Context for the session

The Intended Learning Outcomes are to be able to:

- Discuss common causes of collapse
- Describe how to gather a well-rounded impression of a patient presenting with a previous episode of collapse and formulate a differential diagnosis
- Perform upper and lower limb neurological examination

Students will have covered the following in the two-week collapse block:

In **Case-Based Learning** a 76 year old man was brought to the Emergency Department following a collapse at home; and his granddaughter (who fainted/had vasovagal episode when she witnessed venesection).

In **Lectures, workshops and practicals** students will learn about:

- What do you need to maintain consciousness?
- Neural networks
- Epilepsy
- Stroke syndromes
- Cardiac causes of collapse (electrical and structural)
- Shock
- Aneurysms
- Transient loss of consciousness: syncope vs. seizure
- Use of the clinical laboratory in diagnosis of collapse
- How certain can we be of our clinical observations and assays?
- Incapacity and absence of consent
- Differential diagnosis of collapse
- Diagnosis of suspected poisoning
- Applied Anatomy and Imaging Practical (collapse)

Specifics for Collapse in GP clinical contact

Introduction

This is the final session for the Year Two students. It should help prepare them for the to move into Year 3 where they will be doing more work on examination and clinical skills and start to think more about what to do next and management plans.

As with the previous sessions:

- refer to the Year 2 GP handbook, which covers the information common to all sessions.
- use the attached “session plan” as a guide on how to use your time with your group

Allow time for:

- introductions (reflecting on any learning/action points from the previous session, headache)
- student-led interaction with patient(s), and examination of upper and lower limb examination practice

- Final session: Group reflections on learning in Year 2 primary care, individual feedback and students to complete feedback questionnaire

(Expert) patients

Suitable patients for the block are people:

- with a history of syncope or transient loss of consciousness from any cause
- with a history of epilepsy
- with a previous TIA or stroke

and/or anyone willing to let students practice their examinations.

If possible, get someone to accompany the patient who has witnessed the episode.

Tasks

Start by assessing their learning needs:

- discuss the students' learning during the collapse block
- what do they feel confident in and what are they unsure about?

Prepare for the session. Brainstorm:

- What are the common causes of collapse, how to assess and differentiate between causes of collapse. What specific areas of the history are important?
- Get the students to think about what sorts of symptoms patients present with for each of the major causes (TIA/stroke, cardiac arrhythmias, epilepsy) and to have a think about how they might elicit that information.
- As a GP, when you find out the next person has "collapsed" how do you prepare? What do you need to do or know before you phone the patient or call them in? What information is particularly useful and why?
- Are there any key risk factors for types of collapse that you need to know about before you assess the patient.

Follow the usual timetable of talking to and examining one or two patients:

- Make sure students know how to measure pulse, manual BP and temperature.
- Practice how to perform upper and lower limb neurological examination.
- If time allows, revise cranial nerve examination (covered in the last clinical contact session).

Information given to students

Differential diagnosis of transient loss of consciousness

The below notes are based on Chapter 21: Transient loss of consciousness, Macleod's Clinical Diagnosis.

Transient loss of consciousness can be diagnostically challenging because:

- the event has usually resolved by the time of assessment; and
- critical elements of the history are unknown to the patient

Therefore, witness accounts are crucial.

Common causes of transient loss of consciousness include syncope, cardiac arrhythmias, epileptic seizure, non-epileptic attack disorders.

Table: Features which help discriminate syncope from seizure ((from Macleod's Clinical Examination)

	Syncope	**Seizure**
Triggers	Typically present (pain, illness, emotion)	Often none (sleep deprivation, alcohol, drugs)
Prodrome	Feeling faint, nausea, tinnitus, vision dimming	Focal onset (not always present)
Duration of unconsciousness	<60 seconds	1-2 minutes
Convulsion	May occur but brief myotonic jerks	Usual, tonic-clonic 1-2 minutes
Colour	Pale	Red/blue, may be pale
Tongue button	Very rare	Common
Recovery	Rapid, no confusion	Gradual, over 30 minutes, often confused, amnesic

Syncope

There are four subtypes of syncope:

