in his teenage years, which was deemed idiopathic in etiology. He has always been less than optimally compliant to antiepileptics, and has had one prior episode of status epilepticus two years ago. In the past month he has had 5 breakthrough seizures. He has had difficulty holding down employment, and lost his most recent job 2 months ago, after he had an epileptic fit in front of his boss. He has also become increasingly socially isolated, and financial issues hinder compliance to AED. My issues are:

1. Breakthrough seizures 2' noncompliance to antiepileptic medications 2' financial difficulty
2. Social: financial difficulty 2' unemployment, social isolation
3. Background of idiopathic epilepsy on Keppra

Neuro: Spina Bifida

PAEDIATRICS

How did this child present?

- Spina bifida varies in severity: from obvious myelomeningocele to spina bifida occulta.
- Open myelomeningocele may be detected prenatally on AFP screening and ultrasound, or it may be an obvious lesion noted at birth.
- Spina bifida occulta is usually picked up later. It is difficulty walking, urinary problems, or orthopaedic issues of the lower limbs (these lesions may progress with age - tethered cord syndrome) that prompts medical consult; and an astute GP or a paediatrician who examines the back and finds a sacral dimple or tuft of hair. Worsening neurology may be indicative of tethered cord syndrome, where the cauda equina is tethered to more superficial structures and is stretched as the child grows.
- Symptomatic patients would probably have had spine surgery → in the neonatal period for open myelomeningocele, much later for spina bifida occulta.
- Classic examination findings are: lower limb LMN diplegia with a tuft of hair or surgical scar on the lumbar spine.

What are the current issues; can we optimise management?

(1) Lower limb function & mobility

- What is the LL neurological function like? → which spinal cord levels are affected and to what extent is sensation lost?
- Is the child at least partly mobile
 - Will aids help e.g. motorized wheelchair
 - Has received PT/OT?
- If immobile > any complications of immobility?
- There are also associated orthopaedic problems in the lower limb e.g. foot deformities
 - Will the child benefit from orthopaedic intervention?

(2) Urinary function

- Is there neurogenic bladder?
 - What are the symptoms -- often an insensate bladder with no stretch reflex +/- overflow incontinence
 - Should be confirmed on urodynamic study.
- How has this been managed
 - If cognitive status is good or if carer is available, clean intermittent catheterization is probably best. Assess technique and check that the patient does it frequently enough e.g. 4x a day.
 - Less optimal: permanent IDC, diapers
- Complications
 - Vesicoureteric reflux → Do micturating cystourethrogram; anticholinergic may help
 - Recurrent UTIs → How severe, any admissions, any resistant organism colonization. → Do DMSA for renal scarring, consider prophylactic Abx
 - Hydronephrosis, pyelonephritis --> Renal impairment

(3) Bowel function

- Many patients also have bowel issues: constipation and incontinence → does this patient have it and how is he/she coping?
- Give laxatives, suppository → ensure regular BO and avoid impacted faeces, spurious diarrhoea.

(4) Associated Chiari malformation

- Myelomeningocele (but not spina bifida occulta) may be associated with Arnold-Chiari malformation: downward displacement of cerebellar tonsils, causing CSF outflow obstruction and hydrocephalus.
- This may manifest as hydrocephalus requiring ventriculo-peritoneal shunt
 - In the infant head circumference should be carefully monitored.
 - If a shunt has been done, what complications have arisen? > Infection, blockage, overshunting
 - At each visit ask for any symptoms -- e.g. headache (classically worse on recumbancy) → any symptoms warrant a CT brain looking for interval increase in ventricular size, which would imply shunt malfunction.
- It may also cause brainstem dysfunction e.g. lower cranial nerve palsy, difficulty swallowing
- Finally Chiari malformation tends to affect cognitive development as well.

How is the child's growth, development & overall health

- Growth and development
- Schooling: type of school (special or mainstream), any learning problems

How is the child / family coping with this illness.

- Coping with intermittent catheterization and/or fecal incontinence
- Awareness of disability, self-esteem and peer relationships; social ostracization
- Financial issues
- Mobility and lifestyle

Are future pregnancies protected?

- Explore antenatal risk factors -- spina bifida is a neural tube defect and associated with folate deficiency (dietary or drug e.g. methotrexate), antiepileptic drug use, as well as DM.
- Has mum received counselling on this? > Advice for folate supplementation, review of DM control, stop drugs

Sample summary: Fifi is a 16 year old Chinese girl with background spina bifida occulta, currently admitted for upper urinary tract infection. She diagnosed at the age of 3 when she had delayed gross motor milestones with an inability to be potty trained, and an astute GP noticed a tuft of hair at the small of her back. There has been no progression of her neurological symptoms to suggest tethered cord syndrome. She is ADL-independent and community ambulant with her orthosis and currently attending a local secondary school. She is urine incontinent but bowel continent. She performs self- intermittent catheterization 4 times a day, and sometimes has trouble keeping aseptic technique when she is rushing to do it while in school in between classes or extracurricular activities. She gets lower UTIs once or twice a year treated with oral antibiotics outpatient, but this is the first she has had fever and loin pain. Since admission she has responded well to IV antibiotics and has been told that her kidney function is good. Her mother, an epileptic on valproate, decided against having any more children after being counselled on the link between valproate and her daughter's spina bifida.

Neuro: Spinal Muscular Atrophy

PAEDIATRICS

How did the patient present and how was diagnosis confirmed?

- SMA is characterised by the *progressive* degeneration of anterior horn cells at various tempos
- Presentation differs depending on the clinical phenotype of SMA.
 - SMA Type 1: most severe form, presents in neonatal period, does not survive beyond 1 year due to respiratory failure
 - SMA Type 2: intermediate form, presents between 3-15 months with delayed gross motor milestones.
 - SMA Type 3: mild form, presents ≥ 1 year with delayed gross motor milestones, or more subtly with foot drop or tripping over feet
 - SMA Type 4: adult form, presents in 2nd to 3rd decade of life.
- Outcome depends on severity of muscle weakness at presentation (rather than age of onset), but earlier onset tends to correlate with greater weakness
- Confirmation of diagnosis would have been performed via:
 - Molecular genetic testing: exon 7 deletion of the SMN gene
 - Electromyography: fasciculations, fibrillations. positive sharp waves, high amplitude long duration motor units
 - Muscle biopsy: grouped atrophy
 - Creatinine Kinase will be normal or mildly elevated (unlike myopathy)

What are the visible clinical manifestations?

- Elicit a symmetrical LMN (flaccid, areflexic) weakness, greater in proximal than distal muscles, greater in lower than upper limb. Fasciculations are prominent and best seen in tongue. Sensation is completely spared.
- Look for manifestations of the issues below

What is the clinical course and current issues?

- No disease modifying treatment for SMA exists. Management is supportive.
- As you explore the common issues below, be mindful of the *course of the disease* and the *tempo of progression* (pegged very closely with patient's functional status).

(1) Musculoskeletal

- Muscle weakness is relentlessly progressive and will eventually result in increasing disability
 - The still-walking child → consider orthoses, walking aids where necessary
 - The non-ambulant child → consider functional aids e.g. motorized wheelchair
 - The bedbound child → watch for complications of immobility e.g. bed sores, ask about nursing care, turning; contractures and physiotherapy to minimise contractures
- Orthopaedic issues: e.g. scoliosis secondary to weakness of paravertebral muscles
 - How bad is it? To a point where it is contributing to the patient's respiratory problems?
 - Spinal bracing can be used to delay progression, but use with caution as it can reduce expiratory tidal volume when patient is sitting up

(2) Respiratory muscle weakness

- Respiratory weakness and pneumonia is the ultimate cause of death in many patients, as SMA patients have difficulty clearing lower respiratory tract secretions + high aspiration risk
- Infections: what is the course of infections, how have they been treated?
 - Vaccinations
- Secretions: are these troublesome?
 - Buscopan → Reduce secretion
 - Chest physiotherapy and postural drainage (manual or mechanical)
 - Cough assist device or mechanical insufflation/exsufflation device
 - What is the compliance to such assistance?
- In end stage may require ventilatory assistance, especially during sleep where pts are especially prone to hypoventilation
 - Oxygenation can be aided with non-invasive ventilation or tracheostomy with traditional ventilation
 - However this should be individualized for each patient in each stage of illness

(3) Maximization of social, language and intellectual skills

- Growth and development
- SMA patients are intellectually normal
- Special schools: e.g. Rainbow Centre (Yishun, Margaret Drive) for patients up to 18 years old -- but what happens after the child turns 18?

How is the child and family coping?

- Function: ADLs, ability to attend school or work
- Care: what is the care arrangement, is there caregiver stress.
- Socially: peer integration, family support,
- Psychologically: awareness of disability, self esteem
- Financial issues.

Have end of life issues been explored?

- Prognosis: SMA type 1 babies die within 2 years, SMA type 2 patients survive into adolescence or young adulthood. SMA type 3 patients may live to adulthood.
- Advanced care planning and palliative management is often helpful for SMA type 1 and 2.
- Gently probe patient's and family's understanding of prognosis and if not adequate, should refer for ACP.

Genetic counselling

- Are other family members involved?
- Do parents understand implication for future pregnancies → How do they feel?
- Are patient's siblings aware that they *may* be carriers?
- Affected individuals and families should be referred for genetic counseling. SMA is generally autosomal recessive but may have other inheritance patterns as more than 1 gene is involved.

Sample summary: Bohlak is a 23 year old boy who was diagnosed with spinal muscular atrophy type 2 at 2 years old, when he presented with delayed gross motor development. Over the years he has progressively lost muscle function, having been wheelchair bound since 12 years old, and in the last 3 years losing most hand movements except finger movements. In terms of respiratory status, he has suffered recurrent pneumonias and requires required night-time ventilatory assist. He uses his secretion clearance device (Acapella) regularly. Functionally, he is mentally intelligent, bedbound but able to occupy himself surfing on a laptop, and maintains a widely-read blog site. His mother is the main carer and they are financially tight but coping. My main issues are:

1. Spinal muscular atrophy type 2, bedbound
2. Respiratory muscle weakness with recurrent pneumonias → to vaccinate, improve chest physiotherapy.
3. Advanced care planning.

Renal: CKD & ESRF

ADULTS & PAEDIATRICS

Where along the spectrum of CKD is this patient?

- Ensure that the patient's complaint is CKD and not AKI: by definition renal impairment >3 months (see approaches notes)
- CKD is a spectrum ranging from asymptomatic CKD to dialysis-dependant ESRF
 - KDIGO classification according to GFR: CKD 1 (>90 ml/min/1.73m²), 2 (60-89), 3A (45-59), 3B (30-44), 4 (15-29), 5 (<15).
 - KDIGO albuminuria grading (measures kidney damage): A1 (<30 mg/day), A2 (30 - 300), A3 (>300)
- Know how your patient developed and where he is now -
 - Has patient been told to start, or is already on dialysis? → ESRF
 - If on dialysis, was this scheduled or crash-land?
- Subsequent issues and goals of management depend on where along the spectrum of CKD this patient is:

Task	CKD, not yet approaching ESRF	CKD approaching ESRF (not on RRT)	ESRF on dialysis
Manage etiology	Investigate etiology, treat & avoid further renal damage	Investigate etiology, treat & avoid further renal damage	Need to know etiology and manage as a comorbid
Manage progression	Retard disease progression	Decide HD vs PD Prepare for dialysis:	Titrate dialysis Manage HD/PD issues
Complications	Usually mild, monitor	Close mx of ESRF complications (not on RRT, vulnerable)	Manage complications of ESRF and dialysis

Note: this document describes clinical tasks for all ends of the spectrum but be sure to tailor your approach to where your patient is (as above). For instance if a patient is clearly CKD3 it would be quite inappropriate to ask about dialysis planning.

Why does this patient have CKD/ESRF?

- May be a known obvious cause (e.g. longstanding DM) or require investigation especially if progressive CKD
- Basic clinical workup: the PMHx is most informative.
 - Pre-renal: diabetes (PMHx, fasting glucose), hypertension (measure)
 - Renal autoimmune disease e.g. glomerulonephritis, SLE, vasculitis → PMHx, ask for proteinuria or hematuria, other symptoms (joint pain, rash etc), do UFEME, ANA, etc.
 - Cystic kidney diseases → ballot kidney, ask FHx
 - Postrenal: longstanding obstructive disease → PMHx, palpate bladder
- May require biopsy if cause occult clinically

Managing the patient's renal function

(1) CKD: how can I retard disease progression?

- Treat cause of CKD: e.g. control DM/HTN, treat urological disease, immunosuppression for glomerulonephritis
- Inhibit proteinuria e.g. ACE-inhibition, ARB
- Avoid further renal insults: hypovolemia, nephrotoxins, obstructive uropathy

(2) Advanced CKD: do I need to start dialysis?

- Emergent dialysis required if -
 - A - Acidosis pH <7.2 or unresponsive to HCO₃
 - E - electrolyte imbalances (refractory hyperK, hypoNa, hyperCa)
 - I - intoxicants (salicylates, methanol, ethylene, glycol, Li, ASA)
 - O - intractable fluid overload
 - U - symptomatic uremia (nausea/vomiting, seizure, pericarditis, bleeding)
- Good to prepare for dialysis early... far too often patients crash-land with a complication of ESRF requiring emergent dialysis via temporary vascular access, which has higher complications
 - If patient is approaching ESRF and refusing dialysis → explore why
 - At times in a very elderly patient, poor comorbid, declining dialysis is very reasonable → do advanced care plan (do you still want vascath if you crash?)
- Discuss options: hemodialysis vs peritoneal dialysis (continuous ambulatory PD or automated PD)
 - Lifestyle and patient factors: PD good for highly motivated, more educated patients (need aseptic technique), or patients with competent caregiver; HD good for patients who require help.
 - Consider comorbid: HD is challenging for patients who cannot tolerate large fluid shifts (e.g. CCF), PD contraindicated in patients with previous abdominal surgery
 - Consider long-term outcomes: PD preserves residual urine function longer than HD; starting with PD preserves vascular access for future HD.
- If HD, need to create AVF → can take 3-6 months to mature

(3) ESRF already on dialysis

- Explore dialysis route (HD vs PD) and regime (thrice-weekly HD, PD no. of changes)
 - Is patient coping?
 - Has patient been missing dialysis?
 - If on perm cath → patient should not be on perm cath. Why? Any plans for HD?
- Monitor adequacy of dialysis
 - Clinical: dialysis to dry weight (if not at dry weight post-dialysis, may be inadequate dialysis) → ask about dry weight
 - Biochemical: (1) solute clearance of PO₄, K, acidosis, (2) fluid clearance and blood pressure, (3) Kt/V measurement >1.7 (measures urea clearance)
- Complications of dialysis -- see later.

(4) Is this patient a transplant candidate?

- Explore if the question of transplant has been raised (should be unless patient has bad comorbidities or is old)
 - Are any living related donors available?
 - Is patient on deceased donor waitlist?

What complications of renal failure are there and how are these managed?

Severity of complications increases as GFR falls

- How often has the patient been admitted for complications or required emergent dialysis?
 - How severe: ICU, intubation?
 - Why? AoCKD (infection, cardiac event), non-compliance to diet restrictions, missed dialysis

(1) Complications of ESRF

- Anaemia: normocytic normochromic anemia secondary to decreased erythropoietin production → any symptoms, what is the latest Hb?
 - Target Hb 10.5-12
 - Rule out GI bleed, myeloma (anaemia, CKD, elderly)
 - First ensure iron replete (ferritin >500, transferrin saturation >30%) → Give PO iron, IV iron e.g. ferrinjet
 - Then top up erythropoietin (recormon)
- Blood pressure → what is the baseline?
 - CKD: ACE-I or ARB (all patients should be on), diuretics
 - ESRF: first titrate dry weight to achieve BP, then add antihypertensives.

- Calcium / Vitamin D / bone disease: Initially phosphate retention & hypocalcemia from vitamin D deficiency causes secondary hyperparathyroidism and mineral bone disease → Measure Ca / PO₄ / PTH; any fractures? Proceed stepwise -
 - Diet control: phosphate restriction
 - Phosphate binders: usually calcium-based unless hypercalcemia (then give lanthanum or sevelamer)
 - Only when phosphate controlled - activated vitamin D supplementation (calcitriol)
 - If hypercalcemic - patient may have developed tertiary hyperparathyroidism and hypercalcemia. Think about cinacalcet, parathyroidectomy.

- Electrolytes: hyperK, acidosis → how many admissions?
 - Diet control: low potassium diet → usually works, is patient compliant?
 - Consider bicarbonate supplementation (but with caution as it comes with Na)

- Fluid overload: dyspnoea, pedal edema → assess clinically. How many admissions?
 - Fluid restriction → usually works, is patient compliant?
 - Diuresis if not ESRF.

- Others: uremia and constitutional symptoms → are there any?
 - Immunosuppression: Malignancy screening, vaccinations
 - Anorexia, malnutrition: monitor nutrition status, albumin
 - Pruritus: usually a late issue.

Are there complications from dialysis?

- How often missed dialysis?
- Symptoms and coping with dialysis:
 - HD: end dialysis symptoms e.g. hypotension, giddiness
 - PD: bloating
 - HD: high output cardiac failure
- HD access issues: managing the vascular access is critical as this is the patient's lifeline.
 - On AVF/AVG: any issues with graft stenosis / inability to dialysis etc?
 - On perm cath: why? Any plans for AVF or AVG?
 - Has the patient required new graft creation or perm cath?
 - Does the patient have remaining access options left?
- Infection issues:
 - HD: line sepsis
 - PD: peritonitis → explore aseptic technique, ddx surgical peritonitis

Managing comorbidities

- Cardiac comorbidities important to manage -- these patients are vasculopaths and cardiac is the top cause of death in ESRF
- Remember to adjust medication dosing
 - E.g. diabetes - Stop metformin if CrCl <30, adjust insulin (decreased clearance leads to hypoglycaemias)
 - If ESRF no concern about nephrotoxicity; if CKD not on dialysis be very concerned.

How is the patient coping?

- Dialysis patients are always grumpy for a good reason - it takes a large toll on them
 - Explore compliance with dialysis, diet and fluid restrictions
 - Explore emotional / psychological issues → any depression? → Mirtazapine?
 - What is the patient's function?
- Financial aspects: dialysis is expensive, any subsidy (e.g. NKF?)
- Social aspects: is patient able to maintain any sort of lifestyle between shutting from dialysis centres to hospital?

Sample summary: Mdm Sian is a 66 year old lady with end-stage renal failure on hemodialysis secondary to longstanding diabetes. She has had recurrent admissions for thrombosed AVF and is currently dialysing via a permanent catheter, while awaiting for new left brachiobasilic AVF to mature. She has had one episode of MRSA line sepsis requiring permanent catheter change. Apart from ESRF, my outstanding issues for her include...

1. Access problems: currently on PC dialysis awaiting AVF maturity
2. Anaemia: I note she is pale and has exertional dyspnoea, would like to know her Hb, rule out GI blood loss and myeloma
3. Episodes of fluid overload secondary to noncompliance to fluid restriction
4. Comorbidities: insulin-dependant diabetes with excessively tight control (HbA1c below 6%, hypoglycaemic episodes)
5. Depressive symptoms due to burden of chronic disease
6. Financial problems: she has depleted her medisave and savings.

Renal: Nephrotic Syndrome

ADULTS & PAEDIATRICS

Do I have a diagnosis of nephrotic syndrome?

- Nephrotic syndrome is the cluster of urinary protein loss (urine total protein >3g/day), causing hypoalbuminaemia (<25g/L) and generalized edema.
- This contrasts with nephritic syndrome which presents with hematuria, hypertension, and varying degrees of renal compromise (rise in creatinine).
- Explore how the patient first presented - often leg swelling, shortness of breath,

What is the etiology of this patient's nephrotic syndrome?

- Etiology includes primary glomerulopathies of various histologies, as well as secondary causes.
- Explore possible secondary causes on history and subsequently investigation. These include autoimmune disease like lupus or HSP (joint pain, rash, dsDNA, ANA, complements), infection (HBsAg, HCV IgM, HIV), and diabetes.
- The primary glomerulopathies are distinguished on renal biopsy. Was this done? If so, why?
 - The overwhelming majority of paediatric nephrotic syndrome is due to minimal change disease and hence a biopsy is typically not performed. A biopsy would have been done if there were atypical features (age <1 or >10), nephritic features (hypertension, hematuria), poor response to steroid, suspicion of secondary cause (e.g. joint pain, low complements), worsening renal function or family history of renal failure.
 - Biopsy is standard in adult nephrotic syndrome - may show minimal change disease, FSGS, membranoproliferative and membranous GN. Each has specific disease associations (e.g. membranous with malignancy), and conditions like lupus can present as any pattern.

