

* Affective disorders

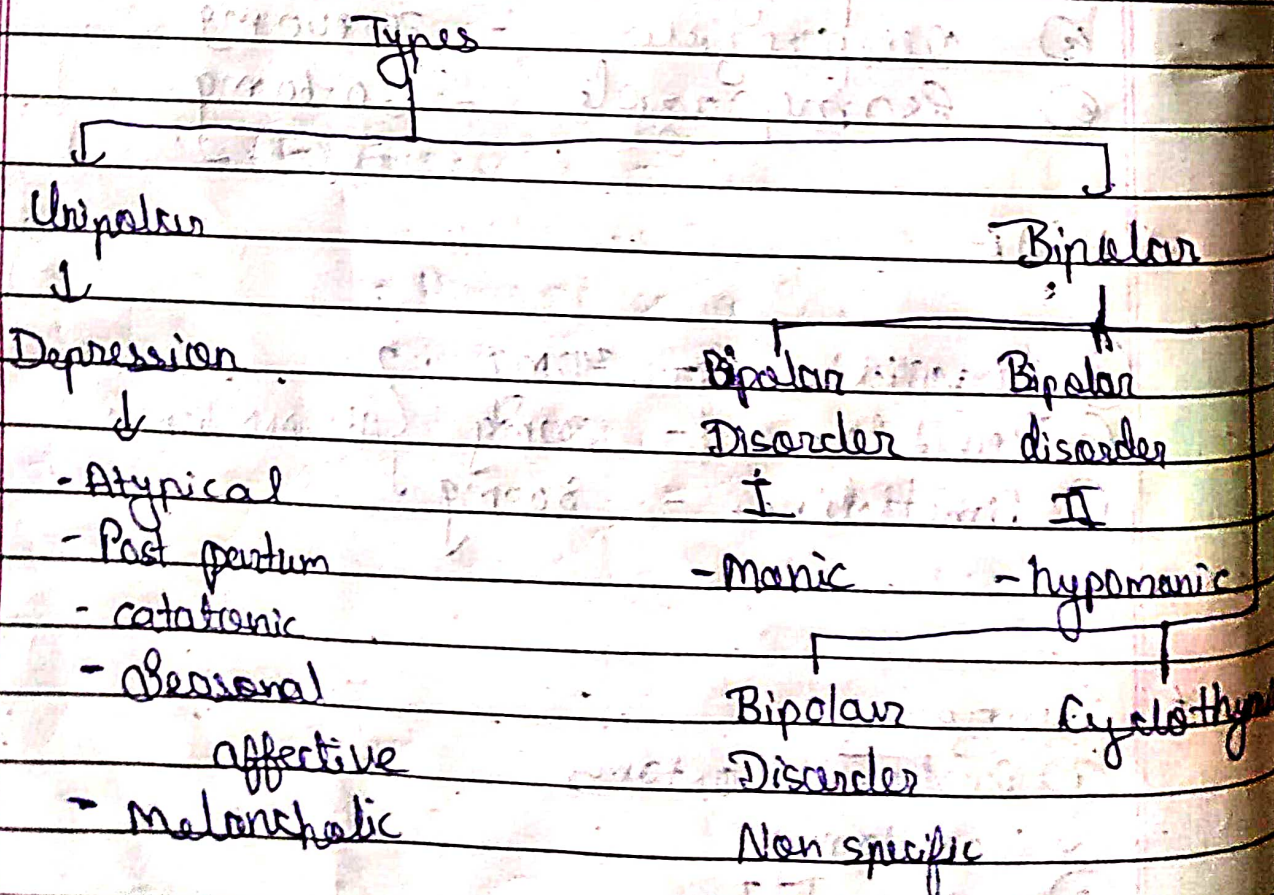
Q1 Define, Types & pathophysiology of Affective disorders

Ans Define: Affective disorders are the set of psychiatric disorders called mood disorders

- This ~~mood~~ disorder is either unipolar or bipolar

- Mainly common is depression and less common is manic or bipolar disorder

* Types of Affective disorders:

