Medicine Short Cases

thinking on your feet

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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.

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'.

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 -

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.

(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.

- 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 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, bizzare 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 -

- Ipsilateral[1] cerebellar dysfunction → Ataxic hemiparesis, a lacunar (subcortical) syndrome.
- Contralateral[1] 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.

PAEDIATRIC POINTERS

(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).

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.

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.

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)

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 > LL[2]
  - 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).

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.

	PAEDIATRIC POINTERS
• Turn patient and examine the back/neck ○ Look for operation scars → spinal trauma, repaired congenital spinal defects	Please do look carefully for scars on the back;
• Examine eyes +/- do fundoscopy ○ Look for multiple sclerosis / neuromyelitis optica: internuclear ophthalmoplegia, optic neuritis.	Less likely in paediatrics
• 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
	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

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

(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 (syphillis)

(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, V_1: Superior orbital fissure lesion
- CN III, IV, VI, V_1, V_2: 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 a 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

PAEDIATRIC POINTERS

In children also think of dystonic cerebral palsy due to

- Kernicterus
- Hypoxic ischaemic encephalopathy

(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 -

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.

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).

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) Characteristicallh 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 impt: hard/nodular? pulsatile? tender? Adults - measure entire liver span; kids - measure cm below costal margin.
Gallbladder	Like liver but globular outline	*Always* 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 paeds)
- 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' 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.

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.

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, pneumonec/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.

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?	**PAEDIATRIC POINTERS**
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	COPD does not happen in children; think asthma vs bronchiolitis
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	*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)
(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.	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
(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)	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.

(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.

(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.

PAEDIATRIC POINTERS

In kids, hyperresonance means pneumothorax

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

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.

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.

2. Consider etiology

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

PAEDIATRIC POINTERS

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

Differential in paeds: hypoplastic lung

	PAEDIATRIC POINTERS

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

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
	ILD is uncommon in kids

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
	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

Consolidation: • Infective (bacterial, TB): listen for productive cough, look for sputum mug, request temperature chart • Neoplastic: look for cachexia, lymphadenopathy, SVC obstruction, clubbing.	Ddx: hypoplastic lung - consolidation plus subcostal scar (repair of (diaphragmatic hernia), midline shift

	PAEDIATRIC POINTERS
Collapse: • Neoplasm (endobronchial or extrinsic compression): as above. • Endobronchial TB • Mucus plugging (e.g. asthma): look for wheezes • Foreign body: consider especially in a patient with dysphagia.	
Pneumothorax: • Marfan's syndrome → look for Marfanoid features: high arched palate, thumb sign, wrist sign, do Beighton scoring, listen for aortic regurgitation • Emphysematous bullae → if COPD present • Idiopathic.	
These do not have as much to look for: • COPD: look for nicotine staining, smell of smoke • Pneumonectomy or lobectomy: e.g. cancer, also done previously for TB.	
3. Look for complications Complications across all diseases include • Respiratory distress: patient using accessory muscles of respiration, sitting forward, not speaking in full sentences, requiring supplemental oxygen • Pulmonary hypertension: look for palpable pulmonary component of second heart sound, parasternal heave Extras for individual diseases • COPD: look for signs of malignancy (cachexia, clubbing), pneumothorax or old chest tube scars.	Comment on growth and nutrition status
4. Requests • Temperature chart • Sputum mug • Pulse oximetry • ENT for bronchiectasis: nasal polyps in cystic fibrosis, otitis media in ciliary dyskinesia	Request growth charts.

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 learning 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 is quite different 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)

- Dysmorphisms: 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 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 have quite unique features and multi-system manifestations. The key is to recognize the diagnose 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 90º - 6 weeks
- Fixate and follow to 180º - 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 feet per step - 3 years
- Alone down stairs, 2 feet per step - 3 years
- Alone down stairs, 1 feet 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 permanance, 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.

APPENDIX

DDST Singapore – a comprehensive DA chart but do not memorise! (MOH 1992)