How treatment-responsive is this patient's nephrotic syndrome?

- ***Ask for nephrotic diary!***
- How frequently does the patient relapse? > In remission, infrequently relapsing (<2 in 6/12), frequently relapsing (>2 in 6/12).
 - Relapse = 3 consecutive days of dipstick at least ++ OR edema, hypoalbuminemia; Remission = 3 consecutive days of dipstick + or less
 - Frequent relapse --- is it because of noncompliance to medication, diet, or failure of treatment?
- Is there residual proteinuria/edema? > Give ACE-I to all.

- How well does the patient respond to therapy? Is he steroid-responsive, dependant (relapse within 2 weeks of stopping steroids or at least 2 relapses while on steroids), or resistant (failure to achieve remission in spite at least 6-8 weeks of high dose steroids).
 - If steroid-dependant, try steroid-sparing agents --- e.g. levamisole, azathioprine, cyclophosphamide (SE infertility), cyclosporine. Have they worked?
 - If steroid-resistant, have the other immunosuppressants worked?

What complications are there?

- Is renal function OK? (usually OK in nephrotic)
- Immunocompromised state: secondary to treatment (steroids → cushing's or immunosuppressants → agranulocytosis) and also as part of nephrotic syndrome (loss of immunoglobulins through urine)
 - Does patient know what to do if fever?
 - Vaccinate
- Hypercoagulability - venous thromboembolism > prophylaxis with aspirin or warfarin
- Hyperlipidemia and cardiovascular effects > give statin.

How is the patient coping with the medications and diet regimen?

- The nephrotic diet is unpleasant - salt and water is restricted, and due to the risk of cushings sugars and fats are also restricted. Many patients have difficulty with compliance which deserves lots of empathy!
- Similar with the medications -- if noncompliant, explore why? Due to poor understanding of disease / poor motivation / side effect / financial reason / lifestyle choice?

How is the patient's general health?

- Comorbids should be managed
- Paeds : growth and development is key.

Sample Summary

Mimi Yen is a 11 year old girl with steroid dependant nephrotic syndrome, diagnosed 2 years ago when she first presented with bilateral leg swelling and facial edema. She has had 3 relapses since diagnosis, the first within 1 week of tailing down her steroids and the other 2 even while she has been kept on steroids. Her primary physician is starting to bridge her over to tacrolimus as she is starting to develop features of cushing's syndrome. Otherwise, she has not had recurrent infections. Mrs Yen has, with much difficulty, kept Mimi on the recommended salt, water, sugar and fat restricted diet and ensured that she takes her medications daily. Mimi is below the 5th percentile in terms of height for her gender and is amongst the shortest in her class, but is otherwise doing well in the St. Hilda's gifted education program and able to participate fully in all activities. She understands that everything she is going through is for her own good, and has a group of best friends in her class who know about her condition and support her when the boys tease her about her chubby face.

Respi: Asthma

ADULTS & PAEDIATRICS

Is this asthma? What else could it be?

- Classic features: History of **variable** (over time and in intensity) respiratory symptoms (wheeze, shortness of breath, chest tightness, cough) often worse at night/on waking, responds to bronchodilators, usually has a trigger (refer below for usual culprits), personal history of atopy, family history of atopy
- Asthma variants: cough variant, exercise-induced variant, occupational variant
- Know the different differentials for each age group: a more comprehensive list is provided below but this requires tailoring to your patient (most important ones *)

Age	Differentials	Symptoms
0-5 y	Viral bronchiolitis also causes wheeze up to 3 years old and hence a diagnosis of asthma is often held off initially. However if the wheeze is recurrent, occurs even in the absence of URTI symptoms, or there is a strong family history of asthma or personal history of atopy, one may lean in favour of diagnosing asthma.	
6-11 y	Inhaled foreign body Bronchiectasis Primary ciliary dyskinesia Congenital heart disease Bronchopulmonary dysplasia Cystic fibrosis	Sudden onset dyspnea, unilateral wheeze Recurrent infection, prod cough Recurrent infection, prod cough, sinusitis Cardiac murmurs Preterm delivery, symptoms since birth Cough & mucus production ++, GI sympt
12-39 y	Vocal cord dysfunction Hyperventilation Bronchiectasis Cystic fibrosis Congenital heart disease Alpha1-antitrypsin deficiency Inhaled foreign body	Dyspnea, stridor Dizziness, paresthesia Recurrent infection, prod cough Cough & mucus production ++, GI sympt Cardiac murmurs SOB, family history of early emphysema Sudden onset, unilat wheeze
40+ y	COPD * Heart failure (cardiac asthma) * Interstitial lung disease * Lung cancer Vocal cord dysfunction Hyperventilation Bronchiectasis Medication related cough Pulmonary embolism Churg Strauss	Cough & sputum, SOBOE, <i>smoking</i> SOBOE, PND/orthopnea, JVP, LL swelling SOBOE, clubbing, non-prod cough Monophonic, cont wheeze, cachexic, hemopt Dyspnea, stridor Dizziness, paresthesia Recurrent infection, prod cough Hx of ACE inhibitor Sudden onset dyspnea, chest pain, LL swellin Eosinophilia, ENT disease, renal impairment

Did the patient undergo spirometry?

- Asthma is a clinical diagnosis but spirometry is a useful adjunct for diagnosis
- Classic spirometry finding is an obstructive picture with reduced FEV1/FVC (normally >0.75–0.80 in adults, >0.90 in children), with documented excessive variability in lung function, e.g.
 - If initial spirometry shows obstruction → do bronchodilator reversibility; should increase FEV1 >12% (withhold before test: SABA ≥4 hours, LABA ≥15 hours)
 - If initial spirometry is 'normal' → do methacholine challenge test, which should result in ≥20% fall in FEV1

Etiology: what precipitates asthma and have these been avoided?

- Is there an atopic setup? > Eczema, allergic rhinitis -- should be treated if present.
- Is there a family history?
- What precipitates asthma and has this been avoided?
 - Allergens: pets, dust mites
 - Occupational exposure -- are symptoms better on weekends?
 - Smoking: first hand and second hand --- stop smoking!
 - Environment: cold, haze, etc
 - Exercise
 - Infection: e.g. URTI
 - NSAIDs

How is recent asthma control?

- Course of disease: previous admissions, recent step up/down of therapy
- Are there features of high-risk asthma?
 - Past ICU admission, intubation, near-fatality.
 - Recent poor control (see below), severe exacerbations in last 12 months
- Asthma Control Test: all criteria met = controlled, 1-2 criteria not met = partially controlled, 3-4 criteria not met = uncontrolled
 - Daytime symptoms i.e. wheeze/cough/difficulty breathing (controlled = <2/week),
 - Nocturnal symptoms/awakening (controlled = 0)
 - Limitation of activities (controlled = 0)
 - Need for rescue/reliever treatment (controlled = <2/week)

If current exacerbation, what is the severity?

- Clinical: severity of SOB (on walking, on talking, at rest), ability to talk (in sentences, phrases, or words), ability to walk (yes, needed assistance, needed to be carried), mental state (alert, confused, or drowsy, ability to eat (too breathless?))
- On PE: SpO2, RR, PR, retractions, conscious level, speech, cyanosis, wheeze
- What has been given so far by GP or A&E? > Level of oxygen supplementation, how many puffs of rescue inhaler or nebulizations, and response to treatment
- Low threshold to do ABG; PCO2 should be low because of hyperventilation, if it is "normal" the patient is tiring out.

How best to manage the asthma?

- Acutely -
 - Ventilatory support: stepup from nasal prongs → face mask → intubate
 - Bronchodilation: inhalers vs nebulizer (1:2:1 ventolin:ipratropium:saline)
 - IV hydrocortisone +/- magnesium if severe
 - +/- antibiotics if clear infection
- Chronically - what is current regimen
 - What is current regimen (prescribed vs actually taken) -- frequent reliever use without preventer use is worrisome
 - How is compliance to treatment > If poor, why, what are the issues? (Medical / financial / social / psychological, perceptions of steroids)
 - Is inhaler technique acceptable
- Is there a need to step up or step down therapy -- not controlled = step up
 - Step 1: SABA PRN
 - Step 2: SABA PRN + low dose ICS
 - Step 3: SABA PRN + low dose ICS + LABA
 - Step 4: SABA PRN + medium/high dose ICS + LABA
 - Step 5: SABA PRN + medium/high dose ICS + LABA + low dose PO CS
- Give a written asthma action plan
- Assess and treat co-morbidities: rhinitis, eczema, sinusitis, GERD, obesity, OSA, depression, anxiety
- Treat precipitants: stop smoking, avoid allergens (see prior).

Any complications from treatment?

- Cushing's from long term corticosteroids

How is the overall coping of the child / young adult?

- Growth and development
- Is he/she able to participate fully in school?
 - What sports/hobbies/interests does the child have, at school and in their spare time? How does the child's level of activity compare with their peers or siblings?
- Social setup
- Financial

Note: The clinical questions above are more suitable for a chronic follow-up consult. In an acute setting, consider instead

- *Is this really asthma? (rule out other dDx) clinical features of asthma + spirometry*
- *Assess the severity of the asthma exacerbation - impending resp arrest? disposition?*
- *Look for etiology of exacerbation - what was the trigger?*
- *Assess underlying control of asthma - and decide if there is a need to step up*

Sample Summary 1: Little Sniffy is a 3 year old Chinese Boy with a strong family history of asthma and personal history of atopic dermatitis. He had two episodes of wheeze last year, associated with fever and rhinorrhoea, which were diagnosed as viral (RSV) bronchiolitis. In the past 2 months, however, he has had three episodes of wheeze, two of which occurred without any fever or upper respiratory tract infection. I feel that I would label him as asthma and commence treatment. I also note that his father is a heavy smoker. Otherwise he is growing well and meeting all milestones. My issues are:

1. Likely asthma (vs bronchiolitis) given atopic setup, family history, and multiple episodes without viral symptoms -- in an older child I would do spirometry as an adjunct for diagnosis but it is difficult to in this 3-year old. I would like to commence treatment with SABA and low-dose ICS.
2. Allergic rhinitis -- I would like to give intranasal corticosteroids.
3. Smoke exposure at home

Sample Summary 2: Biggie Wheezie is a 23 year old Malay lady with high risk asthma. In the past year alone she was admitted for 4 exacerbations and required intubation once. In the past month she has had night cough and alternate day exacerbations. She uses her ventolin heavily but does not comply to ICS - I note also that her inhaler technique is poor. Financially she is unemployed and not always able to afford her medications. The issues I need to address for her are:

1. High-risk asthma, poorly controlled with poor compliance to ICS
2. Financial issues

Respi: Bronchiectasis

ADULTS & PAEDIATRICS

How did this patient present?

- Adults may present as a longstanding (months-years) hx of cough with purulent sputum production daily, after repeated lung infections over the years. A COPD-like picture (wheeze, exertional dyspnoea) is also found in bronchiectasis.
- Children may present as chronic cough +/- failure to thrive
- Diagnostic imaging would have confirmed
 - CXR: tram tracks, ring shadows
 - HRCT: signet ring, bronchiole do not taper to periphery, bronchial wall thickening ("traction bronchiectasis" seen in pulmonary fibrosis is not bronchiectasis).

What is the underlying etiology?

- The pathophysiology of bronchiectasis is a vicious cycle of infection → airway damage → impaired secretion clearance → more infection; various causes lung infections, airway damage, or impaired secretion clearance can lead to bronchiectasis.
- Young patient with bronchiectasis or recurrent infections:
 - Is there cystic fibrosis? → Do sweat chloride, CFTR gene
 - Is there ciliary dyskinesia? (Kartagener's) → Look for dextrocardia, infertility, consider ciliary motility studies
 - Immunodeficiency states → Quantify immunoglobulins
- Is there autoimmune disease?
 - RA, Sjogren's (LL bronchiectasis) → Ask about symptoms, kiv workup (see RA).
 - Inflammatory bowel disease → Ask about symptoms, kiv colonoscopy
 - Sarcoidosis (UL bronchiectasis)
- Is there obstructive lung disease?
 - Longstanding asthma with cough, poor response to inhalers: consider allergic bronchopulmonary aspergillosis (central bronchiectasis) → Do FBC (eosinophilia), IgE (high), aspergillus precipitins, sputum culture.
 - COPD like picture → consider testing for alpha 1 antitrypsin deficiency

- What were previous pulmonary infections?
 - TB scarring: UL bronchiectasis
 - Aspiration pneumonia: LL bronchiectasis → Ask about GERD, swallowing impairment.
 - Previous virulent pneumonia or non-tuberculous mycobacteria infection: focal bronchiectasis (ML in NTM)
- Very focal crepitations
 - Rule out mechanical obstruction (foreign body, tumor) which is amenable to resection → chest imaging
- On examination attempt again to localize crepitations.
 - Focal: mechanical obstruction (FB, tumor), local infection (pneumonia, TB)
 - Diffuse UL: radiation, sarcoidosis
 - Diffuse LL: aspiration, immunodeficiency, fibrosis, idiopathic
 - Diffuse ML: NTM, ciliary dysmotility (ask abt fertility)
 - Central: ABPA.
- First-line etiological workup includes: FBC, sputum cultures (bacterial, TB, fungal), immunoglobulin levels, sweat chloride, CT (also diagnostic) +/- bronchoscopy.

Is underlying etiology treatable?

- Unfortunately, often not.
- Treat: NTM, ABPA
- Control: GERD, rheumatic disease
- Surgery: focal bronchiectasis, tumors, FB.

How is this patient and how is he being managed?

* Prognostic factors

(1) Infection and management

- How many exacerbations in past year? *
 - Frequency: >3, bad
 - Severity: Admission *, ICU?
 - Colonizing bacteria: any pseudomonas*, non-tuberculous mycobacteria?
 - Management: what has been needed?
- Are secretion clearance (bronchial hygiene) strategies being used?
 - Modalities: Chest physiotherapy (postural drainage, clapping), Devices (e.g. acapella), nebulized agents (hypertonic saline, mannitol, mucolytics)
 - Many are tedious → Explore compliance
- Is there a role for antibiotic suppression therapy? (e.g. ≥2 exacerbations a year)
 - Macrolides are both anti-infective and anti-inflammatory
 - Inhaled antibiotics especially for cystic fibrosis bronchiectasis
- Immunize patient.

(2) Baseline symptoms & management

- Cough & sputum production → secretion clearance
- Wheeze and exertional dyspnoea *
 - What is latest lung function test: FEV1 *
 - Bronchodilators
 - Stop smoking.
- What is the functional status?
 - Pulmonary rehab
 - Exercise
 - Nutrition, BMI *

(3) Haemoptysis

- Any episodes? Usually due to infection eroding bronchial arterioles.
- How were they managed

How is the patient coping?

- Able to work?
- Social and lifestyle burden of frequent sputum production, cough, need to use secretion clearance
- Psychological and emotional.
- Financial

Respi: COPD

ADULTS

Is the diagnosis COPD?

- Presentation: progressive exertional dyspnoea & chronic cough in a patient with significant smoke exposure, +/- intermittent exacerbations of SOB and wheeze
- Consider ddx:
 - Asthma → non-smoker, young pt, atopic hx (allergic rhinitis, eczema), exacerbation with allergen exposure, asymptomatic between exacerbations.
 - Asthma-COPD overlap: Asthma and COPD are a continuum -- these patients are considered to have Asthma-COPD overlap: (1) the 'asthmatic' with incompletely reversible airway obstruction, (2) the 'COPD' with reactive airways and exacerbations on allergen exposure, (3) the 'asthmatic' with chronic productive cough ('asthmatic bronchitis').
 - Bronchiectasis → many similarities with COPD and may occur concurrently. Suspect if: cough with daily sputum production is the main complaint (rather than exertional dyspnoea), crepitations, clubbing. Many similarities with COPD and may occur concurrently.
 - Heart failure
 - Pulmonary fibrosis
- Has COPD been confirmed on spirometry?
 - Expected obstructive picture, FEV1/FVC <70%, bronchodilator response <12%
 - FEV1 is prognostically important (determines 'risk')

What is the COPD stage?

- Concept from GOLD - *COPD can be staged in the intersection of symptoms and risk (of exacerbations, and severity of airflow limitation)*. Summary below, details later –

Higher risk FEV1 < 50% predicted, or ≥2 exacerbations / year	Gold C ICS + LABA	Gold D ICS + LABA +/- LAMA
Low risk FEV1 > 50% predicted, and 0-1 exacerbations / year	Gold A SABA or SAMA prn	Gold B LABA or LAMA
	Less Symptoms mMRC ≤2, CAT <10	More Symptoms mMRC 2+, CAT ≥10

SABA: short acting beta agonist; SAMA: short acting muscarinic antagonist (anticholinergic); LABA: long acting beta agonist; LAMA: long acting muscarinic antagonist (anticholinergic)

What is the risk / hx of exacerbations; and how is this mitigated?

- How frequent?
- How severe?
 - Requiring NIV / intubation?
 - Clinical course with usual mx: PO prednisolone, nebulized SABA, Abx
- What is the precipitating factor?
 - Usually infections → which, CAP or HCAP?
 - At times other complications of COPD e.g. pneumothorax
 - Also comorbid e.g. heart failure
- Prevention of future exacerbations:
 - Inhaled corticosteroids if high risk (GOLD C-D): reduce exacerbations, symptoms → How compliant?
 - Vaccinations: influenza, pneumococcal
 - Some patients may be on prophylactic Abx: evidence conflicting
 - Measures as below

What are the baseline symptoms and how are these managed?

Assessment:

- What is the functional limitation: e.g. walking speed limited by SOB, having to stop because of SOB when walking on level ground → qualifies as 'more symptoms'
- What is the baseline SpO₂?

Management: pharmacological and nonpharmacological, in stepwise approach.

- Stop smoking
 - If still smoking, why? Explore motivation and offer cessation therapy
- Mainstay is bronchodilators: beta-agonists or anticholinergics, short or long acting.
 - Evidence based to improve symptoms, reduce risk → if not taking, why?
 - GOLD A: SABA (e.g. albuterol) and/or SAMA (ipratropium)
 - GOLD B-D (inadequate symptom control or high risk): LABA (salmeterol, formoterol, indacaterol, vilanterol, olodaterol) or LAMA (tiotropium, aclidinium, umeclidinium, glycopyrronium). Dual bronchodilation if monotherapy inadequate to control symptoms.
 - Assess inhaler technique
- ICS should be added in GOLD C-D (see above)
- Has gone for pulmonary rehab? → improve functional capacity and QoL.
- Does the patient need LTOT? → evidence based, indicated if SpO₂ baseline <88%
 - Machine is not portable, patient will have to be home to use it
- Role of surgery
 - Bullectomy
 - Lung volume reduction surgery
 - Transplant.

Are there any complications?

- Pneumothorax
- CA lung
 - What is the FHx?
 - Watch for red flags → weight loss, clubbing, chronic cough (concomitant bronchiectasis), haemoptysis (may be as subtle as blood-tinged sputum)
 - Annual chest CT screening decreases mortality in patients with ≥ 30 pack-year history of smoking (including those who quit in the last 15 years).
- Cor Pulmonale → any raised JVP, loud P2, parasternal heave?
- Hypoxaemia
- Polycythaemia

Comorbids and general health

- Address comorbids
 - Especially cardiac → coexistence of CCF/COPD results in difficulty distinguishing the cause of exacerbations. Selective beta-1 blockers are safe.
- Ability to work
- Social setup → 2nd hand smoke?
- Psychological and emotional overtones: anxiety vs disregard.
- Financial situation

Sample summary: Chimney Chuan is a 65 year old Chinese ex-cook and smoker of 40 pack years, currently admitted for yet another infective exacerbation of chronic obstructive pulmonary disease. In the 9 years since diagnosis, he has progressed from GOLD Class A to D: his exacerbations have been increasing in frequency (once a year to 2-3 times a year) and severity (most recent one required intubation and ICU), and he has had progressive functional limitation from his symptoms (walking and wok handling limited by SOB) causing him to resign as a cook 3 years ago. Clinically he has signs of cor pulmonale but has refused all catheter angiographies. Otherwise he has never been complicated by pneumothorax, last screening CT did not show any suspicious lesions or bronchiectatic changes. He has a rather cavalier attitude towards his condition - he still smokes about 1 pack a day, tends to only take his bronchodilators when he needs it and does not bother with the ICS. This admission, his baseline SpO₂ was found to be 86% even after resolution of the infection, and has been counselled for LTOT. He is reluctant for it as he is tight financially and does not like the idea of having to be at home most of the time to use it. He is eager to go home so he can smoke without having to sneak out to a stairwell. My issues for him are:

- (1) Infective exacerbation of COPD (resolved)
- (2) COPD GOLD Class D
- (3) Baseline SpO₂ 86% - KIV LTOT
- (4) Ongoing smoking 1 pack a day

Rheum: Gout

ADULTS

Is this gout?