- Reflex – vasodilatation and/or bradycardia occurs in response to a particular trigger, e.g. 'vasovagal' or 'carotid sinus' syncope. A clear precipitating trigger, e.g. intense emotion, venepuncture or prolonged standing (vasovagal syncope) or coughing, sneezing or micturition (situational syncope), together with typical prodromal symptoms, strongly suggests reflex syncope
- Orthostatic – due to failure of homeostatic maintenance of BP on standing, e.g. caused by antihypertensive medications or autonomic neuropathy.
- Arrhythmia-related – due to transient compromise of cardiac output by a tachy- or bradyarrhythmia, e.g. ventricular tachycardia, complete heart block.
- Cardiac – due to structural heart disease, especially left ventricular outflow obstruction, e.g. severe aortic stenosis, hypertrophic obstructive cardiomyopathy.

Acute pulmonary embolism (PE) or aortic dissection may also cause syncope.

Seizure

A generalised seizure (disordered electrical activity affecting the whole brain) can lead to transient loss of consciousness. Partial seizures may cause altered consciousness (complex partial seizures) without transient loss of consciousness.

Hypoglycaemia

Most cases are iatrogenic, from treatment of diabetes with insulin or sulphonylurea drugs. Causes of 'spontaneous' hypoglycaemia include alcohol, liver failure, insulinoma and adrenal insufficiency.

Impairment of consciousness promptly resolves with correction of capillary blood glucose.

Functional disorders (apparent transient loss of consciousness)

'Pseudoseizure' and 'pseudosyncope' are terms used to describe episodes that resemble seizure and syncope respectively but do not have an underlying somatic mechanism, i.e. no epileptiform activity or cerebral hypoperfusion.

Other causes

Other causes of collapse include cataplexy, narcolepsy, medication, alcohol, or drug intoxication.

History

If witnessed, ask the observer as well as the patient to describe attacks.

- Was there any warning? Faintness, blurring of vision, dizziness or nausea?
- Were there any associated symptoms? Palpitations or sensory symptoms?
- What were they doing at the time? For recurrent episodes, are there any precipitants, e.g. from sitting to standing.
- Did they lose consciousness and if so, for how long?
- Were there any convulsions? Any tongue biting or urinary/faecal incontinence?
- Was there any injury? e.g. from falling to the ground.
- How long did it take them to come around (regain consciousness)? How did they feel afterwards?

Sensory symptoms are common, and it is important to discern what the patient is describing. Clarify that, by 'numbness', the patient means lack of sensation rather than weakness or clumsiness.

Assessing a possible first seizure

The following notes are from NICE Clinical Knowledge Summary on Epilepsy.

An epileptic seizure is the transient occurrence of signs or symptoms due to abnormal electrical activity in the brain. This manifests itself as a disturbance of consciousness, behaviour, emotion, motor function, or sensation.

It is not possible to diagnose epilepsy after a single seizure. There are many other causes for a first seizure (see below).

Epilepsy is a disease of the brain defined by at least two unprovoked seizures occurring more than 24 hours apart.

Risk factors that suggest epilepsy

- Triggers for seizure – on waking or in association with sleep deprivation or flashing lights
- Family history of epilepsy
- Comorbid conditions (cerebrovascular disease, cerebral tumours etc etc)

Any symptoms of auras?

- Simple partial seizures with no loss of consciousness
- Auras arising from the temporal lobe: unexpected tastes, smells, paraesthesia, or a rising abdominal sensation

Specific features of generalised seizures

- **Tonic** seizures that cause impairment of consciousness and stiffening; the trunk may be either straight or flexed at the waist.
- **Clonic** seizures that cause jerking and impairment of consciousness.
- **Tonic–clonic** seizures that cause stiffening and jerking and impairment of consciousness.
- **Atonic** seizures that cause sudden brief attacks of loss of tone, associated with falls and impairment of consciousness.
- **Post-ictal period**: residual symptoms after the attack, such as drowsiness, amnesia, headache, or focal neurological deficit that slowly recovers.
- Injuries may be sustained, including aching limbs and bites to the tongue.

Assessing a possible TIA or stroke

The following notes are from NICE CKS Stroke and TIA.

