Case-taking proforma

History

- Chief complaint
- History of presenting illness
    - Weakness- same format as hemiplegia
        - Progressive weakness (increasing in severity and extent)- more likely to be cord compression
        - Improving weakness- inflammatory, acute transverse myelitis,
        - Waxing and waning- MS
    - Sensory involvement
        - Radicular pain- sharp shooting severe dermatomal pain radiating down a limb or around the trunk. Exacerbated by conditions that increase the pressure in the spinal cord like coughing, sneezing, straining etc
        - Spintothalamic tract- Poorly localised aching pain
        - Dorsal column-
            - **Wash basin sign**- unsteadiness on closing eyes to wash face
            - Difficulty in walking at night
            - Constricting band like sensation
    - Autonomic involvement
        - Bladder
            - Urgency, frequency, precipitancy- UMN
            - Hesitancy, poor stream, retention of urine and overflow incontinence- LMN
            - Can patient feel the bladder filling?
            - Catheterisation done?
            - Credes manoeuvre- pressing the lower abdomen to pass urine
        - Bowel
            - Constipation/incontinence
    - Cranial nerves
        - VII- GBS
        - II- Optic neuritis, colour vision disturbance- MS. Optic atrophy in B12 deficiency, Neuromyelitis optica
        - Opthalmoplegia- Miller Fischer
    - History for aetiology
        - h/o cough with expectoration, weight loss, night sweats, evening rise of temp- Pott's spine
        - h/o recent URI/diarrhoea- GBS, hypokalaemia
        - h/o trauma
        - lower urinary tract symptoms (in elderly male)- Ca prostate
        - breast lump in elderly female
        - cough, haemoptysis, weight loss- Ca lung
        - h/o dog bite and vaccination- rabies
    - Personal history
        - Smoking for Ca lung- quantify pack years
        - Alcohol for B12 deficiency
        - Veg or non veg for B12 deficiency
    - Past history
        - TB and contact with TB
    - Family history
        - Hereditary spastic paraplegia

Examination

Neurocutaneous markers- be ready for questions on the diseases

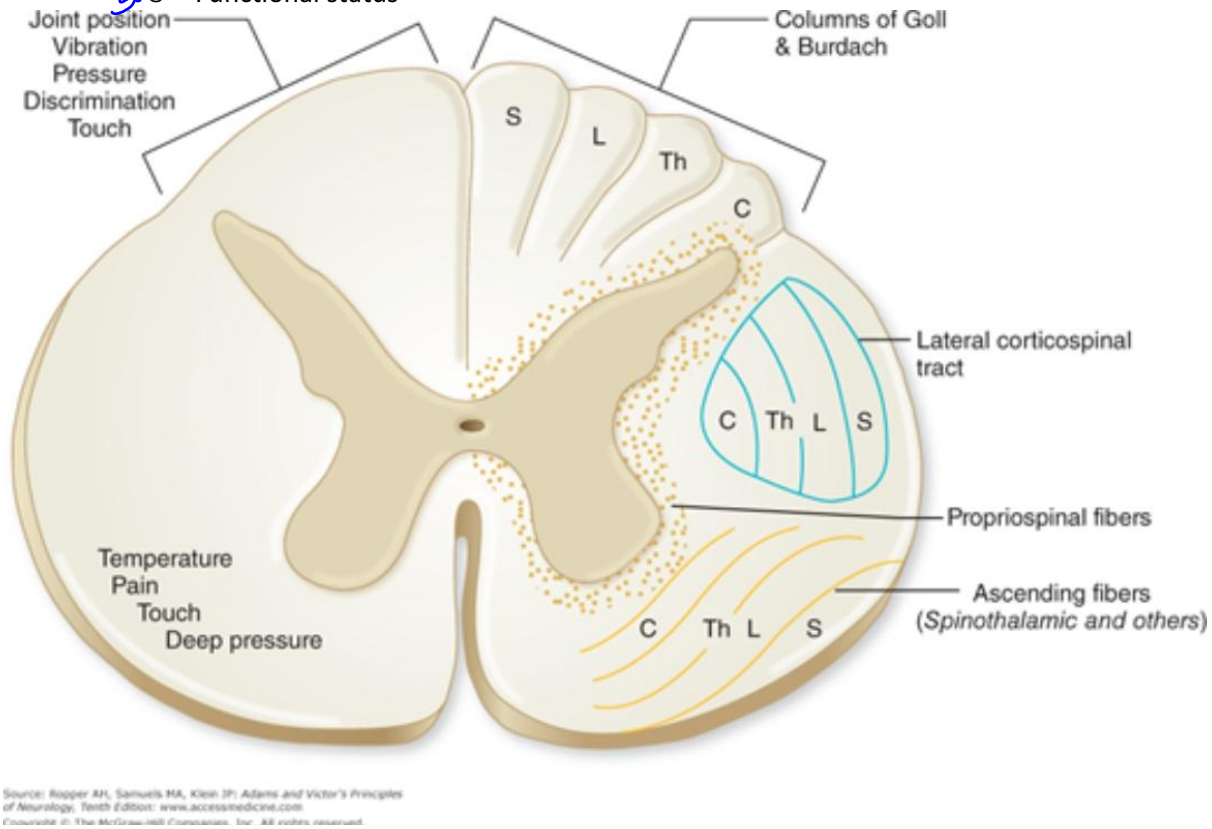
- Pallor- B12 deficiency
- Lymphadenopathy- TB/mets