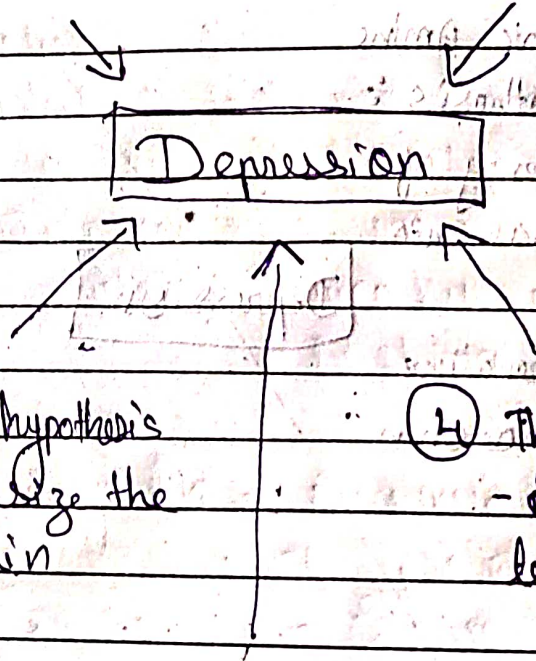


r Pathophysiology

① Depression

① ^{Biogenic} ~~Receptor~~ Amine hypothesis
- Deficiency of monoamine
like noradrenalin &
Serotonin.

② Receptor Sensitivity hypothesis:- Pathological alteration in receptor site



③ The Serotonin only hypothesis
- Explain & emphasize the
role of Serotonin

④ The Premissive hypothesis
- Serotonin & Noradrenalin
level drops

⑤ Electrolyte membrane hypothesis
- Hypercalcaemia &
other electrolyte imbalance
in Endocrine.

② Bipolar disorder

~~Genetics~~, ~~Neurotransmitter imbalance~~

- ① Genetic
- ② Neurotransmitter imbalance
(Serotonin & Noradrenaline ↑)
- ③ Abnormal neural circuit.
- ④ Electrolyte imbalance
(↓ of Ca^{2+})
- ⑤ Environmental factors.

Decrease control
of emotion

↓
Disruption of
emotional
homeostasis

↓
Bipolar disorder

Q2 Clinical manifestation of Affective disorder

u) Etiology

w) Specific cause is unknown but some factors responsible are

- Genetic factor
- Environmental :- Trauma + injury, hemorrhage etc
- Biochemical factor :- Deficiency of neurotransmitter
- Defect in Dopaminergic activity
- Endocrine factor :- Hypothyroidism
- Cushing Syndrome
- Side effect of drugs like :- Analgesic
- Antipsychotic
- Steroids
- Oral Contraceptive
- Antihypertensive

u) Sign & Symptoms

Depression	Bipolar (Manic)
- loss of interest	- Elevated/irritable mood
- Fatigue	- Anger
- Insomnia	- Increase Euphoria
- Low Mood	- less need of sleep
- Poor Concentration	- Aggression
- Suicidal thought	- Hallucination
- less Appetite	- Impulsiveness

w) Diagnosis, test

- ① Psychiatric evaluation
- ② Monitoring of symptoms
- ③ Beck Depression Inventory
- ④ Hamilton Depression Rating Scale

* Treatment

① Non pharmacological

② Pharmacological

w) For the depression antidepressant are used.

Anti depressant.

① SSRI

- Citalopram :- 20-40 mg/day
- Escitalopram :- 10-20 mg/day
- Fluoxetine :- 50-300 mg
- Paroxetine :- 20-60 mg
- Sertraline :- 50-200 mg
- ~~Fluoxetine~~

② SNRI

- Duloxetine :- 30-90 mg
- Venlafaxine :- 75-225 mg
- Desvenlafaxine :- 50 mg

③ TCAs

- Amitriptyline :- 100-300 mg
- Desipramine :- 100-300 mg
- Imipramine :- 100-300 mg
- Nortriptyline :- 50-150 mg