Stoke and TIA present with sudden onset of focal neurological deficits. Widespread cerebral hypoperfusion (for example subarachnoid or intracranial haemorrhage or massive infarction) may present with non-focal or global neurological deficits.

Suspect a TIA if:

- The person presents with sudden onset, focal neurological deficit e.g.
  - Unilateral weakness or sensory loss.
  - Dysphasia.
  - Ataxia, vertigo, or incoordination. (Isolated dizziness is not usually a symptom of TIA)
  - Syncope.
  - Sudden transient loss of vision in one eye (amaurosis fugax).
  - Homonymous hemianopia.
  - Cranial nerve defects.
- Resolve within 24 hours and can't be explained by an alternative cause e.g. hypoglycaemia

Suspect stroke if:

- The person presents with sudden onset, focal neurological deficit which is ongoing (or lasted more than 24 hours)
  - Any of the above but acute presentation or persisting.
  - Confusion, altered level of consciousness and coma.

FAST warning signs

FAST (Face, Arms, Speech, Time) is an acronym to help people identify the most common signs of a stroke, and emphasises the importance of acting quickly by calling 999

- F = Face Drooping – Does one side of the face droop or is it numb? Ask the person to smile. Is the person's smile uneven?
- A = Arm Weakness – Is one arm weak or numb? Ask the person to raise both arms. Does one arm drift downward?
- S = Speech Difficulty – Is speech slurred?
- T = Time to call 999

Other stroke symptoms – sudden onset of:

- NUMBNESS or weakness of face, arm, or leg, especially on one side of the body
- CONFUSION, trouble speaking or understanding speech
- TROUBLE SEEING in one or both eyes
- TROUBLE WALKING, dizziness, loss of balance or coordination
- SEVERE HEADACHE with no known cause

Examination

The below notes are based on Chapter 21: Transient loss of consciousness, Macleod's Clinical Diagnosis.

For anyone with collapse, examination should focus on the cardiovascular and neurological systems.

The aim of the peripheral neurological examination is to identify if there is a an upper or motor neuron lesion; unilateral or bilateral; pyramidal, proximal or distal.

The standard scheme of examining limbs and trunk in a neurological examination is:

- Motor
    - Inspection
    - Tone
- Power
- Reflexes
- Coordination
- Sensory

Motor system

Assess skin, posture, muscle bulk and look for abnormal movements and fasciculations.

- Skin, e.g. rash of herpes zoster, neurofibromatosis (café au lait spots, peripheral nerve tumours)
- Abnormal posture, e.g. in an upper motor neurone lesion with flexion of the upper limb - arm pronated and adducted - and extension of the lower limb
- Muscle wasting indicates primary muscle disease, denervated muscle, or disuse atrophy. Compare both sides and look for patterns, e.g. proximal, distal, generalised, symmetrical
- Abnormal movements, e.g. athetosis or tremor

Assess tone.

- Ask the patient to lie supine on the examination couch and to relax and 'go floppy.' Enquire about any pain or limitations of movement.
- Passively move each joint to be tested through as full a range as possible, both slowly and quickly in all anatomically possible directions. It may help to distract the patient by asking them to count backwards from 20.
- Place a hand under one knee and flick it upwards to cause flexion. In a relaxed patient this should occur without resistance. A spastic leg will remain straight and land back on the bed on the heel of the foot.

Look for:

- hypertonicity - upper motor neurone or extrapyramidal lesion. Is it clasp knife i.e. spastic hypertonicity, or is it lead pipe – i.e. parkinsonian?
- hypotonicity - lower motor neurone lesion
- cog wheeling (periodic resistance to passive movement, felt in a parkinsonian limb)

Checking for patellar and ankle clonus

- Patellar clonus is tested by resting the hand on the lower part of the quadriceps and moving the patella down sharply. Patellar clonus is seen as sustained rhythmical contraction of the quadriceps.
- Ankle clonus is tested for by sharply dorsiflexing the foot with the knee bent to 90-degree and the hip externally rotated. Clonus is felt as repeated beats of dorsiflexion/plantar flexion.

Power

Power is tested by your ability to overcome the patient's voluntary resistance. Normal power is dependent on the patient's age, sex and build.

