Ftplectures Neurology system Lecture Notes

NEUROLOGY

Medicine made simple

Ftplectures Clinical Medicine
Copyright 2014

Adeleke Adesina, DO
Clinical Medicine

1133 Broadway Suite 706,
New York, NY, 10010

The field of Medicine is an ever-changing profession and as new evidence based studies are conducted, new knowledge is discovered. Ftplectures has made tremendous effort to deliver accurate information as per standard teaching of medical information at the time of this publication. However, there are still possibilities of human error or changes in medical sciences contained herein. Therefore, ftplectures is not responsible for any inaccuracies or omissions noted in this publication. Readers are encouraged to confirm the information contained herein with other sources.

Anatomy of Circle of Willis

Objectives of learning:

- Overview
- Origin
- Branches
- Arterial Supply

Overview:

Circle of Willis is an arterial circle made up of branches of two main arteries.

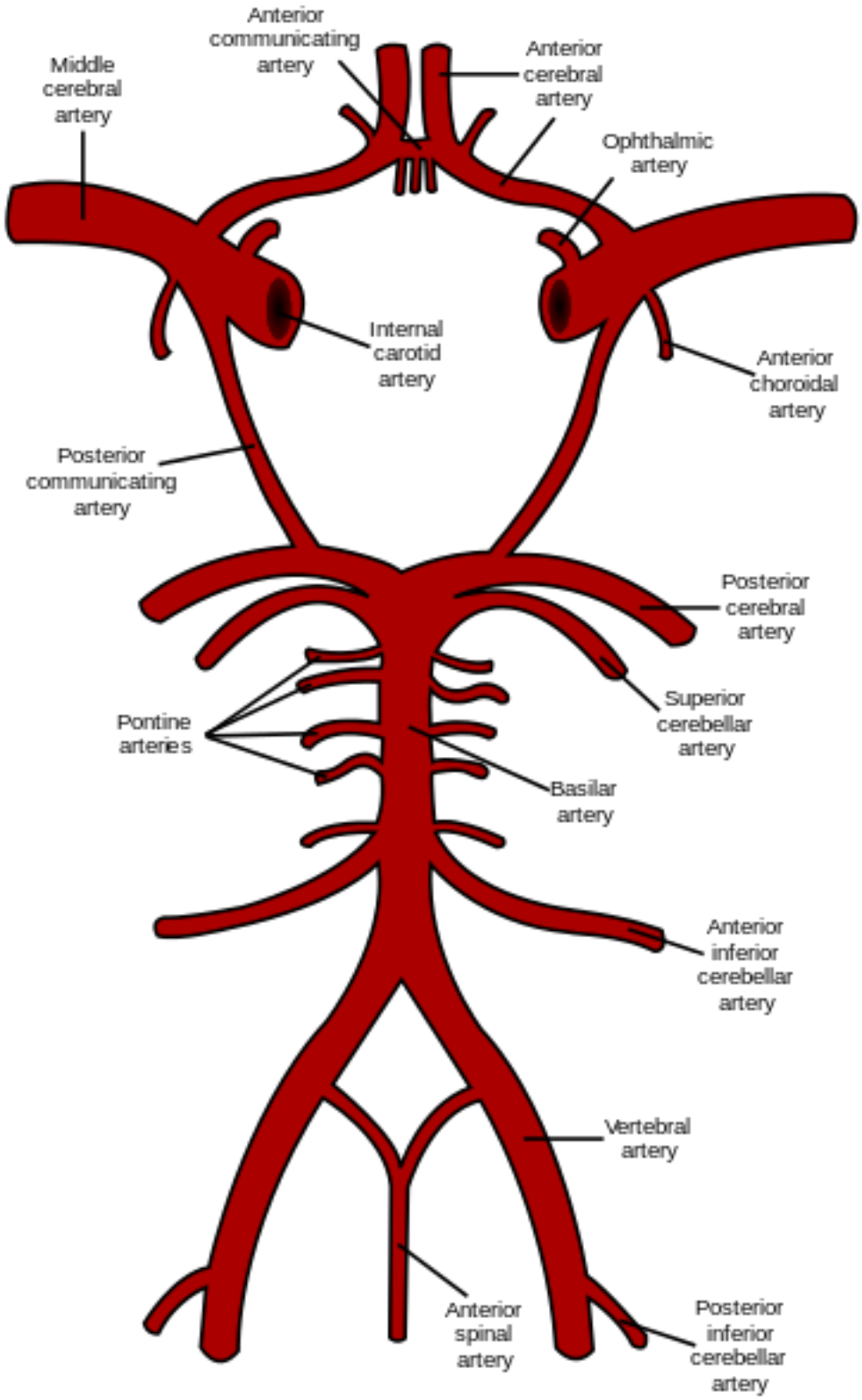
- Internal carotid arteries
- Basilar Artery

Origin:

- The internal carotid artery is a branch of common carotid artery.
- Basilar made up by fusion of 2 vertebral artery which itself is a branch of subclavian artery. Vertebral arteries give 1 anterior spinal artery and two post spinal arteries.