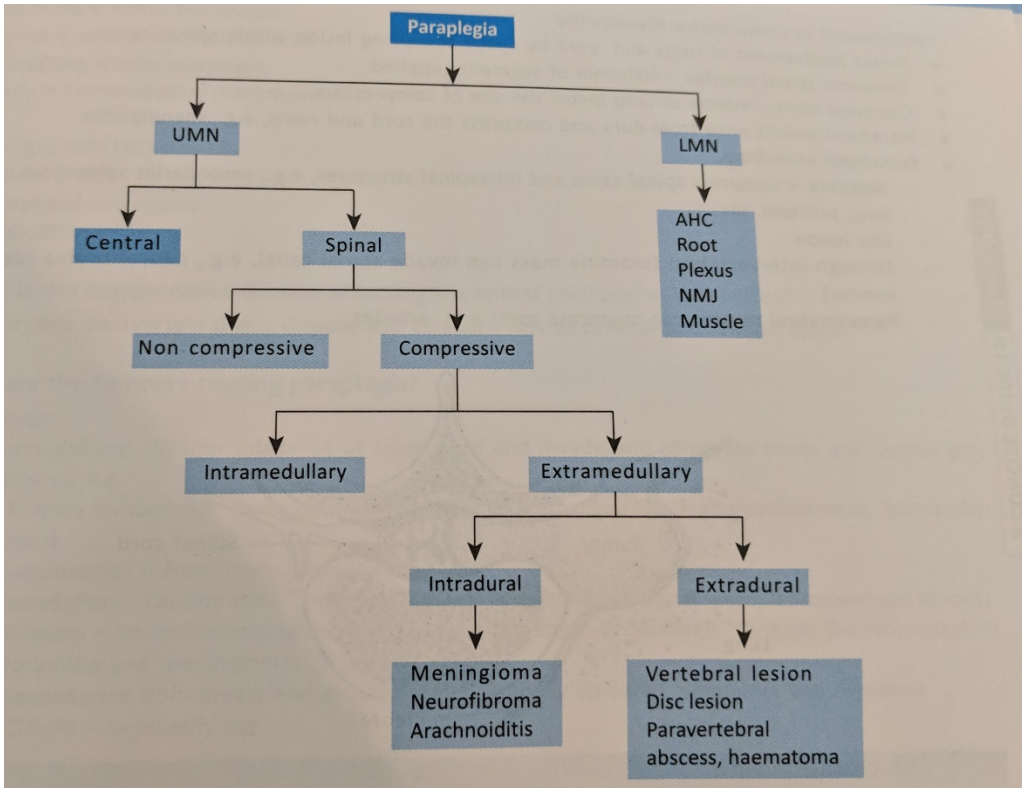
Spine examination

- Gibbus
- Kyphosis
- Scoliosis
- Tuft of hair
- Tenderness over spine

Diagnosis

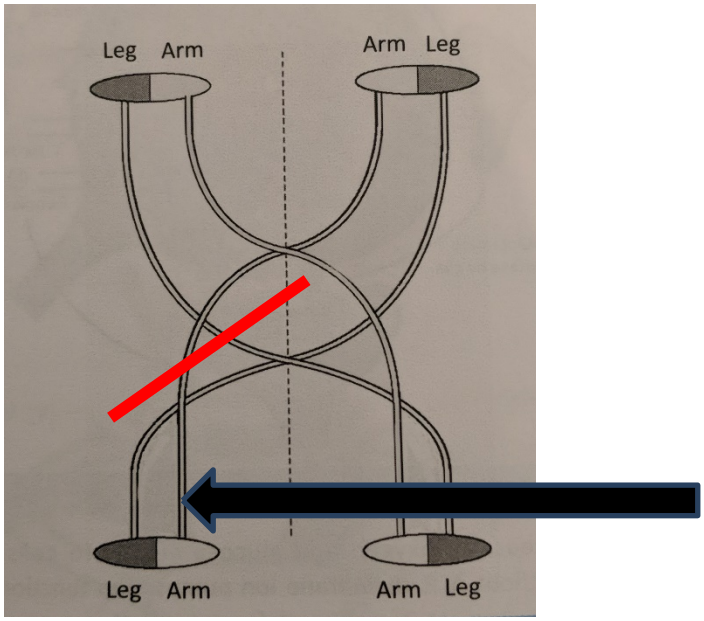
- Paraparesis/paraplegia
- Anatomical level
    - 1. Vertical- Sensory, motor, reflex
    - 2. Horizontal- extradural/extramedullary/intramedullary
    - 3. Pathological- Compressive/Vascular/Infective/Demyelinating
    - 4. Aetiology
    - 5. Functional status





- UMN causes of paraplegia
    - Central
        - Parasagittal meningioma
        - Trauma at parasagittal area
        - Thrombosis
            - Superior sagittal sinus thrombosis
            - Thrombus of unpaired ACA
            - Bilateral ACA thrombosis
    - Spinal
        - Compressive
            - Intramedullary
            - Extramedullary
        - Non compressive
            - Traumatic
            - Post infectious
            - MS, NMO
            - Post vaccinial**- Polio, tetanus, rabies vaccines
            - Familial- Motor neuron disease, Familial spastic paraplegia, Spinocerebellar degeneration
            - Nutritional- B12, Copper deficiency
            - Lathyrism
            - Systemic diseases
            - Paraneoplastic

Cruciate hemiplegia and Elsberg phenomenon



- Pyramidal tract decussates at the cervicomedullary junction
- Arm fibres cross before leg fibres
- Cruciate hemiplegia- controversy
    - https://www.nature.com/articles/3101861