④

- NDRI (Norepinephrine & dopamine reuptake inhibitor)
- Bupropion :- 150-300 mg

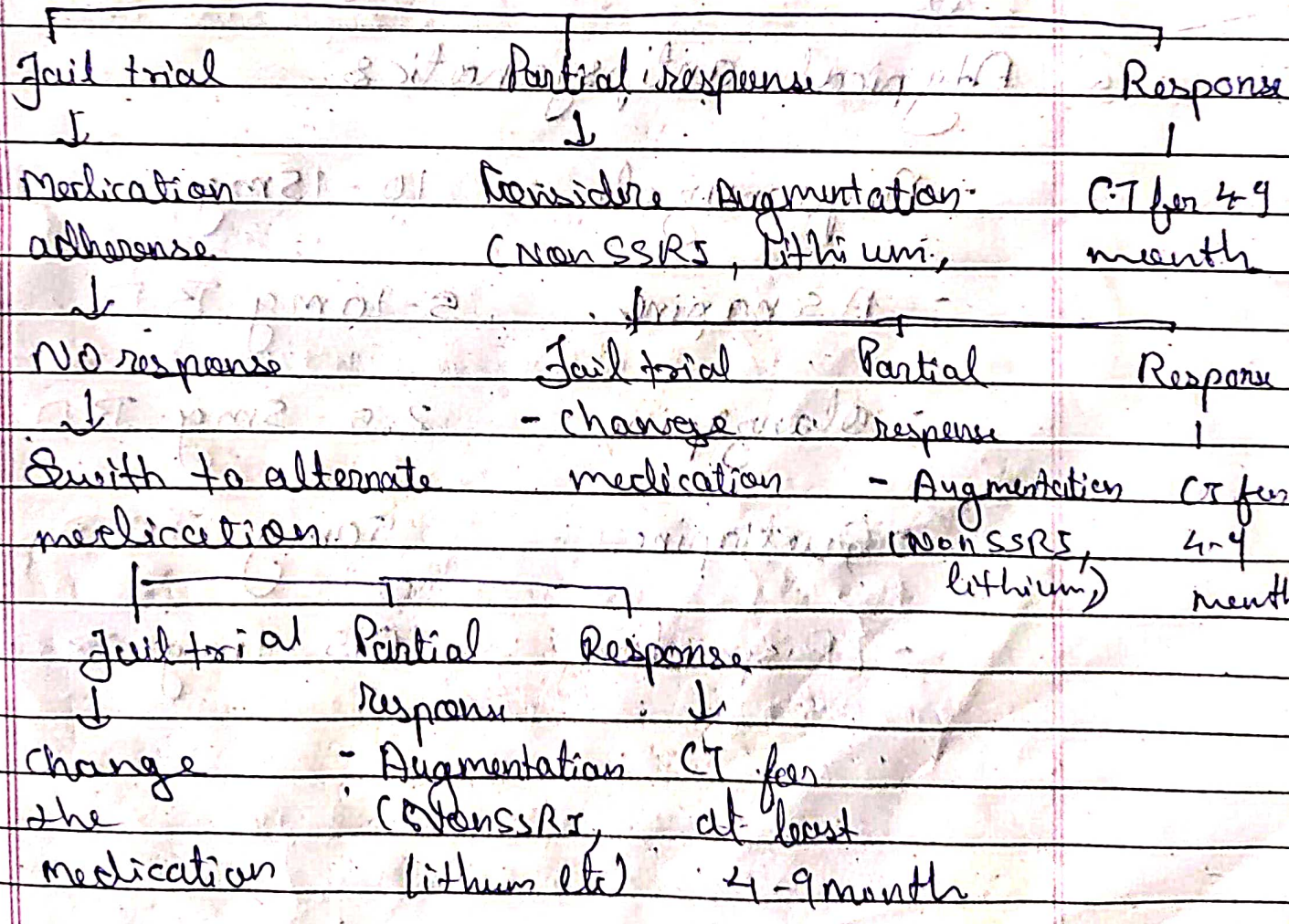
⑤ MAO Inhibitor

Selegiline :- 6-12 mg

Phenelzine :- 30-90 mg

Patient with depression &
not taking any medication

↓
SSRI



iv) For bipolar disorder

① Lithium carbonate :- 300mg BD

② Anticonvulsant

- Valproic Acid :- 250 - 500mg BD

③ Lamotrigine :- 25mg BD

④ Carbamazepine :- 200mg BD

⑤ Oxycarbazepine :- 300mg BD

⑥ Atypical antipsychotics

- Aripiprazole :- 10 - 15mg

- Aripiprazole :- 5 - 10mg BD

- Olanzapine :- 2.5 - 5mg BD

- Quetiapine :- 50mg BD

- Risperidone :- 0.5 - 1mg BD

* Anxiety disorders

Q1 Define, types & patho of anxiety disorder

Ans) Anxiety defined as a subjective sense of unease, dread or foreboding can indicate a primary psychiatric condition

- Phobias are most common with higher rates in Population

* Types

Ans) There are 6 type of Anxiety

① Generalized anxiety disorder (GAD)

② Specific phobia

③ ~~Agoraphobia~~ Post-traumatic stress disorder (PTSD)

④ Social anxiety disorder (SAD)

⑤ Panic disorder

⑥ OCD

① Generalized anxiety disorder :- It affects only 3% of population. Patient worry most days of many matters

② Specific phobia :- 7-9% of population

Teen has more risk of phobias

- mainly animal, natural environment, & situations are specific phobia

③ Agoraphobia :- 1.7% of population agree
- fear of places & situation develops
after one or more panic attacks

④ Social anxiety disorder
:- ~~from~~ ~~for~~ social interaction cause
irrational anxiety, develop rejection
through ~~from~~ ~~from~~ peers.