Branches:

- Internal carotid artery give rise to anterior cerebral artery , middle cerebral artery, anterior choroidal artery and 3 lenticulostriate arteries
- Basilar artery gives post cerebral artery (thalomoperforating artery branch of posterior cerebral).
- Anterior communicating arteries connect both ant cerebral arteries.
- Post communicating arties connect middle and post cerebral arteries.



Source: http://en.wikipedia.org/wiki/File:Circle_of_Willis_en.svg

Arterial supply:

- Ant cerebral artery supplies medial part of the brain i.e. sensory and Motor part of the leg, trunk.
- Middle cerebral artery supplies lateral part of brain i.e. motor part of hands, face and tongue.
- Post cerebral artery supplies post part of brain.

Cranial nerves:

Objectives of learning:

- Overview
- Function
- Signs and Symptoms
- Testing

Overview:

12 pairs of cranial nerves in body.

Mnemonic ⇨ Oh Oh Oh To Touch And Feel A Girl Veil Such Happiness.

1. **O**lfactory N.
2. **O**ptic N.
3. **O**cculomotor N.
4. **T**rochlear N.
5. **T**rigeminal N.
6. **A**bducent N.
7. **F**acial N.
8. **A**uditory (Vestibulocochlear) N.
9. **G**lossopharngeal N.
10. **V**agus N.
11. **S**pinal Accessory N.
12. **H**ypoglossal N

Functions:

1. **O**lfactory N.> smell
2. **O**ptic N.> light reflex, image carrying to brain
3. **O**cculomotor N.> supply superior inferior and medial rectus , inferior oblique, sphincter pupillae, dilator pupillae, palpebral superiorus
4. **T**rochlear N.> supply superior oblique
5. **T**rigeminal N.
    - V1 supply sensory supply to forehead, corneal reflex sensation
    - V2 supply sensory supply to middle 3rd of the face, nasal cavity and upper teeth, palate
    - V3 sensation on ant 2/3 tongue; innervation of masseter , medial and lateral pterygoid, temporalis, ant belly of digastric, mylohyoid, tensor tympani, tensor palatai; sensation on mandible lower teeth
6. **A**bducent N.> Supply lateral rectus
7. **F**acial N.> ant 2/3 tongue taste sensation , salivation, close mouth , facial muscles , wrinkle forehead , close eyes, blink reflex, supply stapedius to dampen sound , platysma , lacrimal gland innervation
8. **A**uditory (Vestibulocochlear) N. > hearing and balance
9. **G**lossopharyngeal N.> gag reflex, post 1/3 tongue sensation, oropharynx innervation, carotid body and sinus innervation, stylopharyngeus

10. **V**agus N> Supply palate (except tensor palatai) pharynx muscle (except stylopharyngeus) , voice production , parasympathetic supply up to mid gut .
11. **S**pinal Accessory N.> innervate sternocleidomstoid ,
12. **H**ypoglossal N> tongue muscle innervation(except palatoglossal)

Signs and Symptoms:

When damaged

1. **O**lfactory N. > Anosmia
2. **O**ptic N. > Anopsia , Loss of light reflex
3. **O**cculomotor N.> Diplopia, External strabismus, Ptosis, Dilated pupil, loss of convergence , loss of accommodation
4. **T**rochlear N.> unable to look down , head tilted down
5. **T**rigeminal N.>
    - V1 Ophthalmic> No sensation on forehead , no blinking on corneal touching
    - V2 Maxillary> No sensation on palate, nasal cavity, upper teeth
    - V3 Mandibular> No sensation on ant2/3 tongue; paralysis of masseter pterygoid temporalis, ant belly of digastric, mylohyoid, tensor tympani, tensor palatai; no sensation on mandible lower teeth ; mouth deviation on side of lesion
6. **A**bducent N.>Internal strabismus
7. **F**acial N.> loss of ant 2/3 toungue taste sensation , loss of salivation, unable to close mouth , unable to wrinkele forehead , unable to close eyes, loss of blink reflex, hyperaccusis , lacrimal duct loss of tears
    - In Lyme disease entire facial nerve damaged all sign n symptoms of paralysis on same side unilaterally.
    - In corticobulbar lesion upper part of face is spared but facial drooling is present unilaterally
8. **A**uditory (Vestibulocochlear) N.> Vertigo, Nystagmus, hearing loss
9. **G**lossopharngeal N.> loss of post 1/3 tongue sensation, loss of gag reflex,
10. **V**agus N.> palate drooping , uvula deviates away from lesion, dysphagia, loss of gag reflex, hoarseness , dysphonia
11. **S**pinal Accessory N.> loss of shrugging shoulder , difficulty combing hair, weakness moving neck
12. **H**ypoglossal N.>tongue protrude towards lesion side

Testing:

1. **O**lfactory N. > Smell
2. **O**ptic N.> can see
3. **O**cculomotor N.> move object close and make an H while moving finger and ask the patient to see it
4. **T**rochlear N.>ask the pt to look down
5. **T**rigeminal N.
    - V1 sensation on forehead
    - V2 sensation on middle 1/3
    - V3 sensation on mandibular part of face
6. **A**bducent N.> ask the patient to look on lateral side

