Introduction to neurology

Manifestations of neurological diseases 1 & 2

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The aim of the history is to address two key issues:

Where is the lesion and What is the lesion.

The key diagnostic questions

Is it neurological ? // If so, to which part of the nervous system does it localise

Central versus peripheral // Sensory versus motor versus both

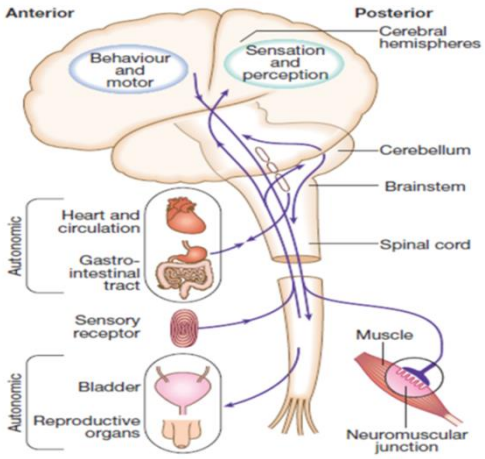
What is the lesion ?

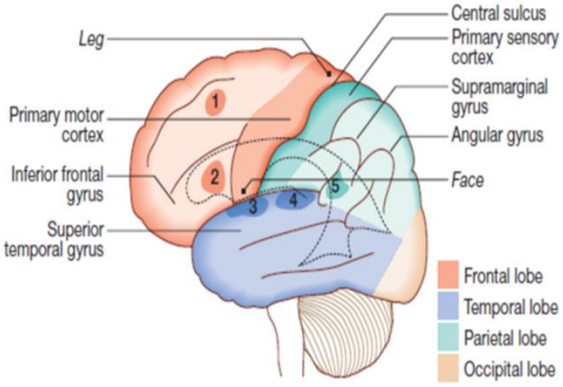
Hereditary or congenital

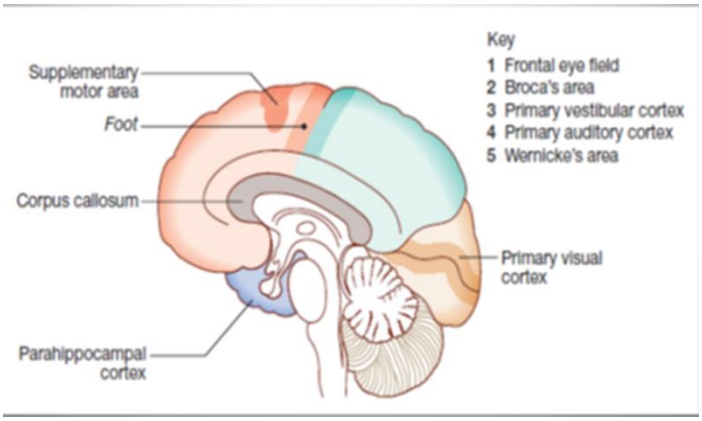
Acquired

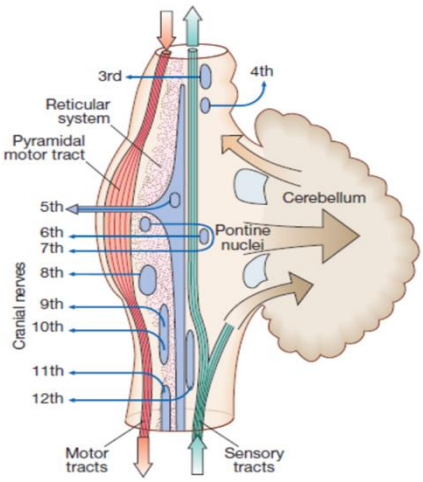
Traumatic / Infective / Neoplastic / Degenerative / Inflammatory or immune-mediated / Vascular / Drug- induced / Nutritional / Toxins / Functional

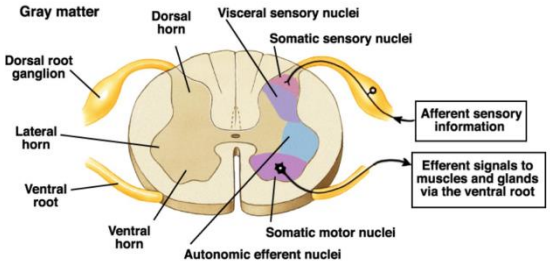
The major anatomical components of the nervous system

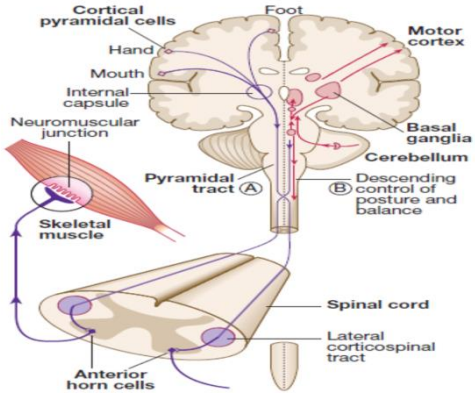












Introduction

- Age and sex
- Handedness

Presenting complaint

- Symptoms (clarify: see text)
- Overall pattern: intermittent or persistent?
- If intermittent, how often do symptoms occur and how long do they last?
- Speed of onset: seconds, minutes, hours, days, weeks, months, years, decades?
- Better, worse or the same over time?
- Associated symptoms (including non-neurological)
- Disability caused by symptoms
- Change in walking
- Difficulty with fine hand movements, e.g. writing, fastening buttons, using cutlery
- Effect on work, family life and leisure