- Acute gout classically presents as a painful, warm, swollen, erythematous joint. Onset is usually at night, and severity peaks in 12-24 hours. Gout is often monoarticular (favouring lower limbs esp. 1st MTPJ - podagra), but can also be polyarticular.
- Natural history of gout: acute gouty arthritis → intercritical/interval gout → chronic tophaceous gout.
- Important differentials:
 - For acute gouty arthritis: Septic arthritis, trauma, pseudogout
 - For chronic tophaceous gout: rheumatoid arthritis, dactylitis, osteomyelitis
- Confirm diagnosis during an acute flare:
 - Joint aspirate: negatively birefringent needle shaped crystals on polarised light microscopy
 - XR of affected joint: “punched-out” erosions with sclerotic margins in a marginal and juxta-articular distribution, with overhanging edges
 - Blood: nonspecific inflammatory picture - leukocytosis, raised CRP, ESR
 - Serum urate levels are hard to interpret during acute gout flare, wait until at least 2 weeks after flare completely subsides to get baseline value

Any secondary causes of hyperuricemia?

- Is the patient on any uricosuric drugs (e.g. thiazide)? → Take patient off them!
- Hematological malignancy with high cell turnover or s/p initiation of chemotherapy ?tumour lysis syndrome

What is the course of the disease; any complications?

- Acute flares: are they increasing in frequency, severity and duration?
 - What are the usual triggers? High meat and seafood intake?
 - Which joints are involved?
- Intercritical symptoms
 - Is there pain between flares (called intercritical segments)
 - Has the patient developed tophi?
- Complications:
 - Is there joint deformity?
 - Urolithiasis → Chronic kidney disease
 - Infected tophi
 - Superimposed septic arthritis

How have acute gouty attacks been managed?

- Monotherapy with any one: NSAID, colchicine, systemic corticosteroid
- If inadequate response, switch to alternate monotherapy, or add combination therapy
- Does patient know how to initiate self-treatment of gout flares?
 - Try to take colchicine or NSAID *at the onset of flare*, before the flare peaks in severity → more effective
 - For colchicine: load 1.0mg, 0.5mg 1 hour later, 0.5mg 12 hours later

Chronic management**(1) Lifestyle measures and treating comorbidities**

- Treat metabolic syndrome: DM, HTN, HLD
- Lose weight
- Diet control: no alcohol, decrease red meat and fish, avoid fructose-enhanced sweetened drinks, reduce fat.

(2) If not on urate lowering treatment: When to initiate?

- All patients: low purine diet
- Indications:
 - Any tophus/tophi on clinical exam or on imaging
 - Frequent attacks of acute gouty arthritis (2 or more in a year)
 - CKD Stage 2 or more
 - Urolithiasis
 - Going for chemotherapy which may precipitate tumor lysis.
- When to initiate: usually wait 2 weeks after acute flare, because urate lowering therapy can precipitate gout flare (controversial - now disputed)
- What to initiate:
 - 1st line: xanthine oxidase inhibitors (allopurinol or febuxostat) → counsel on allopurinol risks especially SJS (see counselling)
 - Alternative: uricosurics e.g. probenecid (ensure GFR ok)
 - Treat to individualised serum urate target: <0.36mmol/L usually, <0.3mmol/L if tophi present
- Give concomitant colchicine for acute gout prophylaxis
 - [Tophi absent] 3 months after achieving target urate level
 - [Tophi present] 6 months after achieving target urate level + resolution of tophi

(3) If already on treatment: how has the patient been coping?

- Any side effects from the medication?
 - Allopurinol: rash, GI symptoms, fever, allopurinol hypersensitivity syndrome
- How has compliance been to medications and to diet?
- How is patient responding to treatment?
 - Is pain under control? Frequency of attacks decreased?

- What is patient's understanding of his disease and treatment?
 - Does he understand the importance of compliance to medications and low purine diet?
 - Does he know how to recognize adverse reactions to his medications?
 - Does he know NOT to stop ULT during a gout flare?

How has this affected the patient?

- Function is extremely important here
 - Is it pain or deformity that is affecting the patient?
 - What is the patient's occupation, how has this affected his ability to work?
 - ADL and ambulatory status - during flare and in between
- Financial situation, social situation

Sample summary: Ah Pooi is a 50-year old taxi driver with tophaceous gout. He first presented two years ago with podagra. Since then he flares every 2-3 months, often triggered by a drinking party. He is on allopurinol and is moderately compliant, missing doses when he drives late, and he still has tophi. He understands his disease well and can self-initiate NSAIDs early in an acute flare - although I wonder if NSAIDs should be given as continuous prophylaxis until his tophi disappear. My main issues for him are:

1. Chronic tophaceous gout
2. Metabolic syndrome: DM, hypertension, obesity.

APPENDIX: how to counsel on allopurinol

Take note because allopurinol has been the cause of Stevens-Johnson-Syndrom and implicated in multiple lawsuits.

Hi sir, I have been asked to talk to you about this drug called allopurinol. May I just check with you what you know about your condition, or about this?

I will go through with you (1) what is your current condition, and why allopurinol is indicated (2) what is allopurinol and how to take it (3) what are the risks and side effects of allopurinol and (4) alternatives. Please stop me anytime along the way if you have questions!

(1) Current condition, indication

You have a condition called gout, which is where you have too much uric acid in your blood and it forms crystals around your joint. This can cause irritation and inflammation, therefore the redness pain and swelling. This has become serious enough that you are getting it 2 or more times a year/forming tophi i.e collections in the skin/damage to the kidneys/urate stones in the kidney, therefore we recommend to start treatment.

(2) What is allopurinol, how to take it

Allopurinol is a drug that will help lower the uric acid levels in your body. It is taken as an oral tablet once a day, after meals.

(3) Side effects

- A very bad skin reaction (Stevens-Johnson syndrome/toxic epidermal necrolysis) may happen. Get medical help right away if you have signs like red, swollen, blistered, or peeling skin (with or without fever); red or irritated eyes; or sores in your mouth, throat, nose, or eyes.
- Signs of an allergic reaction, like rash; hives; itching; red, swollen, blistered, or peeling skin with or without fever; wheezing; tightness in the chest or throat; trouble breathing or talking; unusual hoarseness; or swelling of the mouth, face, lips, tongue, or throat. Get medical help right away.
- Allopurinol can ironically cause gout flares in the initial few months
- Fever or chills, sore throat.
- Nausea, vomiting, diarrhea

(4) Alternatives

- There are other drugs like febuxostat and probenecid
- You can also choose not to start therapy at all

Rheum: Henoch-Schonlein Purpura

PAEDIATRICS

Do I have a diagnosis of Henoch Schonlein Purpura? What else could it be?

- Palpable purpura (usually lower limb) without thrombocytopenia or coagulopathy (do FBC, ESR, PT/PTT) is mandatory. In addition there is ≥ 1 of:
 - Abdo pain: diffuse, colicky, acute-onset (can lag behind rash by 1 week) -- if significant think of intussusception.
 - Arthritis or arthralgia: acute-onset oligoarthritis favouring LL; may be swollen, tender, ROM limited, but not warm or erythematous; may refuse to ambulate.
 - Renal involvement: hematuria +/- red blood cell casts, no or mild proteinuria
- May be preceded by URTI; significant proportion of cases of HSP are triggered by streptococcal infections → do antistreptolysin O titres
- If incomplete presentation, esp if purpura is absent -
 - Biopsy kidney (in glomerulonephritis) or skin (vasculitis) -- there is IgA deposition in HSP. Biopsy is not routine, reserved for unusual presentations (ie, no rash, or an atypical rash) or significant renal disease
 - Consider ddx:

	Differentials	Workup
Purpura	Cutaneous small vessel vasculitis Systemic small vessel vasculitides (Wegener's, Churg-strauss, microscopic polyangitis) Connective tissue disease (SLE) Hep B/C	Plt, PT/PTT, skin biopsy ANCA ANA, dsDNA, C3, C4 HBsAg, HCV RNA
Arthralgia Arthritis	SLE, JIA Rheumatic fever Septic arthritis	ANA, dsDNA, C3, C4, RF Throat c/s, anti-streptolysin O KIV joint aspiration
Abdo pain	Appendicitis, gastroenteritis, etc. (see abdo pain approach)	
Nephritis	IgA nephropathy (common pathogen)	Renal biopsy

What are the organ-based complications and management tasks?

(1) Overall management

- 90% of HSP occur in paediatrics and are self limiting.
- Most can be treated outpatient with oral hydration, bed rest, and symptomatic relief of joint and abdominal pain; admit if child is sick (cannot take orally, severe abdo or joint pain limiting ambulation, altered mental status), complications (significant GI bleed, significant renal disease)
- Manage joint/abdo pain with NSAIDS such as naproxen with adequate hydration
- Systemic glucocorticoids (PO prednisolone or IV methylprednisolone) if severe abdominal pain that interferes with oral intake and non-response to NSAIDs; remember to tail dose.

(2) Renal disease

- Monitor UFEME & Cr → ⅓ of HSP develop renal involvement which may be range from mild to acute nephritic syndrome (hematuria, hypertension) or renal impairment.
- Some clinicians treat with steroids -- controversial. Glucocorticoids and other steroid sparing agents not shown to be beneficial unless renal biopsy shows true crescentic glomerulonephritis.

(3) Abdominal pain +/- complications

- Abdominal pain is a feature of HSP but beware of complications → more commonly intussusception (severe colic, redcurrent jelly stools), GI bleed, pancreatitis, gallbladder involvement, bowel perforation
- If suspect intussusception order ultrasound; treatment is first by attempting radiographic- guided air enema; if fails, open surgery.

(4) Other complications (less common)

- Neurology: headaches, seizures, encephalopathy (both hypertensive encephalopathy and posterior reversible encephalopathy syndrome [PRES]), focal neurologic deficits, ataxia, intracerebral hemorrhage, and central and peripheral neuropathy
- Scrotum: rarely, boys can also present with scrotal pain mimics testicular torsion

Long-term follow up

- Prognosis is excellent although the rash may take time to resolve and a small percentage (<1%) develop long term complications, mainly renal disease.
- Outpatient follow-up: screen BP and for urinary abnormalities to identify patients with significant and potentially progressive renal involvement.

Sample summary: Pokey is a 6 year old boy who first presented with a 2-week history of palpable purpura, mild abdominal pain, and hematuria. He was diagnosed with henoch schonlein purpura and treated outpatient with NSAID analgesia and PO steroids. Subsequently he developed severe abdominal pain and redcurrent jelly stools; he was admitted and an ultrasound diagnosed intussusception. Treatment with air enema was successfully. That episode was four weeks ago and at his latest follow up last week he was well, feeding well, with no residual abdominal pain.

Rheum: Juvenile Idiopathic Arthritis

PAEDIATRICS

What is the clinical presentation > Is it JIA and if so, which subtype?

(a) Pauciarticular JIA: chronic oligoarticular joint pain

- Look at the time course - JIA lasts >6 weeks. If acute, consider septic arthritis, haemarthrosis, and trauma -- do joint aspirate
- Classically, inflammatory pain in ≤ 4 , usually large joints, o/e swollen and tender. But may not present as "pain", simply as limp, clumsiness, refusal to play

(b) Polyarticular JIA: chronic polyarticular joint pain

- Look at the time course - JIA lasts >6 weeks. If acute, consider septic arthritis, viral arthritis, henoch schonlein purpura, haemarthrosis, and trauma
- Classically, inflammatory symmetrical polyarthropathy in ≥ 5 joints (may be preceded by indolent pain in 1-2 joints esp in younger children <10y). Parallels RA.
- ANA is +ve in younger children <10y, RF +ve in older. Lupus-related antibodies (dsDNA, etc) should be -ve.

(c) Enthesitis-related arthritis

- Somewhat hard to classify and psoriatic arthritis seems separate?
- Features include spinal pain, HLA-B27 positivity, inflammatory bowel disease; analogous to spondyloarthropathy in adult.

(d) Systemic onset JIA: sick child with joint pain

- Classically recurrent spiking fever, salmon-coloured rash, hepatosplenomegaly +/- pleuritis/pericarditis; onset of arthritis may be delayed.
- Laboratory findings are: raised inflammatory markers (neutrophils, thrombocytosis, ESR, CRP, ferritin), anaemia. RF and ANA usually negative.
- Ensure that malignancy is excluded: 'normal WBC', lymphocytosis, neutropenia, thrombocytopenia, and disproportionate or night pain is worrisome.
- Other ddx: viral arthritis, malaria.

How has it been treated and is there disease remission?

- Treatment options: NSAIDs, hydroxychloroquine, methotrexate, biologics; bridging corticosteroids in acute setting, intra-articular corticosteroids for oligoarticular JIA.
- Is pain controlled?
- How is child's function?
- Is there any deformity? > e.g. contractures, loss of joints space.
- Are there side effects of treatment? E.g. if on MTX, does the child/parent know how to come to A&E for FBC if there is any fever? These drugs require monitoring.

How is the child's vision?

- Children with JIA are at risk for uveitis, especially if ANA +ve, pauciarticular JIA.
- Are there symptoms of visual loss? However uveitis can be asymptomatic until vision is lost.
- Require regular slit-lamp screening -- if not doing, why?

How is the child's overall health, and quality of life?

- Growth & Development
- Coping with school
- Family support.

Rheum: Rheumatoid Arthritis

ADULTS

Is this rheumatoid arthritis?

- Classic RA: symmetrical inflammatory polyarthropathy affecting the small joints of the hands (MCPJ and PIPJ, sparing the DIPJ), associated with morning stiffness lasting >30min.
 - EULAR/ACR 2010 Criteria: clinical (joints affected, symptom duration), serological (RF, anti-CCP), acute phase reactants (CRP, ESR)
 - Any inflammatory joint pain has to be taken seriously even if it is not a classic rheumatological condition → early specialist referral.
- Differentials
 - Psoriatic arthropathy → Look carefully for any psoriatic plaque, onycholysis and other nail changes.
 - Osteoarthritis of the hand → mainly DIPJ (Heberden node) rather than PIPJ (Bouchard node), pain is mechanical not inflammatory
 - Polyarticular gout
 - Overlap syndromes: look for features of SLE (rash, systemic organ involvement not classical for RA e.g. nephritis), scleroderma (sclerodactyly, facial telangiectasias, pulp atrophy and digital ulcers), do anti-RNP antibody

What has the course and current disease activity?

- Consider how the patient presented, the course of disease and response to treatment, and where the patient is currently.
 - RA is now aggressively treated with early diagnosis, specialist care, early DMARD use, and tight control, therefore it is rare to see new RA cases progress along its natural history to reach an end state with disabling deformities (the 'piano key hands').
 - However longstanding RA patients, or older patients who do not seek help early, may still present with late stage RA
- What is the current disease activity
 - Symptoms: pain, morning stiffness
 - Functional status (very important to assess)
 - Examination findings: boggiess of the joint, erythema and tenderness
 - Serological: trend the CRP, ESR
- What is the accumulated damage?
 - Deformities - e.g. subluxed MCPJ, Z thumb, etc
 - Function is also very relevant here.

What is the extent of involvement?

- Other joints
 - Larger appendicular joints: wrists, elbows, shoulders, ankles, knees, hip
 - Atlantoaxial instability → any neck pain?
 - Hallux valgus
- Extra-articular manifestations
 - Eyes: episcleritis, scleritis → any eye disease?
 - Skin: rheumatoid nodules, pyoderma gangrenosum
 - Vasculitis
 - Lungs: interstitial lung disease, bronchiectasis
 - Pericarditis
 - Haem: anemia, neutropaenia (felty's syndrome)
 - Nerves: carpal tunnel syndrome

How can I optimise management?

(1) Symptomatic relief

- NSAIDs, COX2 inhibitors
- Low dose prednisolone
- Is patient satisfied with functional state?

(2) Prevent disease progression

- Corticosteroids for induction of remission
- DMARDs: Methotrexate hydroxychloroquine, sulfasalazine, leflunomide
 - Baseline tests: CXR (pneumonitis, TB), FBC (cytopenias), LFT (hepatotoxicity), RP for Cr, HBsAg, Anti-HCV
 - Advocate for early DMARD use to prevent damage.
- Biologics: anti-B cell (rituximab), anti-TNF (etanercept, infliximab, adalimumab, etc.)
 - For patients with high disease activity despite DMARDs
 - Long term use can result in antibodies being formed against the drugs and thus loss of response

(3) Mitigate existing disability

- Orthopaedic intervention e.g.
 - Replace destroyed joints
 - Tendon transfers
 - Carpal tunnel syndrome management
- Functional assistance: e.g. orthotic aids, home modifications, occupational therapy

(4) Manage extra-articular manifestations

- Interstitial lung disease → try glucocorticoids, and if refractory, mycophenolate, azathioprine, or cyclophosphamide (avoid methotrexate)

(5) Manage complications of treatment

- Steroid side effects e.g. Cushingoid appearance, osteoporosis, cataracts: see approach to Cushing's disease → monitor fasting glucose, lipids, DEXA and treat osteoporosis
- Agranulocytosis on methotrexate
 - Monitoring
 - Give folic acid
 - Check - does patient know what to do if fever?
- Complications of immunosuppression
 - Are there recurrent infections?
 - Prophylaxis: vaccinate
- If young female on methotrexate - is the patient planning pregnancy? Methotrexate is teratogenic so if considering pregnancy need to switch; if not considering, check that contraception is adequate

How is the patient coping overall?

- Patient's understanding of disease -- what are the concerns and fears
- Functional: Able to work? Able to take care of self?
- Psychological: deformities can be disfiguring and pain debilitating.
- Social: especially if function not as good, what are the care arrangements?
- Financial

Sample summary: Mdm Sang Net is a 60 year old Chinese lady who has a 10-year history of inflammatory symmetrical polyarthropathy mainly affecting MCPJs, PIPJs, and bilateral knees, associated with morning stiffness. She was referred to rheumatology and diagnosed with rheumatoid arthritis but was lost to follow up before she could be started on treatment. She re-presented three years ago with hand deformities including MCPJ subluxation and ulnar deviation and swan neck deformities. She was started then on methotrexate and analgesia, which she is tolerating well. Currently her disease is quiescent with no complaints, minor functional impairment not causing impediment to lifestyle, therefore she is thinking of stopping treatment because she feels better. My main issues are -

1. Rheumatoid arthritis on methotrexate
2. Poor patient understanding of disease → need education.

Rheum: SLE

ADULTS & PAEDIATRICS

Is this SLE?

- What is the presenting symptom? > *Dr House* has a running joke of offering lupus as a differential in almost every episode -- there is some truth to this, the clinical manifestations of SLE are protean.
- When to consider SLE: in the presence of suspicious clinical features,
 - Do autoantibodies: ANA, dsDNA, anti-Sm, anti-Ro, anti-La, antiphospholipid (anticoagulant, anticardiolipin, anti beta-2 microglobulin): beware false +ve ANA (other rheumatic dx, other autoimmune dx, chronic infection, normal pt)
 - Look for multiorgan involvement: FBC, UECr, CXR, as below.
- What else to consider: consider differentials to the presenting symptom (see approach to specific symptoms), e.g.
 - Other rheumatic disease: RA, mixed connective tissue dx (RNP +ve), vasculitides
- When to call it SLE?
 - Ultimately, a clinical diagnosis. Classification criteria (e.g. 2012 SLICC criteria) are meant for research use; they provide a guide but are insufficiency sensitive especially in early disease
 - Much more clear-cut if the clinical course is long

What are the manifestations in this patient?