When assessing power:

- consider if there is a pattern to any weakness – proximal, distal or symmetrical
- take into consideration whether any painful joint or muscle disease is interfering with assessment

The Medical Research Council's power grading scheme is:

0. no visible muscle contraction (complete paralysis)
1. flicker of contraction but no movement
2. movement is possible when gravity is excluded
3. movement is possible against gravity
4. movement is possible against gravity and some resistance (but weaker than normal)
5. normal power

Plantar flexion and dorsiflexion can be tested by the patient walking on tip toes (impossible with S1 lesion) and on their heels (impossible if L4/L5 lesion).

Reflexes

A tendon reflex is the involuntary contraction of a muscle in response to stretch.

- It is mediated by a reflex arc consisting of an afferent (sensory) and an efferent (motor) neuron with one synapse between (a monosynaptic reflex).
- The most important reflexes are the deep tendon and plantar responses.

Dermatomal involvement may further help localise a lesion; for example, pain going down one leg with an absent ankle jerk (S1) and sensory loss on the sole of the foot (S1 dermatome) localises to the S1 root, most commonly due to a prolapsed intervertebral disc (sciatica).

Compare each reflex with the other side; check for symmetry of response.

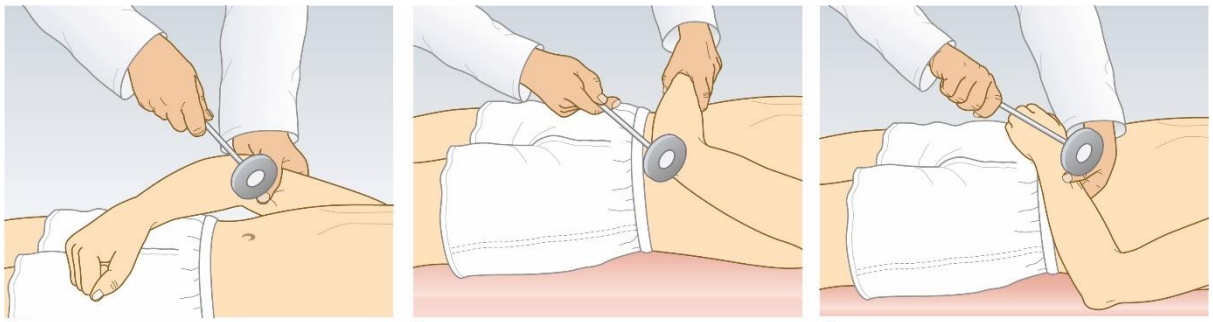
Record the response as:

- increased (+++)
- normal (++)
- decreased (+)
- present only with reinforcement (+/-)
- absent (-)

Upper limb reflexes (figure 1) are:

- biceps C5, C6
- brachioradialis C6
- triceps C7
- finger flexion C8

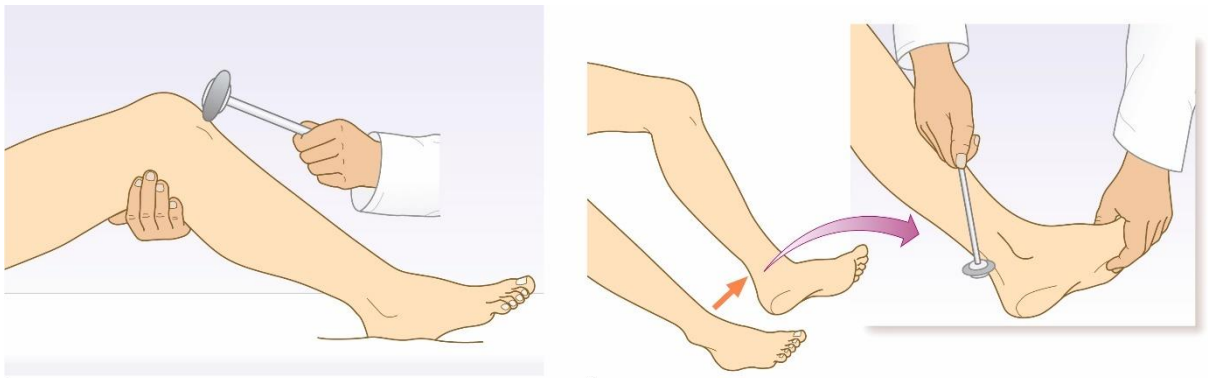
Figure 1: Assessing upper limb reflexes (from Macleod's Clinical Examination)