Background

- Previous neurological symptoms and whether similar to current symptoms
- Previous medical history
- Domestic situation
- Driving licence status
- Medications (current and at time of symptom onset)
- Alcohol/smoking habits
- Recreational drug and other toxin exposure
- Family history and developmental history
- What are patient's thoughts/fears/concerns?

Determining the evolution, speed of onset and progression of a disease is important

Onset	Evolution	Possible causes
Sudden (minutes to hours)	Stable/improvement	Vascular (stroke/transient ischaemic attack (TIA)) Nerve entrapment syndromes Functional
Gradual	Progressive over days	Demyelination Infection
Gradual	Progressive over weeks to months	Neoplastic/paraneoplastic
Gradual	Progressive over months to years	Genetic Degenerative

The impact on day-to-day activities, such as walking, climbing stairs and carrying out fine hand movements, should also be established in order to gauge the level of associated disability.

Estimates of the frequency and duration of specific events are essential when taking details of a paroxysmal disorder such as migraine and epilepsy. Vague terms such as ‘a lot’ or ‘sometimes’ are unhelpful, and it can assist the patient if choices are given to estimate numbers, such as once a day, week or month.

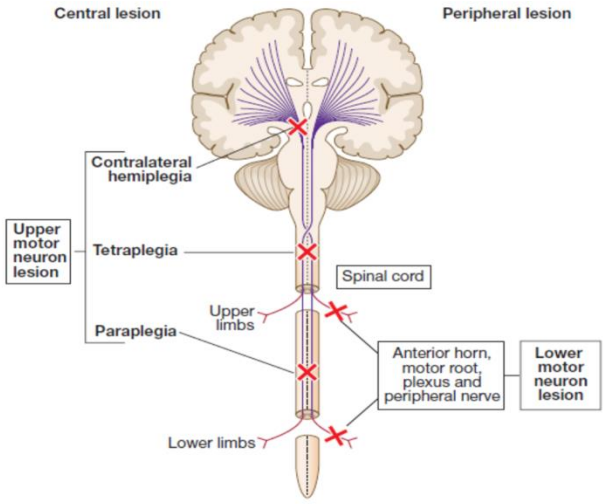
Cardinal symptoms

Headache and facial pain

Most headaches are chronic disorders but acute presentation of headaches is an important aspect of emergency medical care. Headache may be divided into primary (benign) or secondary. Site / Character / Severity / Duration / Triggers …… etc.

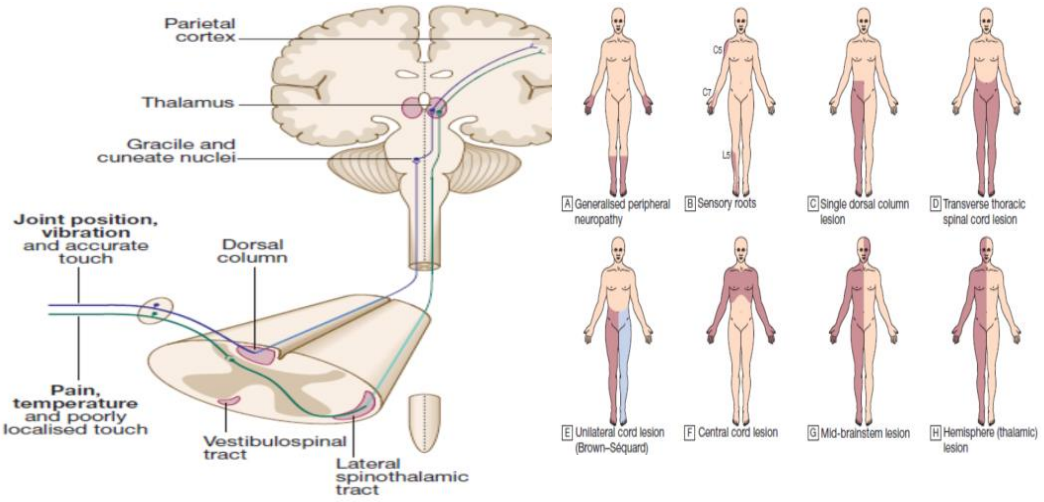
Weakness (Paresis) // Paralysis (Plegia) Hemi – Mono – Para - Tetra

Patterns of motor loss according to the anatomical site of the lesion



	Upper motor neuron lesion	Lower motor neuron lesion
Inspection	Normal (may be wasting in chronic lesions)	Wasting, fasciculation
Tone	Increased with clonus	Normal or decreased, no clonus
Pattern of weakness	Preferentially affects extensors in arms, flexors in leg Hemiparesis, paraparesis or tetraparesis	Typically focal, in distribution of nerve root or peripheral nerve, with associated sensory changes
Deep tendon reflexes	Increased	Decreased/absent
Plantar response	Extensor (Babinski sign)	Flexor

Sensory disturbance : Numbness and paraesthesia



Altered balance and vertigo

Balance is a complicated dynamic process that requires ongoing modification of both axial and limb muscles to compensate for the effects of gravity and alterations in body position and load (and hence (centre of gravity) in order to prevent a person from falling.