Necessary to do a thorough systematic review and comprehensive physical examination to look at organ specific manifestations

- Skin: malar rash, discoid rash, alopecia → any rash
- Face: dry eyes/mouth (2' sjogren's), red eyes (uveitis), oral ulcers
- Cardiac: any cardiac issues (accelerated atherosclerosis)
- Pulmonary: pleuritis, interstitial lung disease → any cough / chest pain / SOB?
- Renal: nephritic or nephrotic syndrome, CKD → any frothy urine, pedal edema, hematuria, hypertension?
- Haematological: anaemia, thrombocytopaenia
- Antiphospholipid syndrome: DVT, recurrent obstetrical loss → Any previous children? Any painful leg swelling?
- Musculoskeletal: joint pain or swelling
- Neurological: stroke (vasculitis), seizures, psychiatric issues → Any weakness / numbness / difficulty seeing / headache
- Constitutional features: fever, loss of weight, fatigue.

What are the current management issues?**(1) Disease activity and flares**

- What is the tempo of flares - and how serious?
 - Each patient usually has a stereotypical flare -- but note ddx: infection
 - Evidence of flare: complements fall, dsDNA rises. [but CRP normal]
- How were they managed?
 - Major flare: pulse methylpred, cyclophosphamide, mycophenolate, cyclosporin
 - Minor: oral prednisolone, azathioprine, hydroxychloroquine
- Any other evidence of disease activity e.g. active nephritis, difficult to manage cytopenias
 - Monitor C3, dsDNA Ab, CRP
- Any other symptoms → manage them
 - E.g. arthralgias → NSAIDs if no renal disease
 - Photosensitive skin manifestations: avoid sunlight, sunscreen

(2) Chronic immunosuppression

- What is the patient currently on and what is the response
 - Is response adequate? Any need to step up/down?
- Is the patient smoking? → Stop!
- Can we optimise treatment
 - All patients should be on hydroxychloroquine
 - If on steroids chronically → any role for steroid sparing agents e.g. azathioprine, mycophenolate, cyclosporin

(3) Complications of medication

- Immunosuppression: predisposition to infections
 - What is the hep B, C, HIV status; baseline CXR
 - Immunizations
- Steroid side effect: e.g. osteoporosis, avascular necrosis, cushingoid features (see approach to Cushing's)
- If on hydroxychloroquine: any retinopathy
- Methotrexate if joints involved

(4) End-organ damage and their management

- Monitor: e.g. UeCr, UPCR, UFEME, FBC, LFT, malignancy screen
- Renal failure → manage as per CKD (ACE-I), may need dialysis
- DVT/PE → may require anticoagulation.
- DM, hyperlipidemia → manage as per usual

How is the patient coping?

- In a disease with many manifestations (and further side effects), important to know exactly what is bothering the patient
- Sexual activity: does the patient intend to become pregnant?
 - If active disease, dangerous maternal and fetal outcomes (lupus flare, miscarriage, preeclampsia, neonatal heart block) → should control disease first for 6/12 before planned pregnancy
 - If active disease → what contraception is used?
- Function: able to work?
- Social: social interactions, family
- Financial

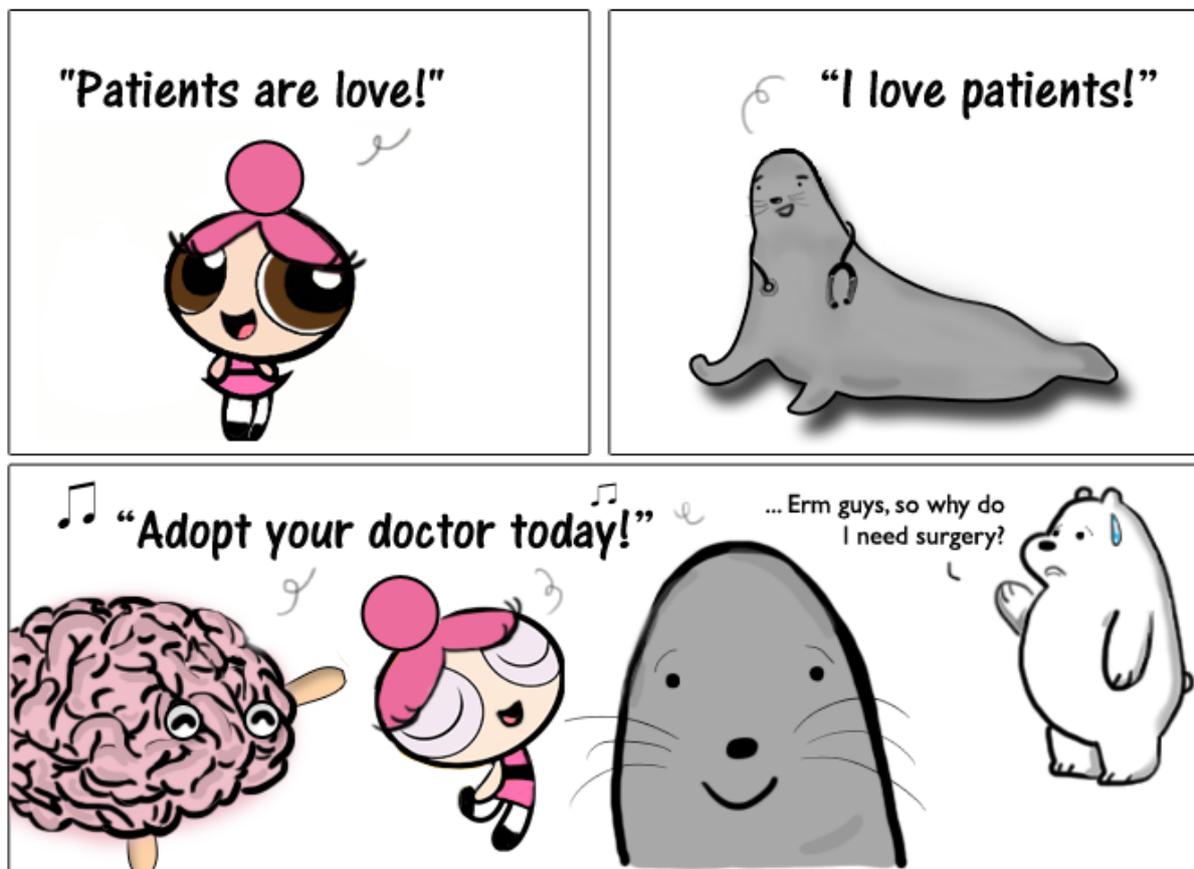
Sample summary: Ms Seow Ling Ee is a 30 year old Chinese teacher with systemic lupus erythematosus. She was diagnosed 5 years ago when she first presented with malar rash, small joint arthritis, lupus nephritis and hemolytic anemia. She has been stable on hydroxychloroquine and methotrexate, with only 2 flares with the same pattern of involvement which remitted with pulse steroids. Her kidney function, CKD stage 3 at the moment on ACE-Is, is being watched closely. She has not experienced any serious side effects from her medications such as vision problems or recurrent infections. As a patient, Ms Seow is highly motivated to comply to her treatment as she wants to avoid dialysis. She is also astute and has a good understanding of her disease, having been able to recognize her flare signature (fever, malaise, rash, joint pain, lower limb swelling, symptoms of anemia) and know to get admitted. She and her husband are still undecided as to whether they want to try for children - they do desire children, but do not want to rock the boat and risk further damage to Ms Seow's kidneys. Until then, they understand the importance of continuing contraception when she is on methotrexate. My main issues for her are:

- (1) SLE with lupus nephritis, hemolytic anemia and arthritis - currently in remission
- (2) CKD Stage 3 secondary to lupus nephritis
- (3) ?possibility of planned pregnancy

PREPARING FOR THE MBBS | CHAPTER FIVE

Communications & tasks

focus on the focused task



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Strategy

Introduction

Communication and task stations are an important part of both medicine and surgery tracks. They feature in about 3 of 8 surgical OSCE stations, and in all 5 medicine OSCE stations.

These stations come in several variants (below) but the majority combine communication skills (whether with colleagues or patients) with medical knowledge. As such, you will require both communication skill (e.g. SBAR handover, dealing with angry patient, breaking bad news, reinforcing compliance), plus specific content knowledge. The majority of scenarios should be realistic for a HO level, although you may get an oddball or two.

Scenarios can broadly be classified into three groups - acute management, communications, and procedural skills. A non-comprehensive list follows -

I. Acute management scenarios

These are urgent situations that you may encounter on call or in the course of ward work. The focus is usually on safety. Your task may include -

- Recognizing and managing an urgent situation (as appropriate for HO level)
- Delegating clear orders to a junior or nurse
- Escalating to a senior: telephone handovers using SBAR, blue letter referrals
- Requesting for a radiological investigation on the phone.

Examples of situations for which you may be asked to fulfill any one of the above tasks include:

- Common on-call complaints: chest pain, hypotension, desaturation, dyspnoea, electrolyte abnormalities
- Advanced Cardiac Life Support: collapse, tachycardias, bradycardias
- Post-operative or procedural-related complications
- Medication errors and complications
- Blood transfusion reaction
- Other emergencies: compartment syndrome
- Needlestick injury

II. Communication scenarios

These communication scenarios usually involve speaking to patients and/or family members about an issue. They may involve a combination of the following flavours (e.g. breaking bad news then counselling on the diagnosed condition)

(a) The Angry Patient or Relative

- Adverse events: medication errors, drug reaction, inpatient falls, misdiagnosis, complications
- Service quality (long wait, postponed operation, etc)

(b) Breaking Bad News

- Serious illness: cancer, HIV, diabetes, ESRF for dialysis initiation.
- Updating relatives: informing DDIL, coroner's case.
- Informing brain death / organ transplant (unlikely for MBBS, too difficult)

(c) Disease counselling (worried / noncompliant patient)

- This is a wide spectrum of patients - from the patient who is noncompliant or requesting for AOR discharge, to the excessively worried patient.
- Disease counselling on wide range of topics including -
 - Paediatric: asthma, gastroenteritis, neonatal jaundice, febrile fits, diabetes, immunization
 - Adult: e.g. HIV, vascular risk factors, cancer screening, TB
 - Medication: warfarin counselling, insulin injection technique, inhaler technique
- Counselling on implications of disease/medication on patient such as
 - Pregnancy safety
 - Fitness to drive: epilepsy, cardiac disease, visual disorders.
 - Fitness to fly
- Each disease may have specific points of content knowledge and concerns to address (see examples given)

(d) Consent taking and/or patient preparation

- For procedure e.g. OGD, colonoscopy, ERCP, lumbar puncture, central line insertion, blood transfusion.
- For radiological investigation e.g. contrast CT
- For operation
- These scenarios may require you to consent the patient, identify contraindications, know how to manage the patient peri-procedurally (e.g. bowel prep, stop metformin before scan).

(e) Ethical issues

- Assessment of capacity
- End of life issues: futility of treatment, palliative management, advanced medical directive
- Patient confidentiality issues: e.g. revealing diagnosis to family members, collusion, the pregnant maid.
- Genetics: inviting family members for testing, implications for future generations.
- Medical error or negligence (as part of angry patient scenario or as a task - see below)

III. Life in the ward

These seemingly routine tasks and incidents you may encounter in the ward may also be tested.

(a) Professionalism

- Patient confidentiality issues - identifying and rectifying
- Dishonest team member

(b) Team and interprofessional communication

- Reporting a safety issue or other lapse.
- Counselling a team member who made an error or professionalism lapse
- Dealing with conflicts between team members.

(c) Other clinical priorities

- Hand hygiene and infection control
- Patient safety, apart from acute situations; e.g. preventing inpatient falls

IV. Procedural skills

You may be asked to perform bedside procedures including blood cultures, catheterization, group cross match, insulin administration, IV drug administration, ECG, arterial blood gas, suturing, even handwashing. Advanced (certifiable) procedures such as CVP insertion, chest tube insertion, lumbar puncture are unlikely to come out. These are rather standard and samples are not provided.

How to prepare

Do your SIP work diligently. These scenarios are typically encountered in the course of HO work and should therefore not come as a surprise (seemingly 'routine' things can also be tested). You will pick up the skills best by seeing them 'live' in the wards - and by trying your hand at them too.

Have a look through the past questions. This gives you a flavour of how the stations are structured and the expectations.

Think of a strategy for each. Know thyself. Some of us are excellent communicators but fumble under pressure in an acute situation; others have good intentions but tend to be tactless and inspire anger in our patients. It is helpful to understand your strengths and weaknesses and work on what you need. There are specific skills to be picked up - e.g. how to break bad news using SPIKES, how to escalate using SBAR - which you should learn and practice.

Practice with each other. Get together as a bunch of friends or in a CG to organise an OSCE circuit. There is nothing like being put on the spot and seeing how you react. The majority of scenarios in this compilation arose from these practice circuits and have been written with candidate instructions, SP instructions, and either marking rubrics or a sample script, so that you may attempt these stations. They often involve multiple communication plus knowledge issues and are slightly more challenging than you should expect for the exam. If you intend to attempt these stations, it will be helpful to avoid reading through the samples beforehand. Alternatively, you can use them as a template to come up with your own stations.

SAMPLE: HOW TO ORGANIZE OSCE PRACTICE

Preparation

Team 1: Nigel, Marianne, Prince, Scott, Milo, Dimples. To prepare:

- 4 Short Cases: 1 Ortho (Hand, nerve lesion), 1 Ortho (Knee), 2 GS (Hernia & Arterial)
- 2 OSCE: 1 Acute Management focused task (Ortho), 1 Comms (GS)

Team 2: Blueberry, Sweetie, Brainy, Peace, Chirpy, Sunshine. To prepare:

- 4 Short Cases: 1 Ortho (Hand, non-nerve), 1 Ortho (Shoulder or Foot), 2 GS (Venous & Other)
- 2 OSCE: 1 Acute Management focused task (GS), 1 Comms (Ortho)

Actual day

Meet at 8 am at [someone's house]

It is helpful to get someone to time (one of you or a family member / sibling / maid).

0815 - 0915 Team 1 examiners, Team 2 students (do 6 stations in a row)

Round 1	Living	Dining	Bedroom 1	Bedroom 2	Bedroom 3	Kitchen
Examiner	Milo	Scott	Prince	Nigel	Marianne	Dimples
Station 1	Blueberry	Sweetie	Brainy	Peace	Chirpy	Sunshine
Station 2	Sunshine	Blueberry	Sweetie	Brainy	Peace	Chirpy
Station 3	Chirpy	Sunshine	Blueberry	Sweetie	Brainy	Peace
Station 4	Peace	Chirpy	Sunshine	Blueberry	Sweetie	Brainy
Station 5	Brainy	Peace	Chirpy	Sunshine	Blueberry	Sweetie
Station 6	Sweetie	Brainy	Peace	Chirpy	Sunshine	Blueberry

0920 - 1020 Team 2 examiners, Team 1 students (do 6 stations in a row)

Round 2	Living	Dining	Bedroom 1	Bedroom 2	Bedroom 3	Kitchen
Examiner	Chirpy	Sweetie	Peace	Sunshine	Brainy	Blueberry
Station 1	Milo	Scott	Dimples	Prince	Nigel	Marianne
Station 2	Marianne	Milo	Scott	Dimples	Prince	Nigel
Station 3	Nigel	Marianne	Milo	Scott	Dimples	Prince
Station 4	Prince	Nigel	Marianne	Milo	Scott	Dimples
Station 5	Dimples	Prince	Nigel	Marianne	Milo	Scott
Station 6	Scott	Dimples	Prince	Nigel	Marianne	Milo

1030 - 1230 Do your own team's other 5 cases (and do your case 5x for your other team members).

Round 3	Living	Dining	Kitchen	Bedroom 1	Bedroom 2	Bedroom 3
Session 1	Sweetie & Brainy	Blueberry & Sunshine	Peace & Chirpy	Nigel & Prince	Marianne & Scott	Dimples & Milo
Session 2	Sweetie & Peace	Brainy & Blueberry	Sunshine & Chirpy	Nigel & Dimples	Prince & Marianne	Scott & Milo
Session 3	Sweetie & Sunshine	Peace & Brainy	Blueberry & Chirpy	Nigel & Scott	Dimples & Prince	Marianne & Milo
Session 4	Sweetie & Blueberry	Sunshine & Peace	Brainy & Chirpy	Prince & Scott	Dimples & Marianne	Nigel & Milo
Session 5	Brainy & Sunshine	Peace & Blueberry	Sweetie & Chirpy	Nigel & Marianne	Scott & Dimples	Prince & Milo

1230-1330 Debrief

1330-1430 End with a nice lunch to reward yourself for the hard work!

CTSP: Abdominal Pain

contributions from David Ng

INSTRUCTION TO CANDIDATE

You are the surgical HO on-call. Your nurse calls you on the telephone to see Mr Ken Ser, a 75yo Chinese gentleman, for fever, abdominal pain, 1 episode of vomiting and low blood pressure.

He has a past medical history of gastric carcinoma, diabetes and hypertension. He underwent a gastrectomy 7 days ago to remove the tumour and has been well since the operation. He is not on nasogastric tube and just took his dinner 4 hours ago

Your task is to:

1. Talk to the nurse and tell her what you would like her to do
2. Outline your management plan

INSTRUCTION TO SP - NURSE

You are a reasonably competent staff nurse. You are quite worried about Mr Ken Ser, who was recovering well after his gastrectomy 7 days ago, and turned sick just after dinner, and decide to call the surgical HO. You have this information on hand

- Vitals: BP 86/50, HR 130, SpO2 98% RA, T 38.7
- Input/Output: Just escalated to normal diet today, took full share of dinner, input today 1800ml. Output today 1400 PU, but there has been no urine output in last 3h.

If the candidate requests, please provide the following information

- Case notes: gastric CA, locally invasive. Scheduled curative resection which was uneventful. Currently POD7 and has been well in last 7 days.
- Procedure notes: routine gastrectomy with roux-en-Y reconstruction, no significant issues.
- Current history / examination: patient is drowsy, c/o abdominal pain and vomiting. Abdomen is soft with no guarding or rebound.

Candidate should escalate to the senior (using SBAR), perform appropriate initial investigations, and arrange either for emergency OT or CT (and make the necessary call).

MARKING RUBRIC

		Performed and competent	Performed but NOT fully competent	Not performed or incompetent
1	Greets patient and introduces self	1	0.5	0
2	Asks nurse for vitals and patient status	1	0.5	0
3	See patient immediately	1	0.5	0
4	Request for procedure notes	1	0.5	0
5	Request for vitals & intake/output chart	1	0.5	0
6	Assess patient; brief history and examine abdomen	2	1	0
7	Informs senior with - Appropriate SBAR: patient is sick - Reasonable impression	2	1	0
8	Performs appropriate bloods e.g. FBC UECr CRP PT/PTT GXM c/s +/- LFT amylase	2	1	0
9	Performs erect chest X ray	2	1	0
10	Reasonable initial management especially IV antibiotics	2	1	0
11	Either: prepare for EOT or urgent CT	2	1	0
12	Call radiologist or EOT and appropriate request / SBAR	2	1	0
13	Able to list post-gastrectomy complications	2	1	0

Immediate failures

- Doesn't see patient immediately
- Does not escalate to senior

DISCUSSION

After a procedure, e.g. intra-abdominal surgery, a scope, a vascular intervention, or some other procedure like TACE/RFA, new complaints can develop.

Learning Point 1: Differentials & Blind Spots

- Closer to a procedure, our blind spot would probably be non-procedure related ddx.
- Further away from a procedure, procedure related complications may not cross our mind so readily.
- One approach is simply to be systematic: when patients present with a complaint, is this related to previous procedural, past medical hx, or something unrelated?

Possible differentials in this case

- Procedure related: anastomotic leak (most likely), intra-abdominal collection, afferent loop syndrome, bleeding at the surgical site
- Related to PMHx (HTN, DM): cholangitis, DKA
- New: e.g. obstructed inguinal hernia

Learning Point 2: always go over the op notes for any complications & instructions

- Post-surgery, anatomy is altered, and different altered anatomies can present with different post-op complications
- It helps to know if anything went wrong during the surgery and if any special cautions have been asked for

Learning point 3: be systematic when talking about management

Can either go by categories of actions or by problem list e.g. for the sepsis, for the suspected anastomotic leak, etc. Example -

- **Monitoring:** Paras Q30mins, inform if BP drops below ____, strict I/O charting, +/- NGT and catheterisation
- **Input/Output:** NBM, IV N/S 500ml over 1/2hr
- **Investigations:** Bloods and Imaging (for imaging/procedures, state view and urgency)
- **Meds:** broad spectrum antibiotics, anti-emetics
- **Intervention:** may need to bring back to OT for exploration and drainage of collections
- **Referrals/Allied Health:** not applicable in this case

CTSP: Desaturation

contributions from Vivien Lee

INSTRUCTION TO CANDIDATE

You are HO on call. You are at Ward 76 currently settling a new admission and speaking to the family. You have been called urgently by a nurse to see a patient at Ward 57 for desaturation.