Lower limbs reflexes (figure 2) are:

- knee L3, L4
- hamstring L5, S1
- ankle (L5), S1
- plantar S1, S2

Figure 2: Lower limbs reflexes (from Macleod's Clinical Examination)



A B

Hyperreflexia (abnormally brisk reflexes) is a sign of upper motor neuron damage. Diminished or absent jerks are most commonly due to lower motor neuron lesions. In healthy older people, the ankle jerks may be reduced or lost.

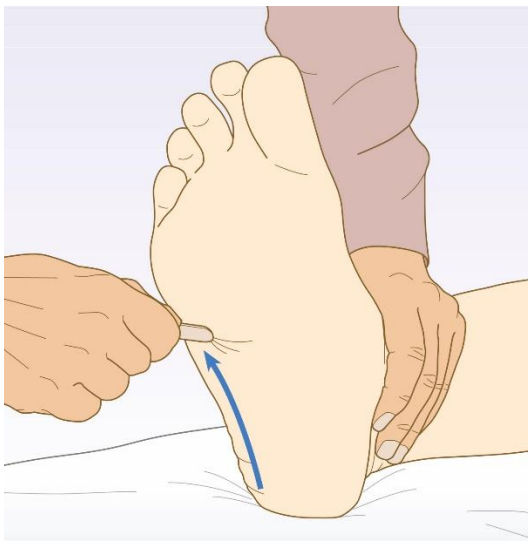
Plantar response (figure 3)

- Run a blunt object (orange stick) along the lateral border of the sole of the foot towards the little toe
- Watch both the first movement of the great toe and the other leg flexor muscles. The normal response is plantar flexion of the great toe (downward movement).

An extensor plantar response (Babinski sign), signifying an abnormal reflex due to an upper motor neuron lesion:

- involves activation of the extensor hallucis longus tendon (not movement of the entire foot, a common 'withdrawal' response to an unpleasant stimulus)
- coincides with contraction of other leg flexor muscles
- is reproducible.

Figure 3: Plantar reflex (from Macleod's Clinical Examination)



Coordination

The cerebellum coordinates muscle movements. Various tests are used to examine the patient's coordination.

Upper limb:

- Finger-nose test
- Rapid alternating movements

Lower limb:

- Assess gait
- Romberg's test
- Heel-shin test

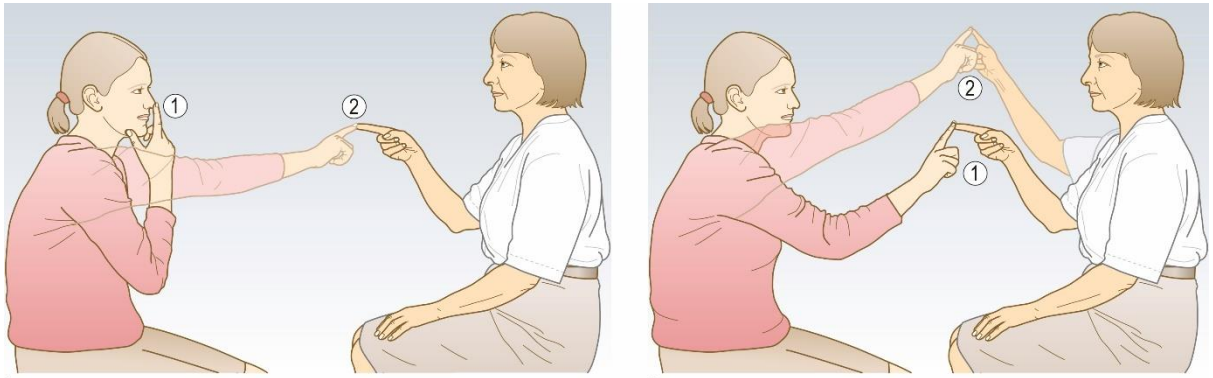
Finger-nose test assesses upper limb co-ordination.