This requires input from a variety of sensory modalities (visual, vestibular and proprioceptive), processing by the cerebellum and brainstem.

The patient may complain of different symptoms, depending on the location of the lesion.

For example, loss of joint position sense or cerebellar function may while damage to the vestibular result in a sensation of unsteadiness nuclei or labyrinth may result in an illusion of movement, such as vertigo.

A careful history is vital

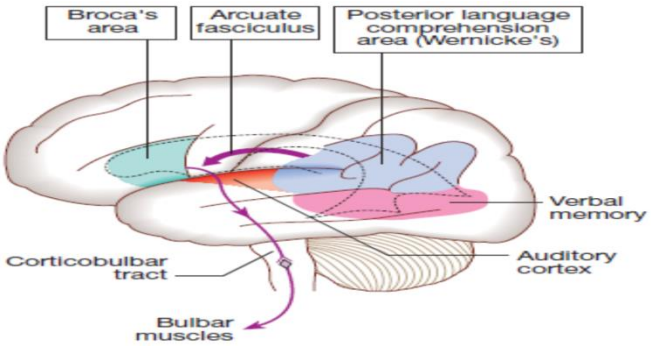
Since vision can often compensate for lack of joint position sense, patients with peripheral neuropathies of dorsal column loss will often find their problem more noticeable in the dark.

Vertigo is defined as an abnormal perception of movement of the environment or self, and occurs because of conflicting visual,

proprioceptive and vestibular information about a person's position in space.

Abnormal speech

Areas of the cerebral cortex involved in the generation of spoken language



Dysarthria

Dysarthria is slurred speech caused by articulation problems due to a motor deficit.

Disturbed articulation may result from lesions of the tongue, lips or mouth, ill-fitting dentures or disruption of the neuromuscular pathways.

Cerebellar dysarthria may be slow and slurred, similar to alcohol intoxication.

Myasthenia gravis is the most common cause of fatiguing speech.

Parkinsonism may cause dysarthria and dysphonia with a low-volume, monotonous voice in which the words run into each other.

Dysphonia

Dysphonia is loss of volume caused by laryngeal disorders.

This usually results from either vocal cord pathology, as in laryngitis, or damage to the vagal (X) nerve supply to the vocal cords (recurrent laryngeal nerve). Inability to abduct one of the vocal cords leads to a 'bovine' (and ineffective) cough.

Dysphasia

Expressive (motor) dysphasia results from damage to Broca's area. It is characterised by reduced verbal output with non-fluent speech and errors of grammar and syntax. Comprehension is intact.

Receptive (sensory) dysphasia occurs with dysfunction in Wernicke's area. There is poor comprehension, and although speech is fluent, it may be meaningless and contain paraphasias (incorrect words) and neologisms (nonsense or meaningless new words).

Global dysphasia is a combination of expressive and receptive difficulties due to involvement of both areas.

Dysphasia (a focal sign) is frequently misdiagnosed as confusion (non-focal sign). Always consider dysphasia before assuming confusion, as this fundamentally alters the differential diagnosis and investigation plan.

Dominant parietal lobe lesions affecting the supramarginal gyrus may cause dyslexia (difficulty comprehending written language), dyscalculia (problems with simple addition and subtraction) and dysgraphia (impairment of writing).

Disturbance of consciousness

Neurological or not // Continuous or episodic (attacks)

Transient loss of consciousness

If patients are unaware of their symptoms, obtain a witness account. This is more valuable than an unfocused neurological examination. Ask the witness about symptoms before, during and after the TLOC – were

there any warning symptoms, any colour changes, did the patient lie still or move, what was the patient like immediately afterwards ?

Fits (Epilepsy) vs Syncope

	Vasovagal 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	Less than 60 seconds	1–2 minutes
Convulsion	May occur but brief myoclonic jerks	Usual, tonic-clonic 1–2 minutes
Colour	Pale/grey	Red/blue, may be pale
Lateral tongue biting	Very rare (may bite tip)	Common
Recovery	Rapid, no confusion	Gradual, over 30 minutes, often confused, amnesic

Grading of level of consciousness

GCS //Drowsy // Stuporous // Confused // Delerious // Unconscious

Memory disturbance (Amnesia)

Transient global amnesia // Persistent amnesia

Other symptoms

Visual symptoms // Visual acuity // Double vision // Hearing // Swallowing // Bladder dysfunction // Sexual dysfunction