Your tasks are to:

1. Take the call from the nurse.
2. Instruct the nurse on what to do.
3. Detail your subsequent management.

INSTRUCTION TO SP - NURSE

You are Staff Nurse Lola.

You begin by calling the candidate “Good evening Dr, this patient, Mr Tan Yeow Bing (S1423544C), 4 hours post total thyroidectomy, has desaturated”. Additional information:

- He is on 2L nasal prongs and SPO2 is 80%.
- Vitals are very stable: BP 155/90 HR 122 RR 30 T 37
- General condition: conscious and talking (if prompted – speaking in short phrases). Patient is very anxious and that has been his personality.
- You do not believe that the condition is serious and will attempt to persuade the candidate to allow you to convert Mr Tan to face mask and call him/her back in one hour for review.

When the candidate reviews the patient, provide the following information

- Difficulty breathing started 30 min ago. No other symptoms.
- Examination: stridor ++, in respiratory distress, conscious, heart and lungs otherwise unremarkable.
- Neck – dressing is soaked.
- Case notes: presented with neck lump 2 months ago, on FNAC, found ‘papillary thyroid CA’. Elective total thyroidectomy, op notes – nothing remarkable, no complications. No other medical history or drug allergy.

When the candidate suggests opening neck dressings – inform that fresh bleeding and blood clots are seen.

MARKING RUBRIC

		Performed and competent	Performed but NOT fully competent	Not performed or incompetent
1	Take the call, assess severity and see immediately	2	1	0
2	On the phone, can instruct nurse to do/prepare NRM at high flow O2 Continuous monitoring + Crash Cart Bloods: ABG, blood tubes for FBC, CE, TFT, Ca ECG	2	1	0
4	Resuscitation – Assess airway, breathing, circulation	2	1	0
5	Brief history and physical examination Onset Associated symptoms eg chest pain, cough, fever. General state: vitals, conscious level Neck: hematoma formation Respi: distress, stridor, lungs (wheeze, silent) CVS: any arrhythmias, CCF	3	1.5	0
6	Quickly review case notes, post-op instructions, and drug allergy	2	1	0
7	Assessment of neck hematoma and management Open dressing and make diagnosis of hematoma Also need to cut subcuticular stitches and stitches holding strap muscles - you must cut until you see the trachea	3	1.5	0
8	Escalate to senior using SBAR Need to prepare for return to OT for evacuation of hematoma and hemostasis.	2	1	0

Immediate fail:

- Fail to recognize patient is in critical condition
- Fail to escalate

Note: Consideration of other ddx is not strictly necessary if the diagnosis is as obvious as in this situation, but also consider

- If thyroidectomy is for Graves' disease – thyroid storm causing CCF
- Other post-op complications of thyroidectomy – e.g. airway compromise from bilateral recurrent laryngeal nerve injury, arrhythmias from hypocalcaemia. (be familiar with post-op complications of thyroidectomy)
- Unrelated – AMI, pneumothorax

CTSP: Dyspnoea

contributions from Eugene Gan

INSTRUCTION FOR CANDIDATES

You are a House Officer in the General Surgical department of XYZ hospital. It is 8pm and you are extremely busy because there are 4 new admissions to clerk. You are called by the nurse to review Mr Chong, a 20-year-old National Serviceman with no past medical history who was admitted for an infected sebaceous cyst for shortness of breath. He is planned for operation in EOT at around 10pm.

Your tasks are to:

1. Speak to the nurse on the phone.
2. You have finally arrived at the ward. Explain what other history you would like to obtain and what you would like to do.
3. Instruct the nurse to institute management accordingly.
4. The patient has been stabilized. Update the GS registrar on the patient's condition and your plans.

INSTRUCTION FOR SP - STAFF NURSE

You are Staff Nurse Declarador. You speak to the House Officer over the phone and inform him that the patient has some shortness of breath.

If the candidate requests, you are to offer a low BP of 90/60, and also note that the patient has facial swelling, some itchiness in the throat, and that you diligently went to listen and heard wheezing. Do not offer vitals, or any supportive management if the candidate does not offer. Apologize to doctor profusely for disturbing and say he can come later if really busy.

At the bedside, provide the following information when asked

- Patient has no past medical history and is allergic to penicillin
- Patient arrived in the ward 2h ago with infected sebaceous cyst. He has been given augmentin (you just served the dose) and is scheduled for emergency OT in 1h.
- Shortness of breath started 10min ago. No chest pain / diaphoresis / cough / fever. Some facial swelling.
- If candidate orders investigations – ECG normal, all bloods pending.

If candidate requests for medication, ask candidate to identify correct drug and dose. For adrenaline, offer candidate the 1:10000 or 1:1000 dilution.

INSTRUCTION FOR SP - GS REGISTRAR

You are the GS registrar. You will receive the HO's call. Ask for the patient's current vitals and congratulate the HO on saving the patient. Tell the HO that since the patient's vitals are stable, and the OT has a free slot, we should send patient to OT before all the emergency cases come in again.

MARKING RUBRICS

		Performed and competent	Performed but NOT fully competent	Not performed or incompetent
1	Requests for patient's vitals over the phone	2	1	0
2	Obtains focused history of shortness of breath to determine diagnosis of anaphylaxis.	2	1	0
3	Asks specifically for drugs that the patient is on and ascertains allergy	3	1.5	0
4	Offers supportive management meanwhile e.g. supplemental oxygen, running the existing drip fast.	2	1	0
5	Escalates medical emergency to senior	2	1	0
6	Attends to ABC of the patient (recognizes airway emergency in anaphylaxis)	2	1	0
7	Orders correct dose of adrenaline - 0.1 mg of 1:10000 IV (1mg in 10ml so you give 1 ml) IV, or - 0.5 ml of 1:1000 IM	2	1	0
8	Orders antihistamine and hydrocortisone 200-300 mg IV	2	1	0
9	Summarizes condition to GS registrar succinctly using SBAR, recommends postponing of op, monitor patient for now, run fluids (KIV hydrocort infusion, bronchodilators)	3	1.5	0

Immediate failure

- Agrees with nurse that this is non urgent and patient can be attended to later.
- Fails to ask for drug allergies in the patient
- Fails to identify that Augmentin is a penicillin, agrees with nurse that the medicines are different.
- Conducts entire station without attending to hemodynamic status of patient.
- Fails to mention adrenaline in treatment of patient.
- Agrees with GS registrar that patient can be sent for op asap.

DISCUSSION**Initial approach to an acute medical emergency**

- You can do quite a lot over the phone
 - Ask for vitals and ABC
 - Ask for a crash cart and resuscitation trolley (i guess even if you are nervous in the future its better safe than not have it)
 - Can institute some acute management if necessary (O2, run the drip fast)
 - Other helpful bedside tests that nurse can help you do: CBG, Urine Ketones ECG
- Always escalate early

Approach to SOB in a young patient: always have differentials

- Cardiac causes possible but less likely
- Respi: Pneumothorax, Asthma exacerbation, Anaphylaxis, PE (unlikely if no risk factors)
- Hemato: Less likely unless hemolysis; very acute
- DKA

Diagnosis of anaphylaxis

- There are 3 different definitions based on likelihood of exposure to allergen
- But hypotension and/or stridor is a clear red flag
- Other systems you can ask: think Muco-cutaneous -- Rash, Respiratory - Upper and Lower, GI surfaces

Management of anaphylaxis

- See marking rubric; adrenaline is a must especially if it is hypotension
- Should observe 24h as there can be a delayed phase reaction.
- Should enter 'anaphylaxis' into drug allergy information.

CTSP: Electrolyte abnormality

INSTRUCTION FOR CANDIDATES

You are the on-call orthopaedic House Officer, called to see Mdm Siao Zhar Boh (S0718923F), a 62 year old lady admitted four days ago for infected diabetic foot ulcer. The nurse informs you that there is a “critical lab result”.

Urea	16.2 mmol/L
Sodium	136 mmol/L
Potassium	7.2 mmol/L
Creatinine	214 μ mol/L
Bicarbonate	12 mmol/L
Chloride	98 mmol/L

Please review the patient. You may take a history, examine the patient, order investigations, and administer medications as required.

Medical record

Past medical history: Diabetes
Hypertension
Hyperlipidaemia
Diabetic retinopathy s/p photocoagulation
Ray amputation left 2nd toe for wet gangrene.
Peripheral vascular disease

This admission: Adm for discharging right sole wound with fever and ascending cellulitis.
Imp: sepsis secondary to diabetic foot ulcer.

I/O	4 days ago	In: 850ml	Out: 2200ml
	3 days ago	In: 1020ml	Out: 1700ml
	Yesterday	In: 995 ml	Out: 1040ml
	Today	In: 720ml	Out: 820 ml

Creatinine trend 67 (baseline) > 102 (3 days ago) > 214 (now)

Medication	IV	Premix (NaCl 0.33%, K 40mmol/L)	
	IV	Augmentin	625mg TDS
	IV	Cloxacillin	500mg QDS
	IV	Gentamicin	100mg BD
	SC	Insulin glargine	30U ON
	PO	Diclofenac	75mg BD
	PO	Paracetamol	1g QDS
	PO	Enalapril	20mg BD
	PO	Metformin	850mg BD
	PO	Glipizide	10mg BD
	PO	Bisoprolol	5mg BD
	PO	Simvastatin	20mg BD

INSTRUCTION FOR EXAMINER

Part 1: Emergent management of hyperkalemia

Candidates act as the house officer called to see Mdm Siao for a “critical lab result” and begin with a UECr result showing hyperkalemia, acidosis, and elevated creatinine. You are to act as the patient and examiner.

Candidates are expected to

- a) Review the patient clinically, checking that the patient is stable and asking for symptoms of hyperkalemia or cardiac instability
 - The patient is stable, no chest pain, palpitations, arrhythmia
- b) Explain to patient the current issue: Hyperkalaemia secondary to AKI
 - If candidate goes straight to giving drugs, prompt (Dr what are you doing to me?)
 - If candidate doesn't explain underlying AKI, prompt (Dr why do I have this, what have you done to me?)
- c) Emergent management of hyperkalemia:
 - ECG (normal)
 - Baseline capillary glucose (9.2 mmol)
 - Insulin (actrapid) 10U + dextrose 40-50ml: to run through stepwise
 - 10% Calcium gluconate 10ml/10min.
 - Resonium 15-30 g

Candidate to run through insulin procedure stepwise

- Flush plug before
- Draw up 40-50ml dextrose in 2x 25ml syringe.
- Choose insulin syringe (*identify the correct syringe!*)
- Choose correct insulin bottle (*actrapid*)
- Inject air into insulin bottle
- Invert and draw 10 units (0.1ml)
- Inject into dextrose syringe
- Connect dextrose syringe to plug and give as slow bolus (syringe without insulin first)
- Flush plug after



- d) Look for and act on underlying causes of AKI and hyperkalaemia – If candidate stops at emergent management of hyperK, prompt “Dr why do I have so high potassium?” / “will the high potassium come back again?”
- Look at vitals (currently afebrile, no hypotension in last 48 hours)
 - Look at I/O chart and examine volume status > note negative balance, likely prerenal AKI; to cautiously hydrate patient.
 - Examine for palpable bladder: none.
 - Look for baseline creatinine (AKI vs CKD) > provide to candidate that Cr on admission was normal
 - Stop medications that can cause nephrotoxicity and hyperkalaemia (provide drug chart on request). This includes ACE-I, gentamicin, NSAID, IV fluid with K.
 - Ask about recent scans > CT arteriogram (contrast) was performed 2 days ago.
 - [Bonus] Suspend metformin as CrCl <30 (if candidate requests for weight to calculate CrCl, provide CrCl = 21 ml/min).
 - [Bonus] Decrease augmentin to BD dosing due to low CrCl
- e) Review plan including capillary glucose monitoring, repeat UECr, ECG.

Part 2: Further review and escalation.

Provide the candidate the following information:

You review the patient 2h later and note that K is still 7.4 mmol/L. Cr is 210 mmol/L. Glucose is 7 mmol/L. A second cycle of insulin/dextrose is given.

You perform a 3rd review and find K 8.0 mmol/L, Cr 240 mmol/L. Glucose is 8 mmol/L. There has been no urine output in the 5 hours since your first review. What is your plan of action now?

If candidate requests ABG: there is acidosis with pH 7.28.

If candidate requests ECG: there are tented T waves.

If candidate requests clinical picture: patient is drowsy but asymptomatic.

Candidates are expected to refer to renal reg for urgent dialysis and conduct a rapid handover over the telephone. Use the SBAR template. For example -

Situation: I have a patient with AKI and hyperK. K has climbed from 7.4 to 8.0 even after 2 cycles of insulin/dextrose; Cr has climbed from 214 to 240 and the patient is anuric in the last 5 hours despite hydration. (There are now ECG changes).

Background: This is a 62 year old lady admitted for sepsis secondary to infected diabetic foot ulcer. Her I/O has been negative for the last 4 days and she has had multiple nephrotoxic drugs.

Assessment: My patient has failed medical therapy for hyperkalemia.

Recommendation: I would like to refer this patient for urgent dialysis.

> If candidate does not conduct adequate handover, prompt “why do you need to dialyse this patient? / AKI just hydrate Ia / hyperK just give insulin Ia”

MARKING RUBRICS

		Performed and competent	Performed but NOT fully competent	Not performed or incompetent
1	Greets patient and introduces self	1	0.5	0
2	Clinical review of patient	2	1	0
3	Correct assessment (AKI cx hyperK)	2	1	0
4	Emergent management: - ECG - Capillary blood sugar - Insulin 8-10U - Dextrose 40-50ml - Calcium gluconate 10% 10ml over 10min	2	1	0
5	Demonstration of correct insulin technique - As described above.	2	1.5	0
6	Rules out palpable bladder	1	0.5	0
7	Hydrates patient	1	0.5	0
8	Stops medication: IV fluid with K, gentamicin, NSAID, ACE-I.	2	1	0
9	Adjusts medication for Cr clearance - Stops metformin - Augmentin to BD dosing	2	1	0
10	Review plan - CBG - ECG - UECr	2	1	0
11	Identifies need for emergent dialysis	2	1	0
12	Handover of patient to renal reg	2	1	0

Immediate fail:

- Did not check 2 patient identifiers
- Overdose of insulin
- Choose wrong type of insulin
- Failure to monitor CBG/UECr thereafter
- Failure to flush before giving dextrose (fail criteria in 2015)

CTSP: Leg Pain

contributions from Aaron Tang

INSTRUCTION TO CANDIDATES

You are the HO on-call.

Mr Tan is a 55-year-old gentleman with a past medical history of HTN, HLD, and poorly controlled DM. He is currently on Enalapril, Metformin, and Atorvastatin. He was admitted to the ward 2 days ago for an Acute Myocardial Infarction.

You are called to see Mr Tan because he complains of right leg pain.

Please take a brief history and do a targeted physical examination. Discuss your diagnosis and management of the patient.

INSTRUCTION TO SP - PATIENT

You are Mr Tan, a 55-year-old cleaner. You were admitted two days ago for severe chest pain which you understand was a heart attack.

The chest pain had resolved on the day of admission and you have been fine since then. 3 hours ago, however, you started getting terrible right leg pain, mainly in the foot. The leg is not swollen but your toes are numb and you are starting to feel slightly weak.

Examination findings

- Right leg: pale, cold loss of sensation, delayed capillary refill and loss of DP, PT pulses. Leg is not swollen or erythematous
- Heart S1S2 no murmur, irregularly irregular pulse
- Lungs clear.
- Vitals stable; no fever or desaturation.

Other results:

- ECG: AF
- Doppler signal: Arterial Inaudible, Venous audible.

QUESTIONS

What are your differentials?

- Diagnosis: Acute Limb Ischemia
- DDX: DVT, Cellulitis

Origins of emboli:

- Cardioembolic - AF
- Artery to artery embolism - AAA
- Puncture site for AMI management

What is your management?

- Escalate to senior as a surgical emergency requiring active intervention e.g. embolectomy vs thrombolysis
- Resuscitate patient; improve existing perfusion by giving 100% oxygen, ensuring BP stable, keeping foot dependent.
- Consider Doppler US to determine if limb is viable, threatened, or non-viable; and determine level of obstruction (or bring patient to OT immediately)
- Initiate anticoagulation with heparin to avoid clot propagation (better than clexane as can be reversed during op). Begin bolus 3000-5000 units, infusion 1000 units/hour. Aim aPTT 2-2.5x normal.
- Prepare pre-op bloods
- Post-op, will require anticoagulation and treatment of AF.

One day after embolectomy the patient complains of generalized lethargy, dark colour urine. ECG shows hyperkalaemia. What is the cause?

- Rhabdomyolysis → aggressive hydration plus IV bicarbonate to alkalinise urine.
- AKI due to contrast angiogram.

What other complications can happen?

- Compartment syndrome
- Reperfusion injury

Miscellaneous questions

- What are the causes/mechanisms of acute limb ischemia? > Arterial embolism, thrombosis, trauma, dissecting aneurysm
- What are the most common sites where emboli lodge? > Bifurcation of femoral artery (most common site), trifurcation of popliteal artery (others: aortic bifurcation, external/internal iliac, arm)
- What are the contraindications to thrombolysis? Name 4.

CTSP: Seizure

INSTRUCTION FOR CANDIDATES

You are the on-call surgical House Officer. You just finished seeing a patient at Ward 57 when you get a call from Ward 75 regarding Mr Jiu Gui (S1234567A), a 67 year old man admitted 3 days ago for acute on chronic pancreatitis. The nurse informs you frantically on the phone that he is having a seizure.

You are currently on the phone with the nurse. Please instruct her on what you will like to do.

Medical record

Past medical history:	Chronic alcoholic pancreatitis Diabetes Hypertension Hyperlipidaemia Multiple strokes
This admission:	Acute on chronic pancreatitis (Glasgow 3)
Electrolytes	At Admission
	Urea 16.2 mmol/L
	Sodium 132 mmol/L
	Potassium 4.0 mmol/L
	Creatinine 68 μ mol/L
	Bicarbonate 22 mmol/L
	Chloride 99 mmol/L
	Corrected Calcium 2.2 mmol/L
Medication	IV Premix (NaCl 0.33%, K 40mmol/L)
	SC Insulin glargine 30U ON
	PO Tramadol 50mg TDS
	PO Paracetamol 1g QDS
	PO Enalapril 20mg BD
	PO Metformin 850mg BD
	PO Glipizide 10mg BD
	PO Bisoprolol 5mg BD
	PO Simvastatin 20mg BD

Upon request, provide:

	At Admission	Now
Urea	16.2 mmol/L	18 mmol/L
Sodium	132 mmol/L	125 mmol/L
Potassium	4.0 mmol/L	3.7 mmol/L
Creatinine	68 μ mol/L	80 mmol/L
Bicarbonate	22 mmol/L	16 mmol/L
Chloride	99 mmol/L	105 mmol/L
Corrected Calcium	2.2 mmol/L	1.5 mmol/L

INSTRUCTION FOR EXAMINER**Part 1: Emergent management of seizure**

Candidates act as the house officer called to see Mr Jiu Gui for seizure.
You are to act as the nurse assistant and examiner.