See figure 4:

- Ask the patient to touch the tip of their nose (A1) and then your finger (A2).
- Move your finger from one position to another, towards and away from the patient (B 1), as well as from side to side (B2)

You are looking for intention tremor - no tremor at rest - and past-pointing - the patient's finger overshoots the target. These are signs of cerebellar disease.

Figure 4: Finger nose test (from Macleod's Clinical Examination)



A B

Rapidly alternating movements

Demonstrate repeatedly patting the palm of your hand with the palm and then the back of your opposite hand as quickly and regularly as possible. Ask the patient to copy your actions. Repeat with the opposite hand.

Dysdiadochokinesis (impairment of rapid alternating movements) is evident as slowness, disorganisation and irregularity of movement.

Gait

Gait is the manner of walking. Important points in its examination include:

- good visibility of the legs
- ask the patient to walk normally for about 5 metres, turn round and walk back
- ask the patient to walk heel-to-toe - testing for cerebellar disease

Romberg's test

The patient is asked to stand with the feet together. If the patient is steady with eyes open but unsteady with eyes closed then the test is positive.

Romberg's sign is said to be positive in patients with sensory ataxia and negative in cerebellar ataxia. In practise Romberg's sign has a low specificity.

Heel-shin test

In the heel shin test, the patient runs the sole of one foot up and down the shin of the opposite leg.

Sensation

The sensory system can be assessed for light touch, pin prick, cold, vibration, and joint position sense (proprioception).

Detailed examination of sensation is time-consuming and unnecessary unless the patient volunteers sensory symptoms or you suspect a specific pathology, such as spinal cord compression or mononeuropathy. In patients without sensory symptoms, assessing light touch only of all four limbs as a screening process may suffice.

Testing the spinothalamic tract

Pain and temperature sensation are carried by small, slow-conducting fibres of the peripheral nerves and the spinothalamic tract of the spinal cord. They enter the spinal cord and cross to the opposite spinothalamic tract a few segments up. The tract then ascends to the brainstem.

Pain testing:

- Using a new pin, the sharpness of the pin is demonstrated to the patient, e.g. by gently touching his anterior chest wall.
- The limbs (proximal to distal) are then tested by touching the patient with a pin in a position approximating to a dermatome (comparing right and left) and asking whether the patient feels the pin as sharp or dull. Pain testing may also be used to ascertain a spinal level of a lesion that affects the trunk.

Temperature testing is not routinely undertaken. The procedure involves using test tubes filled with hot and cold water and testing in a similar pattern to pain testing.

Testing the posterior columns

Proprioception and vibration are conveyed in large, myelinated fast-conducting fibres in the peripheral nerves and in the posterior (dorsal) columns of the spinal cord. The posterior column remains ipsilateral from the point of entry up to the medulla.

Light touch

- Tested by gently touching (not stroking) the skin in each dermatome (figure 5) with cotton wool.
- The patient closes his eyes and asked to say 'yes' when he feels the cotton wool touching.

Some fibres for light touch travel in the posterior columns - ipsilateral - and some in the anterior spinothalamic tract - contralateral. Thus testing this carries the least discriminatory information.

Proprioception

- Hold a distal phalanx and move it slightly upwards and then slightly downwards, whilst telling the patient what movements are being undertaken.
- Tell the patient to close their eyes and repeat the movements, asking each time the patient is asked to say whether the phalanx is going 'up' or 'down'.