7. **F**acial N.> facial expression
8. **A**uditory (Vestibulocochlear) N.> can hear
9. **G**lossopharngeal N.> gag reflex
10. **V**agus N.>voice production
11. **S**pinal Accessory N.> can shrug shoulders
12. **H**ypoglossal N> move tongue

Cerebrovascular accident/Stroke:

Objectives of learning:

- Definition
- Epidemiology
- Classification
- Transient Ischemic Attack
- TIA Risk factor
- TIA symptoms
- Lacunar Stroke
- Sign and Symptoms
- Diagnosis
- Complication
- Treatment
- Prevention

Definition

CVA/Stroke is decreased blood supply/perfusion to the brain for more than 24 hours.

Epidemiology

Stroke is third leading cause of death in U.S. It is the leading cause of neurological deficit in the world.

Classification:

- Ischemic Stroke {85%}: An emboli or a thrombus or in acute congestive cardiac failure block an artery so in absence of oxygen it convert glucose anaerobically into pyruvate, pyruvate into lactic acid. Less and less ATP is formed, leading to infarction and hence necrosis.
- Haemorrhagic Stroke {15%}: Rupture in blood vessel leading to subarachnoid haemorrhage. Most commonly berry aneurysm.

Transient Ischemic Attack:

Transient Ischemic Attack unilateral deficit that last for few minutes but not more than 24 hours without permanent damage with no necrosis. 35% High risk to develop stroke.

TIA Risk factor:

- Age
- hypertension
- High cholesterol
- Diabetes
- Atrial fibrillation i.e. whole atrium doesn't contract simultaneously leading to stasis of blood in atrium and a clot can be formed which can embolize into carotid
- Coronary artery disease
- Family H/O of Stroke
- Carotid bruit i.e. rushing of blood through obstructed vessel

- Oral contraceptive i.e. estrogen lead to increase formation of clot
- Hyper coagulation state
- Drugs i.e. Cocaine causing vasospasm , amphetamine
- Hereditary disease I.e. Sickle cell anaemia and Polycythaemia Vera cause sledging

TIA Symptoms:

- Carotid artery:
  Unilateral Amaurosis fugax (Temporary loss of vision) > blocking of ophthalmic artery which give retinal artery by embolization,
  Temporary loss of speech > Obstruction of middle cerebral artery supplying Brocca's area
  Paralysis and paraesthesia of contralateral limb

- Basilar artery:
  Dizziness > cerebellum at post part of brain control balance and coordination
  Diplopia > post cerebral cortex
  Vertigo
  Ipsilateral face and contralateral limb numbness > facial nucleus in brain stem
  Dysarthria (speech problem) and Dysphagia > vagal nucleus in brainstem
  Headaches

Lacunar Stroke:

A type of stroke in which deep structure of brain is effected i.e brain stem, basal ganglia , thalamus internal capsule **but not cortex.** Hypertension is 90% causative. Thickening of branches of middle cerebral artery **anterior choroidal artery** supplying genu and post limb of internal capsule. Complaints are complete contralateral anaesthesia, complete arm and leg weakness ,contralateral heminomus hemianopia , bilateral hearing loss , contralateral lower half of face weakness , transient deviation of uvula towards side of lesion , tongue deviate away from lesion. Lenticulostriate arteries supply ant part of internal capsule, caudate, putamen and globus pallidus nucleus.

Symptoms and Sign:

Middle cerebral artery Stroke:

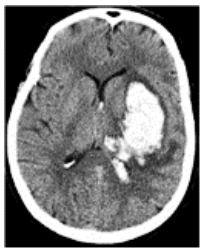
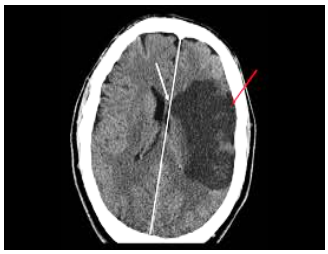
- Contralateral hemiparesis/hemisensory loss
- Aphasia i.e. Brocca's area affected
- Contralateral forgetting body Syndrome

Anterior cerebral artery Stroke:

- Contralateral lower extremity and trunk weakness

Diagnosis:

- CT scan without contrast > to differentiate between haemorrhagic and ischemic stroke. Hemorrhagic strokes are white. Ischemic strokes are black.

Haemhrogic Stroke  Ischemic Stroke 

- MRI
- EKG > for emboli from atrial fibrillation
- Carotid duplex scan > amount of stenosis in Carotid artery
- **MRA (Magnetic Resonance arteriogram**)> Gold standard to differentiate between site of obstruction
- Chest Stroke > Aortic Dissection leading hypotension leading hypo perfusion and Aspiration Pneumonitis in gag reflex absence
- CBC and platelet count and PT/PTT for coagulopathy
- Serum electrolyte
- Glucose level > hypoglycaemia
- Bilateral carotid ultrasound
- Echocardiogram > heart failure

Complications:

- Progressive neurologic deficit
- Cerebral oedema > due to reperfusion leading to Intracranial pressure raise. Treatment is hyperventilation and mannitol.
- haemorrhage
- Infarction
- Seizures

Treatment:

- Supportive Treatment > air way management with intubation , oxygen , IV fluids , check Blood pressure, keep NPO, Elevate head to prevent aspiration,
- Anti-Hypertensive therapy in heart failure , MI , Aortic dissection
- Thrombolytic therapy> Tissue plasminogen activator if stroke is less than 3 hours.
    - Contraindicated in hypertension, H/O of recent trauma or surgery, bleeding disorder, aspirin in 24 hours (anti-coagulant drug).