Candidates are expected to

- a) Drop everything and run
- b) Do what is possible over the phone
 - a. Get important information: when did it start, what made you say pt is seizing
 - b. Ask nurse to do the following: stat hypocount, 1 set of vitals including SpO₂, turn patient to left lateral, prepare IV/IM diazepam 5mg, give INO₂, lateral position clear environment and clothes
 - The patient is stiff and jerking eyes uprolling not responsive, started 2 minutes ago just as pt's dinner plate was being cleared
 - When candidate reaches ward, pt is in left lateral but still seizing. Hypocount 6. Afebrile BP 180/100, HR tachycardic, SpO₂ 95% 2LNP. Check plug. ECG. Prepare rectal diapam or IV lorazepam Get E cart
- c) Assess ABC. Confirm seizure. Abort seizure.
 - a. IV diazepam 5mg or IV lorazepam 4mg
 - b. Patient still seizing after 5 min → IV diazepam 5mg or IV lorazepam 4mg
 - c. Patient still seizing after another 5 min → IV phenytoin
 - i. Need HD bed, call senior and ICU for bed using SBAR
 - ii. How to give phenytoin
 1. IV, through a large vein, at least cubital
 2. 15-20mg/kg loading dose slowly over 20 min, maintenance dose 100mg Q6-8H 12h after 1st dose (adjust dose based on response and serum levels; aim trough 10-20)
 3. Requires cardiac monitoring
 4. Watch for purple glove syndrome

Scenario progress: But before you give the IV phenytoin patient stops seizing

- d) Look for and act on underlying cause of seizure, during the 5min waiting time
 - a. Review vitals over past few days → any HTN, signs of raised ICP,
 - b. Look at seizure type / frequency > first seizure, generalized. ? Scar epilepsy -- not focal, not recurrent.
 - c. Ask for last drink → patient drowsy at the moment, but nurse says she found some beer cans hidden in the patient's locker 2 days ago, and she noticed pt has been agitated/irritable and eats quite slowly because of hand tremors
 - Put patient on CIWA charting
 - d. Review IOs, bloods over last few days
 - Hypocalcaemia: 10ml 10% Ca Gluconate over 10 min
 - Hyponatremia: stop premix 0.33% saline, **slow** replacement
 - e. Review meds - any AEDs?
 - f. Examine pupils
 - g. Investigations: FBC, RP Ca/Mg/Phos, LFT, CT Brain, ECG, cardiac enzyme
 - h. Q1H para + CLC + fit chart, call dr if GCS drops >2, keep NBM, IV NS drip,

CTSP: Transfusion Reaction

INSTRUCTION TO CANDIDATES

You are the HO on-call.

Mr Beer is a 55 year old gentleman with known cirrhosis. He was found lying in a pool of vomit at a Geylang coffeeshop, with 10 bottles of stout beside him. As his Hb was 5.8 on admission, your MO has just started a transfusion of packed red cells half an hour ago.

You are called to see Mr Beer because he is now complaining of chills and rigor. His temperature was 38 degrees. The ward sister will help you with managing the patient.

DISCUSSION

Transfusion reactions can be classified as immediate or delayed. Other conditions (e.g. sepsis) may well mimic a transfusion reaction.

	Acute	Delayed
Immunologic	Hemolytic transfusion rxn (ABO mismatch) Anaphylactic rxn (IgA deficiency) Febrile nonhemolytic rxn (donor WBC cytokines) Urticaria (serum protein rxn) TRALI (recipient neutrophil activation)	Delayed hemolysis
Infection	Sepsis (transfusion transmitted bacteria)	Hepatitis / HIV (rare)
Overload	Volume overload (TACO)	Iron overload

Initial resuscitation

- Stop transfusion
- Keep IV open - normal saline
- Is this the correct blood product for the correct patient? (apparently “correct” does not rule out hemolytic rxn as GXM could have been drawn wrongly)
- Look at vitals (fever, hypotension, desaturation)
- Enquire symptoms and signs: dyspnoea, hemolysis (flank/back pain, bleeding, hematuria, jaundice), puritus

Recognize clinical picture and manage accordingly:

1. Obvious anaphylaxis: hypotension, wheeze, angioedema; acute onset < 15min. No fever.

- Treat as anaphylaxis: adrenaline 0.3mg, antihistamine, steroid,
- Subsequently: test IgA levels.

2. Prominent respiratory symptoms: TRALI vs TACO (the absence of respiratory symptoms make these less likely)

- More likely TRALI: hypotension, fever. More likely TACO: signs of fluid overload, underlying heart failure or ESRF.
- Workup: CXR, NTproBNP, ABG.
- Diagnostic criteria for TRALI
 - ARDS: hypoxemia (SpO₂ ≤90% RA, P/F <300)
 - CXR bilateral infiltrates
 - New within 6h of transfusion (acute onset and not there previously)
 - No circulatory overload
- Suspected TRALI: may need intubation & ventilation in ICU. Inform blood bank (may need to remove donor from donation pool)

3. Likely hemolytic transfusion rxn: mismatched blood, pain at infusion site or flanks, hemolysis (jaundice, dark urine), bleeding (e.g. oozing from catheter sites):

- Call blood bank - urgent as another pt may receive wrong blood
- Return ALL blood and blood set to blood bank (do not transfuse unused units)
- Supportive care
 - Monitor vitals, I/O, airway q1h
 - May need HD/ICU
 - Keep IV line and kidneys open: hydrate aggressively +/- forced diuresis.
 - Escalate to senior
- Bloods:
 - FBC + PBF
 - UECr
 - Hemolysis w/u: Coombs, Haptoglobin, LFT, LDH
 - DIC screen: D-dimer Fibrinogen PTPPT
 - Repeat GXM

4. Possible hemolytic transfusion rxn: serious rxn, not anaphylaxis, respi symptoms not prominent, but not definitively hemolytic at first presentation before bloods are returned. Manage as per hemolytic rxn if patient is sick --- concomitantly, or if bloods return showing no hemolysis,

- Consider transfusion transmitted sepsis (especially if severe fever / chills / hypotension without hemolysis on blood analysis) - Gram stain, Blood culture and empiric cover
- Consider conditions unrelated to transfusion

5. Mild reaction: temp rise $<1^{\circ}\text{C}$, mild symptoms (only fever or urticaria), hemodynamically stable, no hemolysis / hematuria, blood correctly matched.

- Symptomatic rx: paracetamol (febrile non-hemolytic rxn), piriton 4mg and hydrocortisone 100mg (urticarial rxn)
- Restart transfusion at slower rate
- Watch closely and review soon
 - If fever continues or symptoms worsen, mx as for serious rxn.
 - If symptoms resolve, continue. Inform blood bank but no need lab inx.
- Future prevention:
 - Febrile non-hemolytic rxn: use leukoreduced blood
 - Both: premedicate

Focused Task: Drowsy Child

contributions from David Ng

INSTRUCTION TO CANDIDATE

You are the MO at the ED seeing Mr Ng and his 10yo son, David. He has come in because he has been having a fever, cough and is now feeling breathless and a little drowsy.

Please take a short history from Mr Ng, do a quick examination, order investigations and administer management.

INSTRUCTION TO EXAMINER

Candidates are

1. History and examination

- Vitals T 38.0 HR 140 RR 26 BP 110/80 SpO2 95% RA
- Past medical history of T1DM
- Fever and cough and breathlessness for the last 2 days, getting worse
- Today – also drowsy, vomiting. Parent tried to measure blood glucose at home but machine broke down today (it gave some error)
- Examination findings: consistent with right lower zone pneumonia, dehydration

2. Investigations: please provide results when requested

- CBG: 27mmol/L
- UECr: Na 144 K 4.0 Ur 14 Cr 110 Cl 103
- ABG: pH 7.14, pCO2 33, pO2 90, BE -4 HCO3 18 Lactate normal
- FBCL: w 15 Hb 14 Plt 400
- Urine ketones ++, glucose ++, serum BHB 1.2 mmol/L
- CXR: right LZ patch

3. Management

- Resuscitation: supplemental O2, large bore IV cannulae, admit to HD
- Fluids:
 - Estimate dehydration 5% (pt has tachycardia, slight lethargy. cap refill less than 2 seconds, no tenting of skin)
 - Ask for weight: 40kg
 - Use Holliday-Segar method to determine fluid requirements, calculate deficit + maintenance; replace deficit over 2 days (for DKA, vs over 1 day in GE)

- Replace potassium while starting insulin
- Start insulin 0.1U/kg/hr infusion (no bolus in paediatrics but do not mark wrong)
- Start appropriate antibiotics
- Monitoring in the ward: Vitals hourly, BGM hourly, RP 2-6 hourly, ABG every 2-4 hours

4. Candidate should offer to call senior & also update the parent on patient.

Angry Patient: Adverse Event

contributions from David Ng

INSTRUCTION TO CANDIDATE

You are the medical House Officer and has just been called to attend to Mr H.D., an 85 year old man, who had fallen from the bed to the floor. He was not on restraints because his family members had specifically requested to withhold restraints.

Mr H.D. has known dementia with behavioural and psychological symptoms of dementia (BPSD), atrial fibrillation on warfarin (latest INR is 2.3), and poorly controlled diabetes.

He was admitted 4 days ago with fever and agitation, which was diagnosed as community acquired pneumonia. Your team has started penicillin, ceftazidime, and azithromycin; his fever lysed within 6h and he has been calm the last 3 days. Two hours ago, however, he spiked a fever of 38.2 degrees.

Mr H.D. is currently non-toxic but febrile (T 38.5). His vitals are stable. He is oriented to place and person but not time, and he is somewhat confused. He does not have any obvious injury including head wounds or tenderness or bruising over his limbs. His physical examination is absolutely normal.

At this moment, Mr H.D.'s son walks into the ward. He has heard that his father had just fallen. He demands to know what has happened and is rather dismayed. Please speak to him and

1. Explain to him what has happened
2. Detail your current plan of management

INSTRUCTION TO SP - RELATIVE

You are Mr H.D. junior. You heard that your dad had fallen in the ward and rushed over to see him. You are horrified and furious that such a thing has happened even though you are paying \$1000 a day for the bed alone.

You want a full apology and an explanation of what has happened - you had previously requested for your father to not be restrained but fully expected that a dedicated nurse would be watching over him all the time. You also want to know the name of the nurse in charge of monitoring your father, with the intention to sue her (and the hospital) for negligence. You also want to speak to the consultant in charge, and for the entire inpatient stay to be free-of-charge.

You will calm down once the candidate empathises with you and explains how the incident will be investigated. You want to find out if your father suffered any injury and what will be done for him from now onwards.

MARKING TEMPLATE

		Performed and competent	Performed but NOT fully competent	Not performed or incompetent
Communication with angry relative				
1	Greets patient and introduces self	1	0.5	0
2	Request to verify the son's identity	1	0.5	1
3	Explains and apologises for the fall	2	1	0
4	Express empathy	2	1	0
5	Professionalism: not to blame family for requesting not to restrain Mr H.D.	2	1	0
6	Professionalism: not to disclose name of nurse in charge; instead to file incident report	2	1	0
7	Mediates meeting with consultant	1	0.5	0
8	Note request for fee waiver but not to promise	1	0.5	0
Management				
8	Explains no obvious injury at present but need to monitor and investigate	2	1	0
9	Escalate to senior for further management	1	0.5	0
10	Note fever spike - need to redo septic workup	2	1	0
11	Require CT brain and conscious level monitoring due to warfarin	2	1	0
12	Consider restraints (physical or chemical)	1	0.5	0
13	Closure and summary	2	1	0

Immediate fail

- Denies or downplays the fall that has occurred.

DISCUSSION

Dealing with an angry patient or relative is a life skill and can make or break your day. The key is to recognize that this relative has a very legitimate reason to be upset. Inpatient falls are a difficult issue - while minimising falls is a patient safety goal, staff to patient ratio is rarely ideal, and there is a good argument to minimise physical or chemical restraints.

It is important to be frank and upfront - apologize sincerely that it had happened and express empathy (without accepting liability "I am sorry that this has happened" rather than "I am sorry that *we* did this"). Be on the same side as the patient and family - acknowledge that an adverse event happened you will do your best to make sure Mr H.D. is okay. Do not blame anyone - neither the patient's family (for insisting to avoid restraints), nor the staff nurse. Maintain professionalism and do not implicate your nursing colleague. If pressed to give the name, a useful defence is - "This is a serious incident which I have to report to an independent patient safety committee whose job is to investigate the incident from all angles and you may also be called to give a statement. Thereafter the independent investigators will update you on the outcome. We will put measures in place to minimise the risk of future incidents" The family is entitled to complain if they choose to and you should direct them to the patient's relations office, not beg them to not complain. You should arrange for a family conference with your consultant but should not promise a fee waiver.

Management post-fall: in every angry patient scenario it is important to describe what you have done or will do to correct the situation. For falls, most hospitals have a given protocol which should be followed. In this case, you should identify that the patient has spiked a new fever and a full septic workup is required. He is also on warfarin and hence the risk of intracranial bleed is present; a CT brain should be considered. You should also escalate to your senior.

The family's expectation for a dedicated nurse to watch Mr H.D. all the time is not reasonable - but deal with this sensitively. It is wiser not to call them out in a confrontational manner (i.e. don't tell them that they are unreasonable), but you should consider breaching the topic of physical or chemical restraints, at least until Mr H.D. is more lucid.

Angry Patient: Negligence

contributions from Vivien Lee

INSTRUCTION TO CANDIDATE

You are the orthopaedic HO. Your patient Mdm. Tan was admitted for elective total knee replacement. This was complicated by bleeding peptic ulcer on post-op day 3. Mdm Tan has since recovered and you had just completed the discharge summary and finalized discharge medications which are as follows:

- Amoxicillin 1g BD
- Amitriptyline 25mg at bedtime
- Clarithromycin 500mg BD
- Lisinopril 40 mg OM
- Metformin 500mg BD
- Metoprolol 50 mg BD
- Naproxen 500mg BD
- Omeprazole 20mg BD
- Simvastatin 40mg BD

While finalizing other paperwork in the ward, you are confronted by Mdm. Tan's daughter, Ms. Claire.

INSTRUCTION TO SP - DAUGHTER

You are Ms Claire, Mdm Tan's daughter. Your mother was admitted for elective total knee replacement complicated by bleeding peptic ulcer on post-op day 3. She was very sick and had to be admitted to ICU. Emergency endoscopy was performed which found a large bleeding gastric ulcer. She has since recovered and is about to be discharge

You hear that naproxen, a painkiller, was responsible for causing the gastrointestinal bleeding. While going down to the pharmacy to collect your mum's medications, you are horrified to see that the surgical HO has prescribed naproxen again. You stomp back up into the ward to confront the surgical HO:

- 1) You are furious that the surgical HO prescribed naproxen again which harmed your mother. You want an apology, you want to complain, and want to speak to the consultant.
- 2) Secondary point - you notice that antibiotics were prescribed and do not understand why.

MARKING RUBRIC

		Performed and competent	Performed but NOT fully competent	Not performed or incompetent
1	Greets patient and introduces self	1	0.5	0
2	Confirms the relationship between Ms Claire and Mdm. Tan	1	0.5	0
3	Interprets the medication list correctly and identifies the wrong medication prescribed	2	1	0
4	Offers Ms. Claire a seat	1	0.5	0
5	Appropriate handling of angry patient - Acknowledge emotion - Let her vent	2	1	0
6	Apologise to Ms. Claire with sincerity - Not defensive / denial - Not pushing blame away.	2	1	0
7	Respects Ms. Claire's wish to file a complaint and/or speak to consultant → refer to patient relations or hospital incident reporting committee	2	1	0
8	Appropriate answer of why antibiotics were prescribed (triple therapy for H. pylori)	2	1	0
9	Offers solutions to rectify the situation, for example: - Amend prescription immediately - Senior to counter-check prescription - Consultant to call Ms Claire - Offer alternative analgesia - Incident report - Add NSAID to adverse drug rxn (so that she doesn't get it in future Follow-up in clinic KIV scopes (TRO malignant ulcer)	3	1.5	0
10	Summarises and thanks patient.	1	0.5	0

Immediate fail:

- Denies mistake

Discussion: see previous scenario of dealing with the angry patient.

Bad News: Cancer Recurrence

contributions from Eugene Gan

INSTRUCTION FOR CANDIDATES

Mr Tan is an otherwise well 62-year-old gentleman who underwent an Ultra-Low Anterior Resection for Stage II Rectal Adenocarcinoma with neo-adjuvant RT in February 2015. You are the surgical MO who is seeing him in the Colorectal follow-up clinic for his annual surveillance.

Unfortunately, his CEA levels on arrival are elevated and his surveillance CT TAP scans showed an anastomotic recurrence, with no other sites of recurrence or metastases.

Your tasks are to:

1. Break the news of anastomotic recurrence to the patient
2. Take consent for an abdominal-perineal resection for the recurrent cancer.

INSTRUCTION FOR SP - PATIENT

You are Mr Tan, as detailed above. You are initially hopeful but slightly anxious about your surveillance results. You are aware that this is your annual surveillance visit.

You are to initially express shock and disbelief at the news. You will then react in anger by claiming that it is the medical team's fault that the cancer has recurred (though you acknowledge that you were counselled about a small risk of recurrence). You are actually really worried that your lifespan will be abruptly shortened because you are hoping to see the birth of your grandson at the end of this year. You will be more assured if the candidate tells you that there are no systemic metastases and intent is still curative.

Moving on, you are generally agreeable for the second operation, but will keep questioning the candidate if there are alternatives to avoid an operation. You will want to know if this new operation will allow you to pass motion as usual. If the candidate forgets to talk about a stoma, you will ask if it is required for this operation.

MARKING RUBRICS

		Performed and competent	Performed but NOT fully competent	Not performed or incompetent
1	Greets the patient and introduces oneself	1	0.5	0
2	Good body language and management of patient emotions throughout	2	1	0
3	Breaks the bad news that anastomotic recurrence has occurred professionally.	2	1	0
4	Explain that anastomotic recurrence is unfortunate but a possible complication	2	1	0
5	Manage patient's claims of negligence by the medical team	2	1.0	0
6	Express that on the flipside, there are no systemic metastases and that lesion was picked up early	1	0.5	0
7	Explains that surgery is required and initiates consent taking	1	0.5	0
8	Correctly identifies that the operation is an APR with end colostomy	1	0.5	0
9	Explains alternatives but maintains that surgery is the only curative option	2	1	0
10	Explains the procedure and possible complications	2	1	0
11	Explains the requirement for the stoma and how stoma care will be provided for him.	2	1	0
12	Summarizes the consult, expresses empathy and helps the patient to be optimistic	2	1	0

Immediate fail:

- Rude to patient, dismisses anastomotic leak as a risk that should be accepted easily
- Agrees with patient and puts blame on surgeon who operated.

Minus Marks:

- Forget to wash hands before
- Forget to wash hands after
- Usage of jargon or poor rapport.

DISCUSSION

The parts to this station are (1) breaking bad news, and (2) knowledge of anastomotic recurrence, its management, and (3) consent taking.

(1) Breaking bad news is a skill (see the other bad news station on HIV diagnosis) which must be approached sensitively with empathy. It helps to be on the patient side ('we want to help you') and to find the silver lining (we can cure you, there are no distant mets, we picked it up early). 'Blame' being one of the five defined stages of grief, allegations of negligence are not uncommon, and need to be handled professionally instead of emotively. Neither blame other colleagues, nor act overly defensive, or push the blame back on the patient ('you should have known since you were counselled that this could happen gave consent'). See the angry patient scenarios for further discussion on the theme of negligence. The subsequent stations are variations of the theme of breaking bad news.

(2) Do understand the considerations between an ultra-low anterior resection. It has a known risk of recurrence is known, and the decision to perform this operation is a tradeoff between minimizing the risk of recurrence, vs preserving function (anal sphincter). You should be able to justify why anastomotic recurrence requires at least an APR (if not a pelvic exenteration), and identify that APR entails having a stoma.

(3) This is dealt with in greater detail under the consent stations – see subsequent scenarios.

Bad News: Diagnosis of HIV

INSTRUCTION TO CANDIDATE

You are the medical MO in the General Medicine team.

Mrs Banker is the 42-year old wife of a year-old high-flying executive. She has no known past medical history. She presented to her GP with a 3-week history of mild shortness of breath, nonproductive cough, and fatigue. The GP noted that she was significantly hypoxic (SpO₂ 88%) and sent her to the Emergency Department for admission. On admission she was febrile (T 38.3) but much less symptomatic than the degree of hypoxia would suggest. A chest X-ray revealed bilateral patchy infiltrates, and a CT thorax showed ground-glass changes.

Mrs Banker is found to be HIV positive (both antigen/antibody and confirmatory testing is positive), with CD4 count is 92. Bronchoalveolar lavage revealed pneumocystis jiroveci. She is responding well to treatment with sulfamethoxazole, trimethoprim, and prednisolone.

Your consultant will like you to update Mrs Banker about her newly-diagnosed HIV. Please speak to her and answer any questions she may have.

INSTRUCTION TO SP

You are Mrs Banker, the wife of a high-flying forex trader. You have three children aged 4, 7, and 8. However your marriage is on the rocks and you suspect that Mr Banker has been seeking love outside, especially during his frequent overseas trips.