    - Multiple journal articles say ipsilateral UL and contralateral LL because the site of the lesion is between the crossing of UL and LL fibres, where the UL fibres have already crossed
    - Alagappan says contralateral UL and ipsilateral LL which cannot be localised anywhere in the diagram
- Elsberg
        - 1. Ipsilateral arm fibres (black arrow)
        - 2. Ipsilateral leg fibres
        - 3. Contralateral leg fibres
        - 4. Contralateral arm fibres

Table I: Various causes of cruciate paralysis described[1-3,7,8,13-30]	
Traumatic injury	Atlanto-occipital dislocation Jefferson fracture Odontoid peg fracture (most common) C2/C3 fractures and dislocations SCIWORA Penetrating injury, i.e. gunshot
Disc herniation	C2/3 disc herniation
Congenital anomalies	Congenital anomalies of C1 and C2 with atlanto-axial instability Chiari malformations ± syringomyelia
Tumour or infection	Spinal metastasis Odontoid tuberculoma
Rheumatoid arthritis	
Complications of surgery to cervicomedullary junction	

	Feature	Compressive myelopathy	Non compressive
1.	Bony pain	Present	Absent
2.	Bony tenderness	Present	Absent
3.	Girdle like sensation	Present	Absent
4.	Upper level of sensory loss	Present	Absent
5.	Zone of hyperaesthesia	Present	Absent
6.	Root pain	Present	Absent
7	Onset and progression	Gradual onset	Can be acute
8	Symmetry	Asymmetrical	Usually symmetrical
9	Flexor spasm	Common	Usually absent
10	Pattern of neurodeficit	U shaped (in cervicomedullary lesions)	Bilaterally symmetrical
11	Bowel and bladder involvement	Early	Late
12.	Classical example	Pott's spine	MND