Surgical intervention to correct stenosis of carotid artery

Prevention:

Control Diabetes

Control hypertension

Stop smoking

Exercise

Diet modification

Control obesity

Guillain Barre Syndrome

Objectives for learning

Definition, etiology, signs and symptoms, diagnosis, differential diagnosis, treatment and management, and prognosis.

Definition

Guillain Barre Syndrome is demyelinating disease of the motor neurons. It is characterized by the inflammation and polyneuropathy.

Etiology

The GB syndrome is preceded by the infections e.g. upper respiratory diseases, gastrointestinal diseases
GI infections include *campylobacter jejuni*
Viral infections include CMV, Hepatitis, and HIV

Signs and symptoms

It has an abrupt onset which is severe and manifests itself in the form of ascending paralysis i.e. starts from the extremities and goes towards the center of the body. The person is unable to move the extremities but the sensory nerves are intact. Once the disease reaches the center of the body and involve diaphragm, the consequences are disastrous because of respiratory failure.
This disease may also include the autonomic features such as arrhythmia, tachycardia, and postural hypotension.

Diagnosis

CSF analysis by lumbar puncture reveal elevated protein but normal cell count.
Nerve conduction studies reveal decrease motor conduction velocity because of demyelination of the nerves.

Differential diagnosis

Multiple sclerosis. The sphincter and bladder control are intact in GB syndrome as compared to multiple sclerosis.

Treatment and Management

Pulmonary function monitoring is compulsory.
IVIG for severe weakness are useful.
Plasmapheresis is used to filter the antibodies.
NO steroids because it will worsen the disease.

Prognosis

Most of the patient recover in 1-3 weeks.
After 6 weeks, the prognosis is bad.

Head Trauma:

Objectives of learning:

- Types
- Key factors
- Sign of skull base fracture
- treatment

Types:

- Primary Irreversible damage
- Secondary insult (we can prevent damage)
    1. Low BP > hypoperfusion causing ischemia
    2. Low O2 > Anaerobic respiration causing less ATP
    3. Pneumothorax in chest trauma
    4. Brain stem compression > Apnoea
    5. Hypercapnea > leading to vasodilatation lead to increase intracranial pressure
    6. Intracranial Mass Effect in epidural hematoma or subdural hematoma
    7. Anaemia > loss of blood lead to hypoperfusion

Key Factors:

- Increase intracranial pressure
    1. Leading to decrease perfusion pressure in arteries supplying brain> leading to ischemia. Treatment is maintain ICP less than 20 mmHg.
    2. Another effect is transtentorial herniation. Leading to compression of 3rd nerve leading to bilateral dilated pupil. Loss of consciousness, Bradycardia, respiratory compromise.

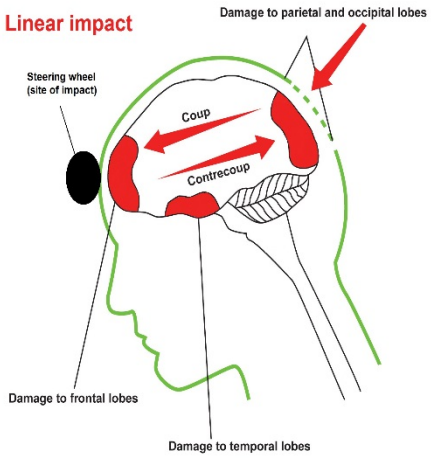
Sign of basal skull fracture

Raccoon eyes (periorbital ecchymosis)

Battle sign (post auricular ecchymosis)

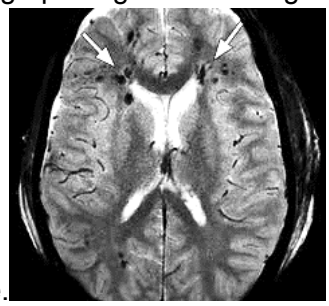
Hemotympanum

CSF rhinorrhoea



Coup and Counter coup injury

Seizures

Diffuse Axonal Injury > shearing ripening force damage entire neuron of brain. In CT scan there is punctate haemorrhage. 

Treatment:

ATLS

Lower intracranial pressure by putting them in reverse Trendelenburg position.