Upon revelation of the diagnosis of HIV, you are to react strongly with denial and anger, and question the diagnosis. You have always been fit and healthy and believe that you are just having a bad flu. You do not feel sick enough to be admitted, and believe that the GP had over-reacted.

You to be convinced when the candidate explains that the diagnosis of HIV is certain and in fact you already have full-blown AIDS. You will then turn hysterical and blame your unfaithful husband for bringing this upon you - you have always been faithful to him and have not had blood transfusions or IV drug use. Invite the candidate to join you in blaming your husband.

You will calm down when the candidate comforts you. You will ask if you are going to die and will be relieved when the candidate explains the treatment for HIV.

You also want to find out if you can still have sex with your husband. When the candidate explains that you have to inform your husband and should abstain from sexual intercourse at present, you will turn distraught. You believe that your husband will take the excuse to divorce you should he find out that you are HIV positive, or should you deny him sex. Furthermore you believe that he deserves to get HIV and die a painful death. You will refuse to inform your husband and challenge the candidate to inform your husband directly if he/she really wants to.

MARKING RUBRIC

		Performed and competent	Performed but NOT fully competent	Not performed or incompetent
1	Greets patient and introduces self	1	0.5	0
2	Assess patient's perspective (what do you already know)	2	1	0
3	Appropriate setting and obtain invitation to break bad news	2	1	0
4	Inform about HIV diagnosis (certain)	2	1	0
5	Provide empathy	2	1	0
6	Explain PCP pneumonia and its relationship with AIDS (CD4)	2	1	0
7	Professionalism: not to agree with patient to blame / condemn husband	2	1	0
8	Explain that HIV has a good prognosis and that treatment is available	2	1	0
9	Explain need to inform partner (by law) and better to abstain from sexual intercourse	2	1	0
10	Professionalism: not to notify husband on your own accord (unless patient provides consent).	2	1	0
11	Closure and summary	2	1	0

Demerit points

- Failure to wash hands BEFORE
- Failure to wash hands AFTER

DISCUSSION

This station tests (1) the skill of breaking bad news, (2) knowledge about HIV and treatment, and (3) professionalism in handling the public health implications and legal obligations of HIV.

(1) Breaking bad news requires appropriate setup - not to confront the patient suddenly with the bad news, but to provide some lead in and warn the patient that there is bad news coming. What happens after breaking bad news is equally important - to show empathy (especially to name the emotion) and explain how the patient will be supported.

(2) In this case the diagnosis of HIV is certain and therefore you may explain the diagnosis with certainty. In other situations, if only a first test is positive, a confirmatory test is required for diagnosis. The clinical situation of PCP pneumonia should also be explained. This is probably not the right setting to provide a full discussion of antiretroviral therapy, but at the very least the patient should be comforted that treatment is available and prognosis is good (even with CD4 <100, it can come up)

(3) The public health implications is a topic that requires some sensitivity. In this situation it does seem likely that the husband is the source of HIV infection but you must maintain a neutral and professional stance - neither to agree with her to blame the husband, nor to express doubt and probe for other sexual exposure apart from her husband (a full sexual history does need to be taken but probably not at first diagnosis!). It is Singapore law that sexual partners have to be informed of HIV status (even if condoms are used etc) - as the physician you should inform the patient of this duty but you are not to take the duty upon yourself (that is a breach of confidentiality; MOH will do so if necessary). Although not a marking point here, you can also inform that blood donation should be avoided.

Bad News: End of Life

contributions from Vivien Lee

INSTRUCTION TO CANDIDATE

You are the House Officer on call. You are called to see Mrs Mets, a 75-year-old lady with known metastatic breast cancer on palliative care. She was admitted last week with a pathologic humeral fracture when getting up from a sitting position. These have been managed conservatively with adequate analgesia. Subsequently, her stay was complicated by nosocomial pneumonia and she was started with IV piperacillin-tazobactam plus vancomycin. She is currently on the Dangerously Ill list (DIL) due to imminent demise.

The team has been dutifully updating Mrs Mets's husband who is her main spokesperson. The team, Mrs Mets, and her husband have mutually agreed on the extent of care: she is for maximum ward management and not for resuscitation or further escalation of antibiotics. *The patient and her husband have also requested that the diagnosis of metastatic carcinoma to be hidden from their children as they do not want to burden them.*

Mrs Mets's daughter, who had migrated to Germany, flew back to Singapore upon hearing the DIL situation and has just arrived in the ward. She has not heard from any doctor about her mother's condition and wants an urgent update.

Mrs Mets's current vitals are: T 37.5, BP 85/55, HR 103, RR 34, SpO2 90% on 100% NRM. She is drowsy and unresponsive.

Your task is to

1. Speak to Mrs Mets's daughter and update her about the patient's imminent demise.
2. Explain the joint decision on extent of care.

INSTRUCTION TO SP

You are Mrs Merkel, Mrs Mets's daughter. You flew back to Singapore urgently because you heard that your mother was in bad shape. This was very sudden because you never knew that your mum had any medical problems, and you feel particularly guilty for not visiting your mother in the past 3 years.

When the candidate informs you that Mrs Mets is dangerously ill, you are distraught that your worst fears are true. You find it strange that a previously healthy lady would sustain a fracture from such a minor injury, or turn so sick so quickly. You will probe the candidate for more

information - surely she must have some other medical problem that makes her so frail. You believe that Mr Met and the hospital is hiding information from her and will attempt to guilt trip the candidate into revealing the diagnosis.

When the candidate explains the extent of care, you are to react with horror. You believe that a DNR means doing absolutely nothing and in fact helping her to die faster. You feel terrible to know that your mum is in such terrible suffering and nothing is being done for her. You wonder if you will get to speak to your mum since she is already so drowsy.

MARKING RUBRIC

		Performed and competent	Performed but NOT fully competent	Not performed or incompetent
Breaking bad news				
1	Greets patient's daughter and introduces self	1	0.5	0
2	Setting: e.g. offer to have someone to be with her	1	0.5	0
3	Perception: determine what she already knows and her outlook on the situation	1	0.5	0
4	Invitation: warns that bad news is coming	2	1	0
5	Knowledge: Breaks bad news gradually and gently (current condition, poor prognosis, imminent demise)	3	1.5	0
6	Empathy: Encourages her to express her emotions, give sufficient time, and show empathy	2	1	0
7	Not to reveal diagnosis of metastatic breast carcinoma	3	1.5	0
Explain extent of care				
8	Explain reasoning for extent of care	2	1	0
9	Empathy: acknowledge her frustration	2	1	0
10	Clarify what is being done (not nothing)	2	1	0
11	Summary and closure	2	1	0

Immediate fail:

- Reveal patient's diagnosis of metastatic breast carcinoma
- Lack of empathy or inappropriate verbal or body language
- Inappropriate body language

Minus marks:

- Usage of jargons
- Inappropriate speed
- Failure to wash hands before
- Failure to wash hands after.

DISCUSSION

The theme of this scenario is (1) breaking bad news, (2) ethical issue of confidentiality, and (3) futility of care.

(1) Breaking bad news: do this using the SPIKES protocol (as above in the marking rubric). The goal is to break the news with the patient primed and not surprised, and thereafter to provide the best possible support. Recognize that this diagnosis is a big thing to the patient's family and do not downplay it. Offer condolences and reassure Mrs Met's daughter that she could not have prevented this. On the other hand, you cannot downplay the severity of the condition and you must inform Mrs Met's daughter that the patient is very ill and likely to die this admission, possibly in the next few hours.

(2) Confidentiality: Mrs Mets has the right to confidentiality - you have to stand your ground and not reveal the diagnosis of metastatic cancer even when pressured to (on the other hand it is alright to tell the daughter about the fracture, pneumonia, and extent of care). You may explain that you are duty-bound by Mdm Met's wishes which you have to respect, or suggest that perhaps Mr and Mrs Mets would like to tell her directly or did not want to burden her because she stays very far away. You can redirect the daughter to speak to Mr Mets directly but you should not insinuate or agree that "yes your dad is hiding something from you" which creates tension.

(3) Futility of care: You must first establish that recovery is not expected and the treatment goal is to keep Mrs Mets 'as comfortable as possible' and 'free of pain'. You may wish to explore whether the patient has expressed any thoughts on end of life care, or has made any advanced care plan. Ask Mrs Met's daughter what she understands by 'resuscitation', if not, explain in laymen's terms e.g. 'if she stops breathing we give oxygen through a plastic tube in a throat, we give medicine and chest compression to artificially make the heart beat. However this is a painful and undignified process for the patient, likely to cause more harm than good and may prolong suffering. Empathise that this is difficult to accept and explain that this is a difficult decision for the treatment team to make and apologise that she still remains ill despite all current efforts to treat.

You should also clarify that palliative management does not mean doing nothing in the face of terrible suffering, but it is a very active process of attempting to relieve suffering and make 'your mother as comfortable as she can be'. Give examples of what has/can be done e.g. medications relieve pain, breathlessness. On the other hand the side effects of opioid analgesia and hypnotics is to cause drowsiness; you can consider a sedation holiday so that Mrs Mets' daughter can have the chance to talk to her mother.

* Note: max ward is a medical decision and not a family's decision, but is usually done in collaboration with the family. There is evidence that there is no benefit to force NG feeding in such situations or in advanced dementia.

Consent: Routine

INSTRUCTION TO CANDIDATE

You are the Neuro MO.

Your patient, Mdm Siao Ting Tong has been admitted for a thunderclap headache which your consultant suspects is a subarachnoid haemorrhage.

Please take consent for lumbar puncture and answer all her questions.

INSTRUCTION TO SP - PATIENT

You are Mdm Siao Ting Tong, a 45-year-old lady.

You were admitted last night for a very severe headache which came on suddenly and was maximal on onset. You tend to have migraines but this particular headache felt rather different from your usual migraine.

You understand the worry about subarachnoid hemorrhage but are reluctant to undergo lumbar puncture. You will show your extreme reluctance and state blankly that you don't want to discuss subarachnoid hemorrhage. You emphasize that you are feeling much better now, and want to go home.

You have also heard that a 'scan' that can be done to check for subarachnoid hemorrhage and are keen to explore this option. When the candidate explains why this option may not be appropriate, you will relent and ask about the side effects of lumbar puncture.

When the student explains to you that there is a risk of bleeding, inform the student that you are on warfarin. If the student orders PT/PTT, inform him/her that your INR is 5.

MARKING RUBRIC

		Performed and competent	Performed but NOT fully competent	Not performed or incompetent
1	Greets patient and introduces self	1	0.5	0
2	Explains the clinical situation	2	1	0
3	Explains the indication for LP - Look for xanthochromic TRO SAH.	2	1	0
4	Explains alternatives	1	0.5	0
5	Explains that CT scan is not able to rule out SAH	1	0.5	0
6	Explains risks and side effects - Headache - Infection - Bleeding - Herniation	2	1	0
7	Notes that patient is on warfarin and orders PT/PTT	1	0.5	0
8	Identifies that patient is coagulopathic and withholds LP	2	1	0
9	Subsequent management - Call senior / haemato referral - Warfarin reversal - CT brain	2	1	0
10	Summarises and thanks patient.	1	0.5	0

Immediate fail:

- Deems LP unnecessary on the basis of CT scan
- Proceeds with LP despite on warfarin or INR 5.

Minus marks:

- Failure to wash hands BEFORE
- Failure to wash hands AFTER
- Poor rapport with patient.

DISCUSSION

Consent stations are rather straightforward but you cannot run away from adequate knowledge of the procedure discussed. You should cover:

- Clinical situation and indication for the procedure.
- Procedure: what the procedure entails and any necessary follow-up (e.g. subsequent monitoring, etc)
- Risks: divide into minor and common, vs major and rare. This is major ground for lawsuits so be careful how you explain risks; be comprehensive especially if the patient is likely to be particularly affected by any risk no matter how minor (e.g. a singer going for thyroidectomy), and ensure that the patient understands. At the same time try not to scare patients; explain the measures that will be taken to mitigate these risks, monitor the patient, and ensure safety.
- Alternatives

You should also look for contraindications before taking consent. In this case coagulopathy is a contraindication for lumbar puncture.

A variety of sample consent scripts are provided in the surgical long cases.

INSTRUCTION TO SP - PATIENT

You are Mr B. Jit, a 78 year old gentleman who has advanced alzheimer's dementia with no decision making capacity. You are orientated to person but not to time and place.

You are exceptionally cooperative and nod vigorously as the consent is taken. Your reply to any questions is restricted to a few favourite phrases including "Yes, yes", "I am okay" and "whatever you say". If the candidate asks what you understand of your illness you are to reply "I am okay", even if the candidate has provided you extensive information about your condition. You are to sign any document the candidate provides you.

MARKING POINTS

Consent taking

- Explain indication
- Explain procedure (colono, sedation, bowel prep)
- Explain risks.
- Explain alternatives.

Documentation

- Do not sign consent form - no capacity (pass/fail)
- Appropriate documentation of consent
- Appropriate documentation of lack of capacity, for example
 - Unable to retain information
 - Unable to understand situation
 - Unable to weigh options and come to a reasoned decision
 - Unable to express a choice.
 - AMT if done (bonus)
- Plan
 - Not to order bowel prep.
 - Collaborative hx from family
 - Acute workup for delirium, if not already done and this is not his baseline.
 - To return with two consultants for consent; and consult family

DISCUSSION

The consent portion is standard (see other consent on lumbar puncture) but what is crucial here is to realise that Mr B. Jit has no capacity. That Mr B. Jit because doesn't interact normally should become obvious when you speak to him. Don't be too anxious to barge through the consent, but pause and realise that he has no capacity.

Counselling: Anxious parent

INSTRUCTION TO CANDIDATE

Allie is a 2-year-old Chinese toddler admitted to your hospital for gastroenteritis, she is currently day 2 of illness. She is moderately dehydrated and was unable to tolerate orally, therefore you plan to start IV fluid rehydration for her.

You are the HO looking after Allie. Please update Allie's mother on the situation, progress and the plan from here.

INSTRUCTION TO SP

You are a very worried mother who has little confidence in the HO and the hospital. You want the best for your child and are very uncomfortable that no antibiotics have been given and Allie is not being fed. You believe that the hospital is neglecting Allie, not treating her (because no antibiotics are given), and making her starve. You will attempt to insist on antibiotic therapy and press the HO to "do something"

You will calm down once the HO empathizes with your worry and explains to you that this is a condition that naturally resolves, and that Allie will be treated with IV fluids. You are to react to the idea of inserting an IV plug which you believe to be very painful and a torture to your kid.

The last segment will be on discharge advice. You will inquire how to take care of Allie when she goes home, when she can resume feeding again, and how to prevent future episodes.

INSTRUCTION TO EXAMINER

The examiner is an observer in this station. Midway through the station you are to provide the following:

It is now D4 illness. Ally is adequately hydrated and the diarrhea is abating to 2 episodes of watery stool a day. She is still on IV hydration but was able to tolerate sips of water this morning. Her mother really wants to bring her home as she cannot cope with work, coming to hospital and having 3 more children at home. Please give her discharge advice.

Marking points:

- General approach and rapport with the mother
- Explore mother's ideas, concerns, and expectations.
- Expression of empathy
- Explanation of IV rehydration.
- Not to prescribe antibiotics
- Adequate discharge advice.

DISCUSSION

In dealing with the anxious patient or relative, several points are crucial

- Rapport is important; express empathy and be on the patient's side
- Strong knowledge of subject matter and the patient; being careful not to promise certain outcomes, and being quick to acknowledge what you are not sure about ('I will check with my consultant') instead of guessing
- Explore the patient/relative's specific ideas and concerns, so that they can be dealt with – until you do so, generic explanations will not satisfy the patient. Often there is something unsaid – probe when necessary.

A sample script is provided below:

Hi Mr ___/Madam _____

Introduction and setup: I am _____, the house officer who is looking after your child. May I first check what you know about Allie's condition and what are your concerns that I should address?

Explanation of condition: Your child has come down with gastroenteritis, which is infection of the digestive tract. Not to worry too much, this is a very common condition among children this age. The infection itself is self-limiting meaning that it will get better on its own, the main problem we usually deal with is dehydration due to the severe diarrhea and vomiting, which can be dangerous.

Plan: When Allie came in she was quite dehydrated [if quizzed, regurgitate; mild (thirsty and restless), moderate (mild + lethargy, irritable and decreased urination), severe (drowsy, cold peripheries, no urination)]. Because Allie cannot take oral fluids right now (she is vomiting +++), It is necessary for us to put a drip in for your child and give your child IV fluids, that means fluid into the vein. I am sorry that we have to poke your kid but it is important - what she needs the most now is to get enough fluid into her body and the only way we can do this now is through the IV because she is still vomiting and having diarrhoea. It is slightly uncomfortable but it is necessary and I will also do it even if it is my own child.

Why are you starving my child? Maam, we are not starving Allie, but we are waiting for her body to be able to tolerate food again. Until then, she will vomit out whatever we give her orally, which is not good either. The fluid we are giving her contains glucose and the various salts that the body needs. It will correct the dehydration, maintain the salt content in the blood, and provide energy in the form of glucose at the same time. We will observe your child for now. When your child gets better, we will stop the fluid, and he can start eating. We will do our best to take care of him.

Why no antibiotics? Most GE is due to a viral infection, and hence there is no benefit to start antibiotics. Antibiotics are not innocent - they have side effects such as allergy, killing all the good bacteria in the gut, and breeding resistant superbugs so that next time you really need it it may not work. However if we find that Allie does not improve or if we worry about a bacterial cause we will definitely give antibiotics when necessary.

It is now D4 illness. Ally is adequately hydrated and the diarrhea is abating to 2 episodes of watery stool a day. She is still on IV hydration but was able to tolerate sips of water this morning. Her mother really wants to bring her home as she cannot cope with work, coming to hospital and having 3 more children at home. Please give her discharge advice.

Discharge advice: We will take off the plug and stop the IV hydration before she goes home. But it is extremely important that she is kept well hydrated and continues to take in enough fluids orally ok? Ally is about 24kg, so ideally she should take in about 1580ml of fluids every day. You should aim for at least 50% of that, which would be about 800ml. Start with oral rehydration salts, water or diluted sugared drinks (sugar is osmotic and can worsen diarrhea) or milk (watch for intolerance, KIV soy formula or lactose free cow's milk). If she does well on that, you can slowly start her on soft bland foods like porridge or soupy noodles, even bread and toast. Danger signs that you should bring her back to hospital again are:

1. If she is drowsy, less active
2. If she has decreasing urine output
3. If she has blood in stool
4. If vomiting restarts
5. If she has severe abdominal pain and fever

Please be aware that you should take contact precautions while she still has diarrhea: Wash your hands and your child's after touching linen, changing diapers, using the toilet and before handling food/eating!

Counselling: Noncompliance

INSTRUCTION TO CANDIDATE

Ali is a 13-year-old Malay boy with thalassaemia.

He was first diagnosed at 5 years old when his kindergarten teacher noticed that he was short of breath whenever he played outdoors. An anemia workup revealed HbE-beta thalassaemia.

He has required intermittent transfusions every 2-3 months and is prescribed chelation therapy with desferrioxamine subcutaneous infusion. However, his ferritin is 1200 (target: 300-500, normal ferritin 200-300 mcg/L) and cardiac MRI has started showing signs of iron overload.

Please counsel Ali and his mother about his condition and compliance to chelation therapy.

INSTRUCTION TO SP - MOTHER

You are Ali's mother. Ali has always been a sickly child and you have had to bring him to the KK clinic every month, and he will also sometimes require blood transfusion, although you don't really understand why. You feel guilty that Ali is so sickly and wonder what you ate wrong while you were pregnant.

The doctors have told you many times that Ali must take his medicine each night, which involves putting a needle into his tummy and turning on a pump. If you are at home you will ensure that this is done, however you work night shifts at times and on those nights no one checks on Ali. When the candidate explains the life-threatening consequences of not giving the injections you are to act worried. You know that alternative oral medicines are available but are tight financially and cannot afford these alternatives.

INSTRUCTION TO SP - PATIENT

You are Ali, a 13 year old boy. You know that you have thalassaemia but don't know too much about it. You feel perfectly alright at present and do not understand why the doctors (and your mum) keep nagging you about the injections. You dislike having to inject yourself and find it cool to stay up all night playing computer games. You also feel very alone that among all your peers, you are at times too breathless to play football, and are the only one who needs to inject yourself.