Determining the level of the lesion

- The presence of a horizontally defined level below which sensory, motor and autonomic function is impaired is a hallmark of a spinal cord lesion
- Sensory loss below the level of the lesion is due to damage to the spinothalamic tract on the **opposite side, one or two segments higher in case of a unilateral lesion and at the level of a bilateral lesion**. This is because the second order sensory fibres of the STT ascend for one or two levels as they cross anterior to the central canal to join the opposite STT.
- Lesions that transect the descending CST and other motor tracts cause paraplegia or quadriplegia with heightened DTR, Babinksi and eventual spasticity
- Transverse damage to the cord also produces autonomic disturbances consisting of absent sweating below the implicated cord level and bowel, bladder and sexual dysfunction

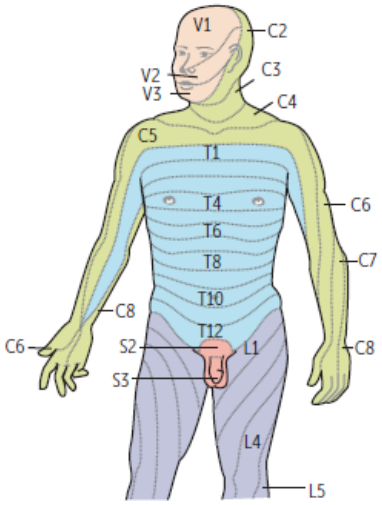
- A band of **altered sensation** (hyperalgesia or hyerpathia) at the upper end of the sensory disturbance, **fasciculations, atrophy** of the muscles and muted or absent DTR may be noted
- Cervical cord:
    - Quadriplegia and weakness at the diaphragm
    - Horner's syndrome may accompany a cervical cord lesion at any level
    - Loss of biceps jerk, finger and wrist extensors and triceps jerk
- Thoracic cord
    - Localised by sensory level on the trunk, and if, present, by the site of midline back pain
    - Nipples- T4
    - Umbilicus- T10
    - **Beevor's sign**- T9-T10 lesions paralyse the lower, but not the upper, abdominal muscles resulting in an upward movement of the umbilicus when the abdominal muscles contract
- Lumbar cord
    - Knee jerk, Ankle jerk - L_3L_4 - S_1S_2
    - Hip movements
- Sacral cord/Conus medullaris- refer conus medullaris syndrome

Spastic vs flaccid paraplegia

Legend	Spastic (UMN)	Flaccid (LMN)
History	Feeling of tightness	Looseness
Examination – bulk	Minimal wasting	Wasting present
Tone	Increased	Decreased
Fasciculation	Absent	Present
Superficial reflex	Lost	Lost
Deep tendon reflex	Exaggerated	Lost
Plantar reflex	Extensor plantar response	Flexor or no response

Important dermatomes for localisation

Landmark dermatomes



C2—posterior half of the skull.
C3—high turtleneck shirt.
C4—low-collar shirt.
C6—includes thumbs.
T4—at the nipple.
T7—at the xiphoid process.
T10—at the umbilicus (important for early appendicitis pain referral).
L1—at the inguinal ligament.
L4—includes the kneecaps.
S2, S3, S4—erection and sensation of penile and anal zones.

Causes table

Duration	Cerebral	Non-compressive	Extradural extramedullary	Intradural extramedullary	Intramedullary
Acute	Unpaired ACA thrombosis, SSS thrombosis	Acute transverse myelitis	1. IV disc prolapse, 2. Epidural abscess	None	Haematomyelia
Chronic	Parasagittal meningioma	B12 deficiency, Hypocupric myelopathy, Frederich Ataxia, Lathyrism	Pott's spine	Neurofibroma	Ependymoma, Astrocytoma, Syringomyelia, Glioma

Approach to spastic paraplegia

- 5 T's- Trauma, TB, Thrombosis, Tumour, Transverse myelitis

- Can be gradual or sudden onset
- Gradual onset
    - Cerebral cause- Cerebral diplegia (Little's disease), Parasagittal meningioma
    - Spinal- Cord compression, non compressive (motor neurone disease, MS, SACD, Lathyrism, syringomyelia) ↳ Exception-GBS
- Sudden onset
    - Cerebral causes- Superior sagittal sinus thrombosis, Unpaired Anterior Cerebral Artery Thrombosis
    - Spinal causes- Compressive- PIVD, Non compressive- Acute transverse myelitis, Post vaccinal, thrombosis of anterior spinal artery
- Of two types, Paraplegia in flexion and paraplegia in extension