Hyperventilate to decrease CO2 to decrease vasodilation to decrease intracranial pressure. Maintain between 35-40 mmHg. If too low it can lead to vasoconstriction can lead to ischemia

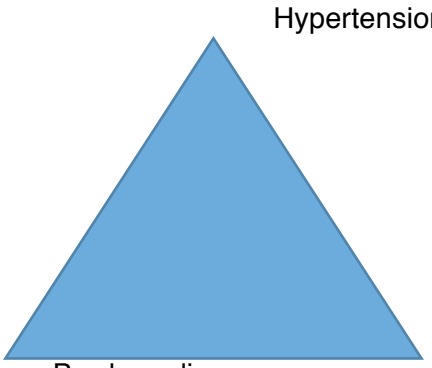
Mannitol

Sedation with Fentanyl morphine

Decrease Body temperature

Venticulocatherization

CUSHING TRIAD – Bradycardia and Hypertension


Hypertension
Bradycardia
Respiratory
distress

Intracranial Haemorrhage

Objectives of learning:

- Anatomy
- Intracranial haemorrhage
- Risk factor
- Location
- Clinical feature
- Diagnosis
- Complication
- Physical exam
- treatment

Anatomy:

Three coverings of brain parenchyma:

1. Dura matter
2. Arachnoid matter
3. Pia matter
   Cerebrospinal fluid is secreted by ependymal cells of choroidal plexuses into lateral ventricles> 3rd ventricle >cerebral aqueduct > 4^{th} ventricle > arachnoid granulation> subarachnoid space> blood vessel around brain
   Or from 4^{th} ventricle> lateral foramina of Lushka > subarachnoid space of spinal cord

Intracranial Haemorrhage:

- bleeding in Parenchyma

Risk factor:

- Hypertension> micro aneurysm is developed in chronic aneurysm and can rupture
- Ischemic stroke >> haemorrhagic stroke
- Amyloid angiopathy
- Anti-coagulopathies
- Brain tumour
- Av malformation

Location:

Basal ganglia 66%

Pons

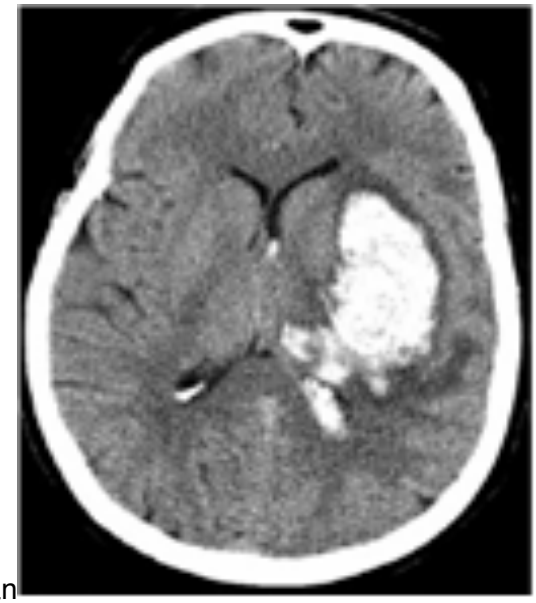
Cerebellum

Clinical features:

- Neurological deficit
- Altered mental status
- Headache/vomiting

- increase ICP

Diagnosis:

CT scan 

PT/PTT test

Complications:

- Increase ICP
- Bleeding
- Vasospasm on Blood vessels
- Hydrocephalus
- Syndrome of inappropriate ADH

Physical examination

1. If pons is affected then pin point pupil
2. If Thalamus is affected then poly reactive pupil
3. If putamen is affected than dilated pupil

Treatment:

- ❖ Admission to ICU
- ❖ ABC is followed
- ❖ Control BP by nitroprusside and beta-blocker
- ❖ Mannitol
- ❖ Surgical evacuation of hematoma

Subarachnoid Haemorrhage:

Overview:

- ➢ Causative factor
- ➢ Clinical feature
- ➢ Diagnosis

- Complications
- Treatment

Causative factor:

Rupture berry aneurysm (polycystic kidney disease person have more common) is in anterior communicating artery and junction between post communicating and post cerebral.

Trauma

Av malformation

Clinical Feature:

- WORST HEADACHE OF THE LIFE i.e. irritation of meninges
- Sudden transient loss of consciousness
- Vomiting
- Neck Rigidity
- photophobia

Diagnosis:

- Non-contrast CT Scan
  If negative do Lumbar puncture to confirm Blood in CSF or yellow CSF due xanthochromia due to lysis of Rbc. > cerebroangiogram and if positive Surgical clipping

Complication

Rupture

Vasospasm i.e. give calcium channel blocker

Hydrocephalus due to blood stuck in subarachnoid space blocking absorption of CSF

SIADH

Treatment:

Neurosurgery

Bed rest

Stool softener

Acetaminophen

Control hypertension

Multiple Sclerosis:

Objectives of learning:

- Definition
- Epidemiology
- Pathophysiology
- Pathways effected
- Clinical features
- Different course
- Prognosis
- Diagnosis
- Treatment

Definition:

Multiple (Many) Sclerosis (Scar tissue formation).

It is an idiopathic selective demyelinating disease of white matter of the CNS.

Epidemiology:

High incidence in people above the equator. Female are more susceptible 2-3 times.