Vibration testing

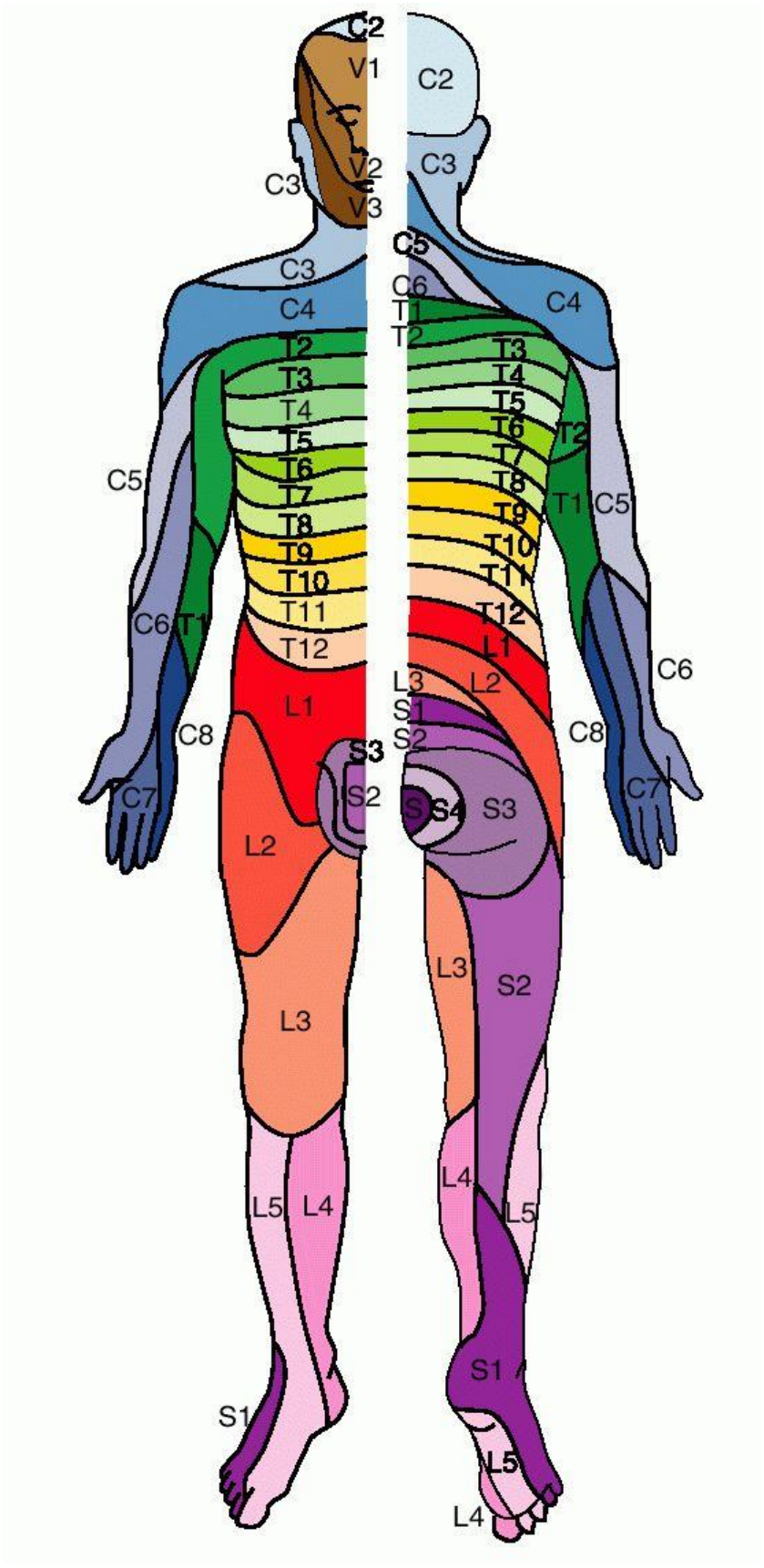
- Ask the patient to close their eyes and place a vibrating 128 Hz tuning fork on a bony surface, e.g. the ulna at the wrist
- Stop the vibrations from the tuning fork.
- The patient should be able to identify when the tuning fork has stopped vibrating.

Investigation

Not all patients require investigation. Avoid doing tests because you can or because you do not know what else to do; further investigations should be guided by the history and physical examination.

Magnetic resonance imaging (MRI) of the brain may unearth incidental findings of no clinical relevance in up to 20%, depending on age, and there is an irony – usually lost on your patient – in attempting reassurance with a scan only to identify an incidental ‘abnormality.’

Figure 5: Sensory dermatomes



Resources

Core reading is available via library textbooks: Macleod’s Clinical diagnosis and Clinical examination.

Geeky medics Upper limb neurological examination

Geeky medics Lower limb neurological examination

Video of Romberg's test