Feature	Paraplegia in extension	Paraplegia in flexion
Definition	Lower limbs take an extension attitude and extensor muscles are spastic	Lower limbs take a flexion attitude due to flexor spasm
Pathology	Only pyramidal tracts are involved	Pyramidal and extrapyramidal involvement
Evolution	Early	Late
Attitude	Hip extended, adducted Knee extended, feet plantarflexed	Thigh and knee flexed Feet dorsiflexed Lateral decubitus position in bed
Tone	Clasp-knife spasticity in extensor muscles	Tone increased in flexor muscles
Jerks	Brisk	Present but diminished
Plantar response	Extensor	Extensor, but associated with flexor spasm
Reflex evacuation of bowel and bladder	Absent	Present
Mass reflex	Absent	May be present

- Cerebral causes of paraplegia
    - h/o Jacksonian fits and cortical sensory loss
    - Spastic paraplegia in extension
    - Features of increased ICT
    - Bladder dysfunction common
    - Abnormalities in higher mental function, speech, cranial nerves

Flaccid paraplegia

- UMN in spinal shock
- Anterior horn cell lesion- Poliomyelitis, Non-polio enterovirus, progressive muscular atrophy
- Nerve root (radiculopathy)- GBS, Tabes dorsalis
- Peripheral neuropathy
- NMJ- Myasthenia, LEMS
- Muscle- myopathy, muscular dystrophy, hypokalaemic periodic paralysis

Compressive vs. Non-compressive

- Compressive lesions
    - Bony deformity
    - Bony tenderness
    - Upper level of sensory loss

    - Root pain
    - Zone of hyperaesthesia
    - Gradual onset
    - Asymmetrical
    - U shaped pattern of neurodeficit (Elsberg phenomenon/Around the clock phenomenon- Harrison's) Ipsilateral UL, Ipsilateral LL, Contralateral LL, Contralateral UL.
    - Early bowel and bladder involvement
- Transverse myelitis is a non-compressive pathology that behaves like compressive

Localisation

- The uppermost level of a spinal cord lesion can also be localised by segmental signs corresponding to disturbed motor or sensory innervation by an individual cord segment
    - Band of altered sensation- hyperalgesia
    - Atrophy or fasciculations of muscles supplied by that root
    - Absent or inverted DTR at that level
    - May be associated with a nerve disorder as well if the nerve shares that root value
- However, **none of this is reliable in spinal shock**
- Foramen magnum- atrophy of SCM, downbeat nystagmus, C2 sensory loss, Horner's syndrome, lower cranial nerve palsy
- **Hemiplegia cruciata**
    - Sign of a cervicomedullary junction lesion
- C2- suboccipital pain/sensory loss. **Descending tract of V nerve affected, so pain and temperature loss over face**. Loss of trapezius reflex
- C3- Loss of trapezius reflex
- C5- inverted biceps jerk, inverted supinator jerk, **sensory loss over deltoid** (regimental badge)
- C6- loss of or diminished biceps and supinator, exaggerated finger flexor
-
- Inverted reflexes
    - Inverted supinator jerk- brisk finger flexion instead of elbow flexion. Associated with an absent biceps jerk and exaggerated triceps jerk. Indicative of a lesion at C5 or C6. Occurs because LMN lesion of C5 is combined with a UMN lesion affecting reflexes below C5.
    - Inverted biceps jerk- extension of elbow instead of flexion. Indicates a lesion at C5
    - Inverted knee jerk- Flexion of the knee instead of extension. Indicates L3 or L4 lesion

Lesions

- Extramedullary (95%)
    - Intradural (15%)- Meningioma, neurofibroma, AV malformations
    - Extradural (80%)- Pott's spine, mets, PIVD, spondylolisthesis, trauma
- Intramedullary (5%)- glioma, ependymoma, chordoma, syringomyelia

Spinal shock

- Flaccid areflexic paralysis below the level of the lesion in the spinal cord after acute insult
- There may be urinary retention and constipation
- Plantar response may be equivocal or no response

Acute transverse myelitis features

- Often follows a viral illness or post-vaccinal
- Flaccid paraplegia. Onset is acute or subacute
- Fever may be present before paralysis develops
- Bladder (urinary retention) and bowel (unable to pass stool) involvement early

- Although it behaves like a compressive variety, there is absence of root pain, spinal tenderness, spinal deformity
- Back pain and progressive paraparesis are presenting complaints
- Plantar response is extensor and there is partial or complete sensory loss with a definite upper level (vs. GBS- flexor response, no demonstrable sensory loss)
- MRI to exclude cord compression
- Majority recover within 3 months