Pathophysiology:

CNS consist of grey matter and white matter. Grey matter consist of the cell bodies of the neuron having nucleus, Golgi apparatus and endoplasmic reticulum. While white matter consist of the axons of the neuron enclosed in a myelin sheath.

In multiple sclerosis the myelin sheath of the axons are affected both in CNS and PNS.

Pathway effected:

- Pyramidal and Cerebellar pathway
- Medial longitudinal Pathway > have a role in conjugate movement of eye ball , MLF is activated by paramedian pontine reticular formation in pons > when pathway effected leads to:
    1. Internuclear Opthalmeplegia with intact convergence
    2. Monoocular Nystagmus
- Optic nerve and posterior column pathway(touch vibration)

Clinical features:

Relapse Attack occur commonly after 1 year and continue for 24 hours or more.

- Transient sensory deficit > post column neuron affected
- Fatigue
- Motor Symptoms> pyramidal tract
  Weakness
  Spasticity
  Hemiparesis, parapresis , quadripresis
- Visual symptoms

Pain on eye movement
Central scotoma
Diplopia

- ❖ Ataxia, intention tremor, Balance problems
- ❖ Dysarthria
- ❖ Bowel and Bladder loss of function
- ❖ Impotence
- ❖ Constipation
- ❖ Neuropathic pain such as Trigeminal Neuralgia, Hyperaesthesia.
- ❖ Psychological problems Anxiety, memory loss, Depression

Different Course:

a) Clinical Silent
b) Relapsing Course > Exacerbation of disease and then patient got better and then again Symptoms appear and then again disappearance of the disease (MOST COMMON)
c) Secondary Progressive> Relapsing Course of the disease but each time Symptoms reappear it is worse than the last time , than again disappear and again symptoms appear with worsen condition.
d) Primary Progressive> slowly and Steady worsening of the Symptoms over time. Often appear later after 40, with less visual symptoms.

Prognosis:

Highly variable, with 1/3 of pt develop severe disability . Lot of frequent attack or cerebellar symptoms have poor prognosis.

Diagnosis:

- ❖ Clinical examination;
- ❖ MRI > lot of demyelination of CNS
- ❖ Lumbar puncture > CSF have oligoclonal bands of IgG.
- ❖ Evoked Potentials > for demyelination

Treatment:

Goal is to prevent relapse and if there is acute exacerbation relieve it.

- ❖ Acute Attack :high dose IV Corticosteroids,
- ❖ Start early Interferon Beta1a and Beta 1b flu like symptoms sideaffects, or Glatimara acetate
- ❖ Bactrofen for muscle spasm
- ❖ Carmazapenine and gabapentin for neuropathic pain

Myasthenia Gravis:

Objectives of learning:

- Definition
- Epidemiology
- Pathophysiology
- Symptoms
- Diagnosis
- Treatment

Definition:

Autoimmune disorder, IgG antibody bind with Nicotinic Ach receptor blocking Ach released by axon. This will lead to inability of muscle to contract.

Epidemiology:

Women 20-30 years and male above 50.

Pathophysiology:

AP is fired at axon. Neuron fires at neuromuscular junction by calcium. Ach vesicles fuse with the membrane and Ach is released. Ach bind with receptor at NMJ. Calcium is released from sarcoplasmic reticulum. Calcium bind and activate and ATPase. Actin and Myosin shorten and contract.

Ach at NMJ is broken out by Ach esterase and it is absorbed by neuron.

Symptoms:

- Skeletomuscular weakness with intact reflexes and sensation (reflexes and sensations are driven out by dorsal aspect of spinal cord).
- Muscle fatigue and increased fatigue with continuous contraction.
- Drooping of eyelids
- Double vision i.e. inability of extra ocular muscle to contract
- Dysphagia i.e. upper 1/3 skeletal muscle weakness
- Facial weakness
- Slurred speech
- Difficult chewing
- Weakness of Proximal limb muscle
- **Myasthenia Crisis** > Diaphragm and intercostal muscles get fatigued eventually leading to respiratory failure.

Diagnosis:

- Ach receptor antibody test.
- Electromyography (slow decrease in muscle contraction)
- CT scan to Check for Thymoma.
- Edrophonium (Tensilon test). Ach esterase inhibitor > + amount of Ach > better contraction

Treatment:

- Anti cholinestrase inhibitor i.e. Pyridostigmine.
- Thymectomy
- Immunosuppressant immunoSteroids, Azathyporine or cyclosporine
- Plasmapheresis to remove antibodies in Myasthenia crisis
- Monitor forced vital capacity at 15ml/kg
- Contraindicated aminoglycosides beta-blockers antiarrhythmic drugs

Lambert-Eaton Syndrome:

IgG antibodies bind to axonal presynaptic neuron Calcium influx channel leading to inability of calcium influx. Associated with Small cell lung Cancer. PT have proximal muscle weakness/ hyporeflexia. To differentiate it from Myasthenia gravis, in Lambert-Eaton Syndrome patient contraction is improved after repeated contraction.