MARKING RUBRIC

		Performed and competent	Performed but NOT fully competent	Not performed or incompetent
1	Greets patient and introduces self	1	0.5	0
2	Explores patient's understanding of disease	2	1	0
3	Explains disease (thalassaemia)	2	1	0
4	Reassures mother that it is not her fault that Ali has thalassaemia	2	1	0
5	Explores patient's understanding of treatment (chelation) and current compliance	2	1	0
6	Explains current iron status and adverse outcomes of hemochromatosis	2	1	0
7	Explains the importance of compliance to chelation therapy.	2	1	0
8	Identifies barriers to compliance (both mother and son)	2	1	0
9	Express understanding of barriers to compliance instead of blaming patient or mother	2	1	0
10	Explore suggestions to improve compliance (e.g. alternative drugs, ask father to check on Ali, support groups)	2	1	0
11	Offer support and summarise	2	1	0

Immediate fail

- Failure to identify noncompliance
- Harsh attitude, blames patient and mother for noncompliance.

Minus marks:

- Failure to wash hands before
- Failure to wash hands after

DISCUSSION

Your first task is to correctly identify the clinical problem of noncompliance. Suspect this if a patient is given the right treatment but clinical or biochemical measures are not improving – and first address the noncompliance, not top up with additional medication or measures to which the patient might not be compliant! Differentials for a patient who is not improving:

- Wrong diagnosis
- Correct diagnosis but wrong treatment
- Correct treatment, but not taking
- Correct treatment, taking, but not taking properly (e.g. technique)
- Correct treatment, taking, but not addressing precipitant (or other aspect of mx)
- Inadequate treatment or disease has progressed.

The general strategy to a noncompliant patient is *not* to nag at, blame, or scold the patient for recalcitrance, but to explore the *root cause* of noncompliance and address these factors. Very often patients have very valid reasons to not want to take their medications - because they do not understand its importance, cannot afford the medicine, or cannot tolerate the side effects. *Always find out more and preach less.*

A general approach is to (the order in which this is done will vary depending on the patient

- Establish non-compliance
 - Do not assume non-compliance simply due to poor response to treatment
 - Update that condition is not improving / complications have set in and invite patient to suggest why.
 - If patient denies and says everything is fine, gently challenge patient with the evidence and make a normalising statement (rather than 'you haven't been taking your medications right?', say 'how many times this week have you taken your medicine?')
- Explore patient understanding of disease and treatment
- Identify the barriers to compliance
 - Patient understanding and perceptions: does not see importance, feels OK,
 - Do not know how to use medicine
 - Medical factors: side effects, too many tablets
 - Patient factors: lifestyle, inconvenience, forgetting.
 - Financial factors: cost issues
- Express understanding with patient's difficulties
- Explain condition and treatment and importance of compliance
- Offer appropriate suggestions to solve issues (e.g. refer medical social worker for financial issues, change to another medicine that has easier dosing or fewer side effects)

Sample Q&A:

What is thalassaemia? Thalassaemia is an inherited disorder in the protein which that helps your red blood cells carry oxygen to your body. This makes your red blood cells unstable and break easily, so there is a shortage of working red blood cells to deliver oxygen.

Mother: What did I eat wrong when I was pregnant? Thalassaemia is inherited, which means it comes from a mistake in the gene in your DNA, which is passed down from parent to child. It has nothing to do with eating wrong food in pregnancy and it is absolutely not your fault. The reason why Ali has it but you and husband seem fine, is because you and husband each have only one gene defect which your bodies are able to compensate for, but Ali inherited 2 - one from each of you!

Ali: Doctor I feel ok, whats the big deal? Ali, I understand that you feel totally fine. And that's good, we want to keep it that way! The big deal is that with all the blood transfusions to help with your shortage of red blood cells, you are also getting an overdose of iron. This iron deposits itself everywhere -- your heart, liver, testes, etc, and causes all these organs to slowly fail (Heart: arrhythmias, heart failure; Liver: liver failure; Endocrine: delayed puberty, hypothyroid, panhypopit, diabetes). In the past when people received chelation without chelation, they died in the late teenage years from heart problems. This is why I keep scanning your heart and liver: to see how much iron has been deposited. Your body is already showing that it already has too much iron. This is why your chelation therapy is so, so important: the chelating medicines kiap/bind to the iron so that they cannot go into your organs!

If you really find it very difficult to stick to the daily injections, especially with all your school activities and going out with friends, there is now a new alternative of taking oral tablets. They work just as well, but they are very expensive.

Information: the available chelation therapies are as follows -

Agent	Route	Half-life (h)	Schedule	Clearance	Side effects
Deferoxamine (Desferal)	Slow infusion: intravenous or subcutaneous	0.5	OD	Renal, hepatic	Dermatological, ocular, auditory
Deferasirox (Exjade)	Oral	12 to 16	OD	Hepatobiliary	Gastrointestinal, renal, hepatic
Deferiprone (L1)	Oral	2 to 3	TDS	Renal, cardiac	Hematological (neutropenia, agranulocytosis, arthropathic

Note: deferoxamine is a slow infusion over several hours, not just an injection.

Counselling: Medication Issues

contributions from Eugene Gan

INSTRUCTION FOR CANDIDATES

Mr Chad is a 60-year-old gentleman admitted for a minor accidental fall. He has a significant medical history of hypertension, hyperlipidemia and atrial fibrillation on the following medications: amlodipine, simvastatin and warfarin 4mg ON.

He is clinically stable, alert and oriented, and examination was unremarkable apart from mild tenderness and a large bruise over his right upper arm. Investigation results revealed

- FBC, U/E/Cr, Glucose, ECG, and CT head were normal.
- X-Ray of his right arm was also normal.
- However, his INR was 6.3

Please speak to Mr Chad to explore his deranged INR, elicit any concerns and to formulate a management plan for him. You do NOT need to take the history for the fall, nor examine him again.

INSTRUCTION FOR SP - PATIENT

You are Mr Chad, a 60-year-old taxi driver. You were admitted to the hospital because of a minor fall which caused a large bruise. You wonder why such a minor fall caused such a large bruise.

You were started on warfarin 2 years ago due to an irregular heartbeat; you have not had issues with warfarin these 2 years. Your wife usually manages your many medicines, but she passed away last month. This month, you have been having problems remembering to take your pills; when you forget a dose and remember the next day, you will try to compensate by taking both doses together. You know that your dose is 4mg which is 4 small tablets.

You had a sore throat 2 weeks ago, for which a GP gave you erythromycin. Apart from this, and the hospital medication, you take no other drugs, over-the-counter painkillers, or traditional medicine. You are aware that you have to watch your diet and have not changed the amount of green leafy vegetables you eat, and take neither alcohol nor grapefruit juice. You have no past medical history of liver disease or recent jaundice.

Your main concerns are: (1) can you go home now, and (2) what to do if you miss a dose.

MARKING RUBRIC

		Performed and competent	Performed but NOT fully competent	Not performed or incompetent
Introduction				
1	Greets patient and introduces self	1	0.5	0
2	Inform patient of INR and establishes diagnosis of overwarfarinization	1	0.5	0
3	Brief warfarin history	2	1	0
Elicit cause of over-warfarinization				
4	Understanding of and compliance with warfarin dosing 4mg ON = 4 x 1mg tablets	3	1.5	0
5	Drug interactions Recent antibiotic use Other drugs: SSRI, antifungals, amiodarone, COX-2 inhibitors Show some awareness of what foods interact with warfarin	3	1.5	0
6	Recent changes in diet (e.g. travel, illness)	2	1	0
7	Liver function: elicit any history of liver disease, jaundice	2	1	0
Management and counselling				
8	Stop warfarin, stat vitamin K (e.g. PO 1mg), recheck INR next day	3	1.5	0
9	Counsel patient on how to handle missed doses (take if within 4h, never take double dose, if unsure call hospital to check)	2	1	0
10	Counsel patient how to remember better e.g. using a pillbox, ticking off calendar, phone reminders	2	1	0
11	Discharge with advice to return to A&E if uncontrolled bleeding, head trauma, joint swelling	2	1	0

DISCUSSION

This case illustrates the management issues and pitfalls surrounding warfarin.

Do also spend some time thinking through warfarin counselling – this is important and illustrates many of the considerations of medication counselling (for other medication – apply the same template). As for consent, you should cover: indication, contraindications, how to use the medication, risks and side effects, how to mitigate these risks, contraindications, and alternatives.

Indications

- You have an irregular heart beat, which causes an increased risk of a clot forming that may go to your brain and cause a stroke
- We need to give you warfarin, which is a blood thinning medication, to reduce the risk of this clots to

Contraindications: check relevant ones before starting

- Pregnancy: warfarin is teratogenic and may cause developmental problems, so you need to be switched to another medication if you are planning to get pregnant
- High bleeding risk: GI bleed, peptic ulcers, recent major surgery or plans for surgery
- Allergy
- Drug interactions: other anticoagulants, potent liver enzyme inhibitors (see below).

How to use warfarin safely:

We will give you a warfarin book detailing the food and medications you should avoid, and also for you to record your dosage of warfarin and INR

(a) Dosing

- Strictly follow the dose that the doctor has prescribed for you
- If you miss a dose and remember within 4h, take the dose. If you only remember the next day, DO NOT take double doses. If you are unsure, call the hospital.
- How to remember to take your medication: tools like pill boxes, phone alarms.

(b) Monitoring

- After going home, you must visit the anti-coagulation clinic at any polyclinic for regular blood tests to check how thin your blood is. your dose may be adjusted
- Frequency: daily when starting, then twice weekly for 2 weeks, then monthly
- If you ever require surgery, you may need to be admitted earlier, and may require bridging therapy*

(c) Avoiding drug interactions

- Screen through current drugs
 - Drugs that increase effect of warfarin: Antibiotics (ciprofloxacin, macrolides, isoniazid, ketoconazole, isoniazid, bactrim, metronidazole), NSAID, cimetidine, SSRIs, protease inhibitors.
 - Drugs that reduce effect of warfarin: anti-epileptics, oral contraceptives, rifampicin, nnRTIs
- Always inform any doctor or pharmacist that you are on warfarin, especially if you visit a GP or buy over the counter medicine.
- Avoid taking complementary or traditional medicine

(d) Dietary restrictions

- Eat a fixed amount of green leafy vegetables each day
- AVOID: Alcohol, cranberry juice, ginkgo, ginseng, fish oil
- Do not begin to attempt to lose weight without discussing with your doctor (warfarin is dosed by weight)

Side effects and risks

- Main risk is bleeding
- Come back to A&E immediately if you notice: bruises on arms, legs; painful joint swellings, black stools or blood in stools, vomiting of blood
- Any head injury, blurring of vision, weakness is potentially serious and you should come to A&E.

Alternatives: Novel oral anticoagulants

- Indicated only for non valvular AF
- Advantages – no need monitoring and multiple blood taking
- Disadvantages – no antidote for overdose, very expensive

*** Notes on bridging anticoagulation**

- Need for bridging depends on
 - Procedure bleed risk: minor procedures are low risk
 - Perioperative thrombotic risk: AF CHADS 0-2 with no stroke, DVT/PE >12 months and no other risk factors, or bileaflet aortic valve without AF, is low risk.
- If no bridging – stop warfarin 5 days before and restart 12-24h post-op
- How to do warfarin bridging
 - Stop warfarin 5 days before, start heparin or clexane
 - Last dose clexane 12-24h preop (omit night dose on POD -1 and POD0), last dose heparin 6h preop.
 - Restart warfarin 12-24h post-op based on clinical judgement

Ward Life: Needlestick Injury

contributions from May Na

INSTRUCTION TO CANDIDATES

You are the Medical HO. Your SIP gets a needlestick injury while taking a blood culture, next to you in the ward. She is distraught. Please speak to her and advise her what to do.

INSTRUCTION TO SP – SIP STUDENT

You are an SIP student who just sustained a needlestick injury the minute before your medical HO walks into the ward. You are distraught and do not know what to do. You do not know if the source patient is HIV or hepatitis positive.

You will calm down after being counselled by your HO. You will also want to find out

- What is the risk of transmission of HIV / hepatitis B & C
- What the occupational health clinic will do for you (post exposure prophylaxis)
- What is the consequence if you end up as HIV or hepatitis +ve.

MARKING RUBRIC

		Performed and competent	Performed but NOT fully competent	Not performed or incompetent
1	Calm SIP student down	2	1	0
2	Explore incident (what has happened, type of injury, what has been done)	2	1	0
3	First aid: Squeeze blood out, wash	2	1	0
4	Report to supervisor / ward sister	2	1	0
6	Go to staff clinic / Emergency Dept	2	1	0
7	Notify NUS (online system)	2	1	0
8	Need to take blood from patient to establish BBV status (HO can offer to do)	2	1	0

9	Follow up at occupational health clinic or infectious diseases, may require post-exposure prophylaxis	2	1	0
10	Risks of transmission	2	1	0
11	Consequence if results positive	2	1	0

DISCUSSION

Steps to take after a needlestick injury

- First aid: Immediately squeeze out as much blood as possible, wash with soap and water and/or disinfectant
- Establish patient's blood borne virus status: send blood for testing (HBV, HCV, HIV). Will need consent from patient; can ask another healthcare staff to take
- Report to supervisor and nursing officer (sister) on-call so that electronic hospital occurrence report can be filed.
- Immediately proceed to staff clinic (during office hours) or Emergency department (after office hours)
 - Bloods to be taken: HBV (HBsAg, anti-HBs IgG, anti-HBe IgG, HBsAb), HCV (anti-HCV IgM, HCV PCR), HIV Ag-Ab
 - Post-exposure prophylaxis started as soon as possible and continued for 4/52
 - Post exposure prophylaxis card issued by DO will cover the cost for investigations and PEP prophylaxis
- Follow up at NUS Occupational Health Clinic or ID
 - Get a referral with a copy of serology results from the doctor at staff clinic or ED.
 - Follow up at 1mo, 3mo, 6mo for seroconversion, toxicity from ARTs
- Notify NUS Occupational Health Clinic via online reporting form within 24h of incident

Further information

- Risk of transmission if not immunized is HBV 30%, HCV 3%, HIV 0.3%.
- Consequence of testing positive for Blood-Borne Diseases (BBD):
 - Cannot perform or assist in Exposure-prone Procedures (EPP): where there is a risk that patient's open tissues may be exposed to blood of the worker. Generally, ward work is OK, but not surgery.
 - Can still become a doctor.
 - But limitation in career pathways: cannot do surgery
 - Confidentiality will be protected
- Information about post-exposure prophylaxis (MOH guideline):

POST-EXPOSURE PROPHYLAXIS (PEP) AGAINST HBV INFECTION FOR HCW EXPOSED TO BLOOD AND/OR BODY FLUIDS

Immune Status of HCW	Patient Source HBsAg (+)	Patient Source HBsAg (-)	Source Not Tested Or Unknown
Unvaccinated	One dose HBIG and start one series of HB vaccination.	Start HB vaccine series.	Start HB vaccine series.
Previously vaccinated			
Known responder (anti-HBs ≥ 10 mIU/ml).*	No treatment.*	No treatment.*	
Known non-responder.	One dose HBIG and start one series of HB vaccine.	No treatment.	If known high risk source, treat as if source were HBsAg (+).
Antibody response unknown.	Check anti-HBs: If ≥ 10 mIU/ml, no treatment.* if < 10 mIU/ml, one dose HBIG and vaccine booster.	No treatment.	Check anti-HBs: If ≥ 10 mIU/ml, no treatment.* if < 10 mIU/ml, one dose HBIG and vaccine booster.

HBIG - Hepatitis B immunoglobulin
 HCW - Health care worker

HBV - Hepatitis B virus
 HB - Hepatitis B
 HBsAg - Hepatitis B surface antigen

* HCWs with a high risk of exposure may require a booster dose of HBV vaccine if anti-HBs is <100mIU/ml

POST-EXPOSURE PROPHYLAXIS (PEP) AGAINST HIV INFECTION FOR HCW EXPOSED TO BLOOD AND/OR BODY FLUIDS

Exposure	Source patient HIV (+)	Source Patient Unknown	Considerations
Mucous membrane or skin, integrity compromised • Small (few drops or short duration).	Low titer Source patient asymptomatic and high CD4 counts – may not need PEP, discuss with HCW.	No treatment.	Skin integrity is compromised if there is evidence of chapped skin, dermatitis, abrasion or open wound.
	High titer Source patient has advanced AIDS, primary HIV infection, high or increasing viral load or low CD4 count – consider prophylaxis with zidovudine 600 mg/day in two or three divided doses and lamivudine 150 mg twice a day.		
• Large (several drops, major blood splash and/or longer duration i.e. more than several minutes).	Low titer Source patient asymptomatic and high CD4 count – recommend prophylaxis with zidovudine 600 mg/day in two or three divided doses and lamivudine 150 mg twice a day.	If there is a possible risk for HIV exposure, consider prophylaxis with zidovudine 600 mg/day in two or three divided doses and lamivudine 150 mg twice a day.	

POST-EXPOSURE PROPHYLAXIS (PEP) AGAINST HIV INFECTION FOR HCW EXPOSED TO BLOOD AND/OR BODY FLUIDS

Exposure	Source patient HIV (+)	Source Patient Unknown	Considerations
Intact skin	High titer Source patient has advanced AIDS, primary HIV infection, high or increasing viral load or low CD4 count – recommend prophylaxis with zidovudine 600 mg/day in two or three divided doses and lamivudine 150 mg twice a day and either indinavir 800 mg every 8 hours or nelfinavir 750 mg three times a day. PEP not needed unless it is high exposure to blood e.g. extensive area of skin exposed or prolonged contact with blood.	No treatment.	
Percutaneous exposure • Less severe e.g. solid needle, superficial scratch.	Low titer Source patient asymptomatic and high CD4 count – recommend prophylaxis with zidovudine 600 mg/day in two or three divided doses and lamivudine 150 mg twice a day.	If there is a possible risk for HIV exposure, consider prophylaxis with zidovudine 600 mg/day in two or three divided doses and lamivudine 150 mg twice a day.	Combination of factors e.g. large bore hollow needle and deep puncture contribute an increased risk for transmission if source patient is HIV positive.

POST-EXPOSURE PROPHYLAXIS (PEP) AGAINST HIV INFECTION FOR HCW EXPOSED TO BLOOD AND/OR BODY FLUIDS

Exposure	Source patient HIV (+)	Source Patient Unknown	Considerations
• More severe e.g. large-bore hollow needle, deep puncture, visible blood on device, or needle used in source patient's artery or vein.	High titer Source patient has advanced AIDS, primary HIV infection, high or increasing viral load or low CD4 count - recommend prophylaxis with zidovudine 600 mg/day in two or three divided doses and lamivudine 150 mg twice a day and either indinavir 800 mg every 8 hours or nelfinavir 750 mg three times a day.		
	Low or high titer Recommend prophylaxis with zidovudine 600 mg/day in two or three divided doses and lamivudine 150 mg twice a day and either indinavir 800 mg every 8 hours or nelfinavir 750 mg three times a day.		

Final Advice

1. Don't lose marks in silly ways

- Wash your hands before and after. This has made the difference between pass and fail for borderline candidates.
- Always check 2 patient identifiers, especially if it is a procedural station.
- If you feel flustered – pause and take a deep breath, don't dash in and do something you will immediately regret

2. Be honest and be nice.

- Never fabricate history or make up signs.
- Be patient, gentle and courteous to the patient – however unhelpful, irrelevant, or confused the patient is.
- Be humble and teachable. Don't argue with the examiner – express your thought process, but if the examiner disagrees, it is usually better to concede and move on. Listen to their hints – the answer to 'will you like to re-examine' is always 'yes'!
- Take care of each other and look out for those around you – this is good for academic and emotional health.

3. Take charge of your learning

- Don't just keep charging ahead, but think about how you are doing. If something is not working, change strategy! Find something that works for you
- As you practice in the wards and each other, it is helpful to write down the mistakes you tend to make (e.g. forget to check for incisional hernia, forget to assess function, forget to take drug allergy). Learn from them and remind yourself just before you go in.

4. Take heart

- It's a new start every time you walk into another room or another exam. If one station has gone badly, remember that the examiners in the next station do not know that. Don't let your discouragement snowball.
- The bar is not too high: all you need to do is to show that you have been in the ward, can interact with patients, and will be a safe houseman.
- Unfortunately, accidents do happen and people do fail MBBS – at times due to factors in their control, sometimes not > But there is another chance 6 months later. In the long run, so what?

All the best!