Interpretation of the history

- Root pain definition**- sharp, stabbing, constant shock like pain below the level of the lesion radiating to both limbs. Characteristic of extramedullary pathology
- Funicular pain- poorly localised burning pain. Characteristic of intramedullary pathology
- Intramedullary syndromes- Arise from within the substance of the cord
- Extramedullary syndromes- Lie outside the cord. Radicular pain is prominent, early sacral sensory loss, spastic weakness in the legs, incontinence
- Compressive myelopathies- Warning signs like back, neck pain, bladder disturbances and sensory symptoms precede the paralysis.
- Non-compressive myelopathies
- Extradural vs Intradural
- Causes of ascending paralysis
- Causes of descending paralysis

- **Positive sensations**: girdle like sensation, pins and needles
- **Negative sensations:** numbness
-

Extramedullary	Intramedullary
More common	Less common
Prominent root pain	Funicular pain
Early sacral sensory loss (sacral distribution in spinothalamic tract is close to the outer side .. Hence)	Sacral sparing
Early spastic weakness of legs (same reason)	Less pronounced spasticity and it's a late feature
Reflexes markedly brisk early	Less brisk late
Bowel bladder involvement is late (controversial point – harrisons says early but both kundu and alagappan mention late)	early

Extra dural	Intradural
More common	Less common
Long duration symptoms	Shorter duration
benign	malignant
Both sides root pain (symmetrical symptoms)	One side root pain (asymmetrical)

Conus medullaris syndrome

- Damage to the spinal cord segments S3-S5 (conus medullaris) which are situated at the level of L1 vertebra
- Aetiology
    - Spinal tumours
    - Trauma- vertebral fracture, spondylolisthesis
- Sudden bilateral onset
- Lower back pain
- Less severe radicular pain
- Symmetric, hyperreflexic distal paresis of the LL, but Ankle jerk may be absent (UMN+LMN)
- Symmetric bilateral perianal numbness (saddle block anaesthesia)
- Sensory dissociation
- Early onset of bowel and bladder incontinence
- Erectile dysfunction

Cauda equina syndrome

- Damage to or compression of the cauda equina with nerve fibres of L3-S5 (below L2)
- Aetiology
    - Disc herniation
    - Trauma
    - Tumours
- Gradual unilateral onset
- Severe radicular pain
- Asymmetric, areflexic, flaccid paresis of the legs
- Saddle block anaesthesia (may be asymmetric)
- Asymmetric, unilateral numbness and/or paraesthesia in LL dermatomes
- Late onset of urinary retention
- Change in bowel habits
- Decreased rectal tone or bulbocavernosus reflex
- Erectile dysfunction

Chronic myelopathies

- Spondylotic Myelopathy
- Vascular malformations
- Retroviral associated

- Syringomyelia
- Chronic myelopathy of MS
- SACD
- Hypocupric myelopathy
- Tabes
- Familial spastic paraplegia
- Adrenoleukodystrophy

Non compressive myelopathies

- Spinal cord infarction
- MS, NMO
- SLE, Sjogren associated
- Post infectious/post vaccination
- Acute infectious- EBV, CMV, HSV, Polio, Enteroviridae

Compressive myelopathies

- Neoplastic spinal cord compression
- Spinal epidural abscess
- Pott's spine
- Spinal epidural haematoma
- Haematomyelia

Viva

- Paraesthesia
- Causalgia
- Dysaesthesia
- Hyperaesthesia
- Hypaesthesia
- Anaesthesis dolorosa
- L'hermitte sign
    - MS
    - B12 deficiency
    - Cervical compressive myelopathy
- Uthoff sign- MS symptoms worsening with exercise
- Motor unit- single α motor neuron and the muscle fibres supplied by it
- ****__Cranial causes of paraparesis__- any defect in the paracentral lobule
    - Parasagittal meningioma
    - Superior sagittal sinus thrombosis
    - Unpaired ACA thrombosis
    - Cortical sensory loss, Jacksonian fits help to differentiate
- Urine complaints
    - Initiation
    - Hesitancy
    - Fullness of bladder
    - Maintenance of stream of urine
    - Post voidal dribbling of urine
- ***__Amaurosis fugax:__ Transient uniocular loss of vision. Indicates carotid artery atherosclerosis
- Nerve thickening: Leprosy, Amyloidosis, Sarcoidosis
- Cervical myelopathy
    - Onion pattern of facial involvement
    - Check V nerve carefully