Seizures:

Objectives of learning:

- Definition
- Causes
- Types
- Diagnosis
- Treatment
- Status Epilepticus

Definition:

Seizure is sudden abnormal electrical activity in the Brain. Epilepsy is recurrent Seizures.

Causes:

Treat the cause we treat the seizures. H/O is very important.

- Metabolic:

1. Hypernatremia <120 mEq
2. Water intoxication
3. Low blood glucose
4. Hypocalcaemia
5. Uraemia
6. Thyroid Storm
7. Hyperthermia

- Mass Lesion:

1. Tumour
2. Intracranial or subarachnoid haemorrhage

- **Missing Drugs** Non-Compliance: Most common cause

1. Acute Alcohol withdrawal ,Benzodiazepine

- Miscellaneous:

2. Psych origin (check EEG) Pseudo seizure
3. Eclampsia > Treatment is Magnesium sulphate and deliver baby

- Intoxications :

1. Drugs Cocaine
2. lithium lidocaine
3. lead poisoning
4. CO poisoning

- Infections:

1. Sepsis
2. Bacterial/viral meningitis

- Ischemia/Stroke/TIA:
- Increased Intracranial pressure:

Types of seizures:

- Partial Seizures >18 years old in temporal lobe
    a) Simple partial seizures > Level of consciousness is intact

b) Complex partial seizures > impaired consciousness with postictal confusion automatism (repetitive purposelessly movement), hallucination can occur.

- Generalized Seizure > Loss of consciousness and whole brain is discharging
  a) Tonic-Clonic (Grand mal seizures) > loss of consciousness after that fall down first phase is tonic (rigid + apnoeic) and then the clonic phase occur (jerking musculature) with biting of tongue and pt become flaccid it can occur with vomiting, urination and defecation it followed by postictal confusion.
  b) Absence Seizures > school aged children, disengagement of the current activity, symptom is staring at the space and regain orientation after some time, no postictal confusion.

Diagnosis:

- If Known seizures patient:

  Check drug level

- First episode of seizure:

a) CBC > ++WBC indicate infection possible sepsis or meningitis
b) Chest Radiograph> indicate pneumonia
c) Electrolyte
d) LFTs
e) Glucose
f) Renal function test> ++Urea level indicate uraemia
g) Calcium level
h) Urinalysis>++WBC with nitrates indicate urosepsis,
i) EEG
j) CT scan> mass in brain causing seizures
k) Lumbar function test > rule out meningitis
l) Pregnancy test > Seizures drugs are teratogenic so contraindicated in pregnancy

Treatment:

Follow Airway > Breathing > Circulation

- Patient With known seizures if phenytoin dose is less than therapeutic level give then a loading dose.
- If 1st episode of seizures Patient give them anticonvulsant and get EEG

anticonvulsant drugs are

a) Phenytoin
b) Carbamazepine
c) Phenobarbital
d) Valporic acid
e) Pyrimidine

- If Absence seizure drug of choice is **Ethosuccimide** or Valporic acid

Status epilepticus:

It is a medical emergency with 20% mortality, in it after seizures patient don't gain consciousness, occur due to poor drug compliance, tumour, drug overdose, metabolic disturbances.

Management is to maintain airway, Drug of choice is IV Diazipine >if did not work phenytoin > if did not work give 50 mg of dextrose and phenobarbital > intubate pt.

The Basal Ganglia:

Objectives of learning:

- Anatomy
- Functions
- Pathways

Anatomy:

Basal ganglia consist of

- Striatum Is consist of 2 parts
    1. Caudate
    2. Putamen
- Globus Pallidus
    1. GP external
    2. GP internal
- Substantia Nigra
    1. Pars Compacta
    2. Pars Reticularis
- Midbrain Nucleus

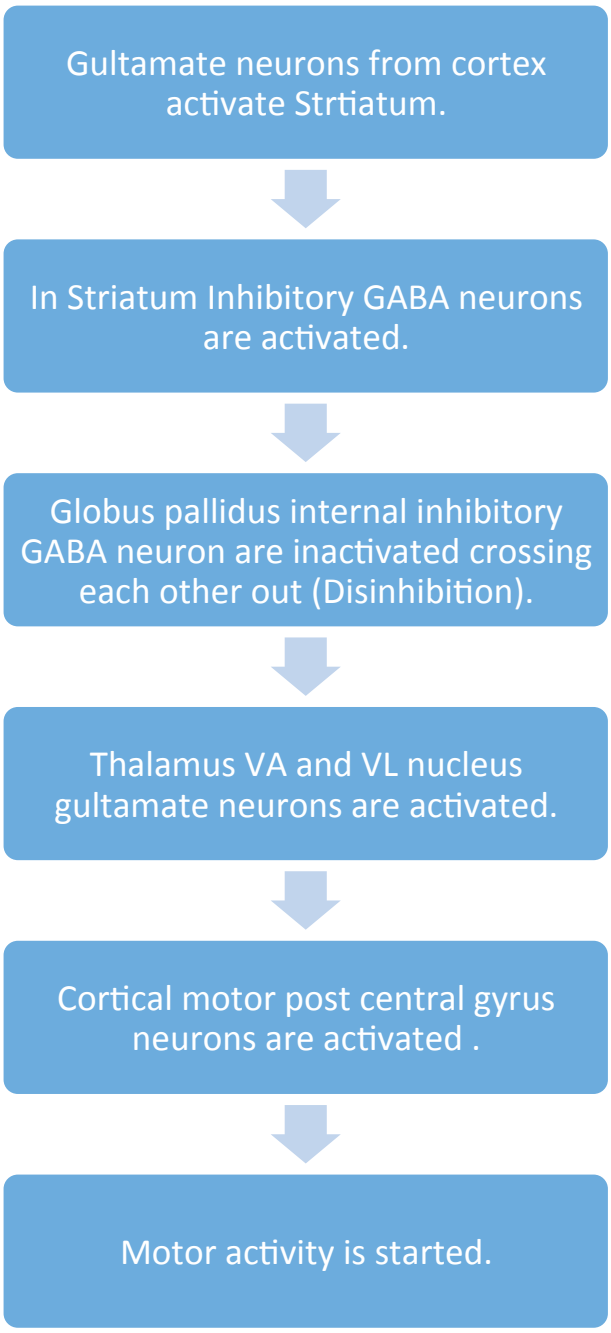
Functions:

- Movement initiation
- Affect upper motor neuron
- Uses Glutamate and GABA
- Uses Dopamine and acetylcholine

Pathways:

❖ Direct Pathway:

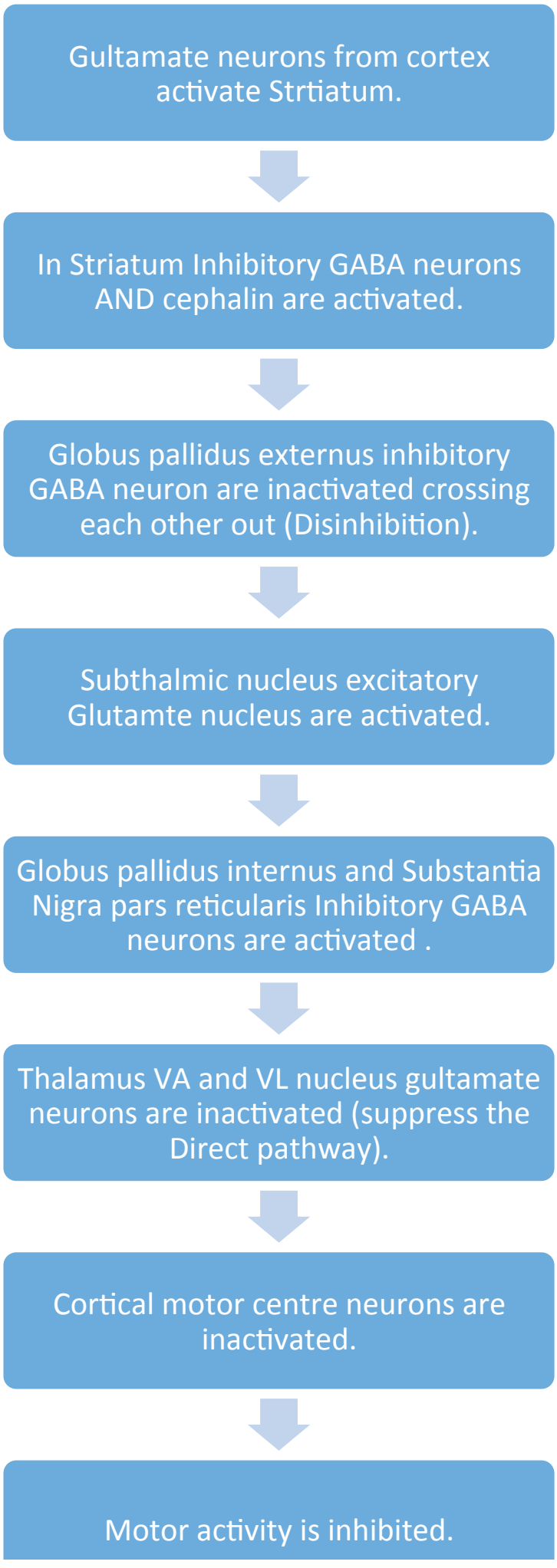
It initiate the motor activity.



Substantia Nigra compacta Dopamine neurons enhance cortical glutamate neurons

❖ Indirect Pathway:

It inhibits the motor activity.


Gultamate neurons from cortex activate Strtiatum.
In Striatum Inhibitory GABA neurons AND cephalin are activated.
Globus pallidus externus inhibitory GABA neuron are inactivated crossing each other out (Disinhibition).
Subthalmic nucleus excitatory Glutamte nucleus are activated.
Globus pallidus internus and Substantia Nigra pars reticularis Inhibitory GABA neurons are activated .
Thalamus VA and VL nucleus gultamate neurons are inactivated (suppress the Direct pathway).
Cortical motor centre neurons are inactivated.
Motor activity is inhibited.

Striatum Ach neurons enhance the effect of pathway. Substantia Nigra compacta Dopamine neurons has inhibitory effect on pathway.