Theory part
SACD, ATM, GBS

GBS

- Aetiology
  - About 2/3 of GBS patients experience symptoms of URI or GI infection 1-4 weeks prior to the onset of GBS
  - Pathogens associated with GBS include Campylobacter, CMV, EBV, HIV, Influenza
    - Campylobacter enteritis is the most common disease associated with GBS
- Pathophysiology
  - Postinfectious autoimmune reaction that generates cross-reactive antibodies (molecular mimicry)
  - Autoantibodies against gangliosides or other unknown antigens of peripheral Schwann cells causes immune mediated segmental demyelination
- Clinical features
  - Ascending paralysis- bilateral flaccid paralysis from the LL to UL in a "stocking glove" distribution
  - Sensory involvement usually absent. Sometimes mild sensory symptoms can be present
  - Relatively symmetric weakness
  - Bladder involvement rare
  - Cranial nerve involvement- frequently b/l VII N palsy
    - Lower cranial nerves are also frequently involved, causing bulbar weakness
  - Fever and constitutional symptoms are usually **absent**
  - Landry paralysis- involvement of the respiratory muscles
  - Reduced or absent reflexes
  - Autonomic dysfunction- arrhythmias, voiding dysfunction, intestinal dysfunction, fluctuations in BP
- Atypical GBS
  - Transient sensory level with band like sensation
  - Urinary retention
  - Babinski sign
- Subtypes and variants
  - Acute inflammatory demyelinating polyneuropathy (AIDP)
    - Adults affected more than children
    - Accounts more >90% of cases in the western world
    - Rapid recovery
    - Anti GM1 antibodies in <50%
  - Acute motor axonal neuropathy (AMAN)
    - Children and young adults
    - China and Mexico
    - May be seasonal
    - Rapid recovery
    - Anti GD1a antibodies
  - Acute motor sensory axonal neuropathy (AMSAN)
    - Mostly adults
    - Uncommon
    - Recovery slow, often incomplete
    - Closely related to AMAN
  - Miller Fisher Syndrome (MFS)

        - Adults and children
        - Ophthalmoplegia, ataxia, areflexia
        - Anti GQ-1b antibodies
- Diagnosis
    - CSF
        - Albuminocytologic dissociation
            - Elevated protein levels but normal cell counts in the CSF
            - CSF cell counts >50/µL indicate GBS is unlikely
            - Transient rise in CSF cell counts may occur, but prolonged high values make the diagnosis of GBS unlikely
        - Antibodies against gangliosides
    - Electrodiagnostic studies
        - Nerve conduction studies and EMG are useful in confirming the diagnosis and providing some information regarding prognosis
        - Demyelinating forms of GBS are supported by features of demyelination including decreased motor nerve conduction velocity, prolonged distal motor latency etc
        - Axonal forms of GBS- decreased distal motor and/or sensory amplitudes
- Criteria- Brighton criteria. **All of these**
    - Bilateral and flaccid weakness of limbs
    - Decreased and absent DTR in weak limbs
    - Monophasic illness pattern and interval between onset and nadir of weakness between 12h and 28 days
    - Electrophysiologic findings consistent with GBS
    - AC dissociation and CSF WBC <50/µL
    - Absence of alternative diagnosis
- Treatment
    - Supportive management
        - Monitor cardiac and respiratory function
        - Prevent decubitus and/or thrombosis
    - High dose IVIG (2g/kg) as five daily infusions for a total of 2g/kg
    - Plasmapheresis
        - In adults- equivalent outcome as IVIG
        - In children- only recommended in cases with rapidly progressing or severe disease
- Prognosis
    - Up to 70% of patients with GBS have a good prognosis. Symptoms recede in reverse order of development
    - 3-7% of patients with GBS die due to acute complications like respiratory paralysis, pulmonary infection, pulmonary embolism, cardiac dysfunction

CIDP

- Similarities with GBS
    - Symmetric
    - Demyelination in nerve conduction studies
- Characteristics
    - Progression over months
    - Impotence, incontinence can be present
    - Relapsing and remitting course
    - Responsive to corticosteroids

    - Usually sensory and motor involvement
    - Nerve enlargement
- Criteria
    - Mandatory clinical criteria
        - Lasting for **2 months or longer**
        - Symmetrical weakness
        - Hypo or areflexia
    - Laboratory
        - NCS suggestive of demyelination
        -

Subacute combined degeneration (SACD)

- Presents with subacute paraesthesias in the hands and feet, loss of vibration and position sensation and progressive spastic and ataxic weakness
- Tracts affected- SCD- Spinocerebellar (ataxia), Corticospinal (spastic weakness), Dorsal column
- Important diagnostic clue- Extensor plantar (because of UMN) with absent ankle jerk (because of peripheral neuropathy)
- Optic atrophy and irritability may be prominent in advanced cases
- Romberg's sign positive
- Diagnosis
    - Macrocytic RBC
    - Low serum B12
    - Increased serum homocysteine and methylmalonic acid
    - Anti parietal cell antibodies
    - Schilling test
- Treatment
    - 1000 µg B12 every day for one week
    - Every week for 1 month
    - Every month for 6 months

Neurogenic bladder

- Anatomy part
    - S2, S3, S4 contracts the detrusor- parasympathetic innervation
    - T11, T12, L1, L2 contract the internal sphincter
    - External sphincter is under voluntary control
    - Periaqueductal gray in the midbrain and Prefrontal cortex inhibit the pontine micturition centre which initiates micturition by contracting the detrusor and relaxing the internal sphincter
- Infantile/Cortical/Uninhibited bladder
    - Loss of cortical control
    - Voiding in socially inappropriate situations
    - Lesions in the frontal cortex
    - Complete voiding occurs though
- Overflow incontinence/LMN bladder/Autonomous bladder
    - S2, S3, S4 lesion
    - Decreased detrusor contraction causes increased bladder volume and decreased bladder pressure
    - Atonic, flaccid bladder
    - Intermittent voiding and hesitancy
- UMN bladder/Automatic bladder/Spastic bladder
    - Lesion anywhere from the Micturition centre to sacral area

    - Detrusor becomes overactive
    - Spastic, hypertonic bladder
    - Small volume, high pressure bladder
    - As soon as it fills, it empties, so it is called automatic bladder
- Sensory denervated bladder
- Motor denervated bladder

Care of a paraplegic

- Management of the disease
- Positioning in bed- limbs are positioned so contractures do not develop
- Pressure points are adequately padded to prevent pressure sores
- Frequent turning in bed to prevent pressure sores
- Personal hygiene of the patient should be taken care of
- Catheterisation
    - Intermittent catheterisation is better
    - But an indwelling catheter is usually kept
    - Changed once a week
    - Regular urine culture
- Bowel care- periodic enema may be required
- Physiotherapy
- Rehabilitation
    - Physical rehabilitation- Activities of daily living
    - Psychological and social rehabilitation
    - Economic rehabilitation

Acute transverse myelitis

- Aetiologies
    - Demyelinating disease- MS, NMO
    - Systemic immune mediated disorders
    - Post infectious
    - Infectious
- MS
    - Can present with ATM, especially in non-Caucasian populations

Chronic myelopathies

- Spondylotic myelopathy
    - Neck pain and shoulder pain with stiffness
    - Radicular arm pain, often with a C5, C6 distribution
    - Decreased tendon reflexes in UL, often biceps
    - Slowly progressive spastic paraparesis
    - Urinary incontinence in advanced cases
- Vascular malformations
    - Typical presentation- middle aged man with progressive myelopathy that worsens slowly or may be waxing and waning mimicking MS
    - Incomplete sensory, motor, bowel and bladder disturbances
    - Motor deficits may predominate and produce a mixed UMN+LMN involvement like ALS
    - Intermittent claudication may also be seen
- Retroviral myelopathies
    - Tropical spastic paraparesis
        - HTLV-1
        - Progressive syndrome with variable sensory and bladder disturbance

        - May resemble primary progressive MS
    - HIV myelopathy- resembles SACD
- Lathyrism
    - Toxin- BOAA- Beta oxalyl amino alanine
    - Latent stage- altered gait
    - No stick stage- short, jerky steps without the aid of a stick
    - One stick stage
    - Two stick stage
    - Crawler stage
    - Vitamin C prophylaxis
    - Banning the crop prevents it
    - Removal of the toxin by parboiling
- Syringomyelia
    - More than half of all cases are associated with a chiari 1 malformaton where cerebellar tonsils herniate through foramen magnum
    - Many young patients acquire a cervical-thoracic scoliosis
    - Regional dissociated sensory loss- loss of pain and temperature
    - Suspended anaesthesia- above and below is normal
    - Extends to involve the CST