⑤ Panic disorder :-
Sudden Episode of intense fear & anxiety

⑥ OCD :- Obsession & Compulsive Behaviour
develops.

* Patho.

There are 3 phase in anxiety disorder

No Anxiety Symptoms Disorder	Subsyndromic Symptom	DSM-IV Anxiety Disorder
------------------------------------	-------------------------	------------------------------------------

*) Mainly all of people have experience
the anxiety occasionally.

The 5 approaches are given

① Noradrenergic model :-

This model suggest that



Hypersensitive & overreacts of autonomic nervous system



Activation & release of Norepinephrine regulate by locus ceruleus cause stimulation of parasympathetic & sympathetic nervous system.

* Chronic hypersensitive cause RAD & posttraumatic stress disorder (PTSD)

SAD → hyperresponse of corticocortical to stress

② ✓ Aminobutyric (GABA) receptor model :-

Underactivity or downregulated of GABA or benzodiazepine receptor



Abnormal response to stress & stimuli



Anxiety (PTSD)

→ In RAD, benzodiazepine partially bound
In SAD, GABA receptor malfunction.

③ 5HT model:-

Hyperactivity or ~~excessive~~ Excessive transmission of 5HT

↓

In SAD, 5HT & Dopamine system involved

④ Corticotropin model

Hypersecretion of corticotropin releasing factor

↓

Abnormal level of cortisol

↓

PTSD

⑤ ~~Amygdala model~~

⑤ Neuroimaging studies differ amygdala

(a) Increase in brain fear circuitry & ↑ activity in prefrontal cortex

↓

GAD

(b) Abnormalities of midbrain structure

↓

Panic disorder

(c) Hypoactivity in amygdala & insula

↓

SAD

(d) Persistence of traumatic memory
↓
PTSD

Continue in another book.

Hyposecretion of corticotropin
releasing factor
↓

Dysregulation of
hypothalamic-pituitary-
adrenal axis
↓

Abnormal cortisol
level.
↓

~~Trauma~~

PTSD

Q2 Clinical manifestation & treatment of anxiety disorder.

→ Clinical manifestation

(i) Cause or Etiology

+ Stress

- Life challenges fear

- Drug side effect

- Trauma

- Genetic

- Abnormalities of brain function

- Depression

- Excess alcohol

- Gender

- Insomnia

- Unhealthy meal

(ii) Sign & symptom

Signs :- Phobic behaviour.

- Sweating in social gathering

- High heart rate in stress

- Headache

- Dizziness

Symptoms.

- Excessive worry
- Nausea
- Diarrhoea
- Impatience
- Mind going blank
- Poor concentration

① GAD

- Restlessness
- Fatigue
- Poor concentration
- Muscle tension
- Irritability
- Sleep problems

② Panic disorders

- Heart palpitation
- Sweating
- Shaking
- Feeling out of control

③ Phobia

- Fear from particular
- Avoid the situation
- Immediate

④

- SAD : Chest pain , Palpitation
- Chills , Sweating
 - Itch/clashe , Trembling

② PTSD :- Avoidance Syndrome

- Traumatic event
- Anhedonia
- Insomnia
- Anger burst

+ Diagnosis

① CVs examination

② Behaviour examination

③ Psychiatric diagnosis

④ Respiratory system evaluation

⑤ Monitoring or reviewing Drug therapy

⑥ DSM-5 criteria - completion

+ Treatment

+ Drug

① CAD :- 1st line :- Duloxetine :- 30-60

Escitalopram :- 10mg

Paroxetine :- 20

Venlaflexin :- 75

Sertraline :- 50

OD

2nd line :- Benzodiazepam

Bupropion :- 7.5 mg TID

Imipramine :- 50mg

Megaboline :- 150 mg

② SAD : 1st line : Escitalopram
 Paroxetine
 Sertraline
 Venlaflexine

2nd line : clonazepam 1-4mg
 Citalopram 20-40mg

3rd line Carbapentine

③ Panic disorder

1st line :- SSRI

- clonazepam :- 1-4mg
 - Venlaflexine :- 75mg

LCAD

2nd line :- Alprazolam :- 4-10
 Clonazepam :- 1-4
 Diazepam :- 5-20
 lorazepam :- 2-8

~~Anxiety drugs like :-~~

④ PTSD : 1st line :- Paroxetine
 Sertraline

2nd line :- Amitriptyline :- 75
 Imipramine :- 75
 phenelzine :- 45

Algorithms

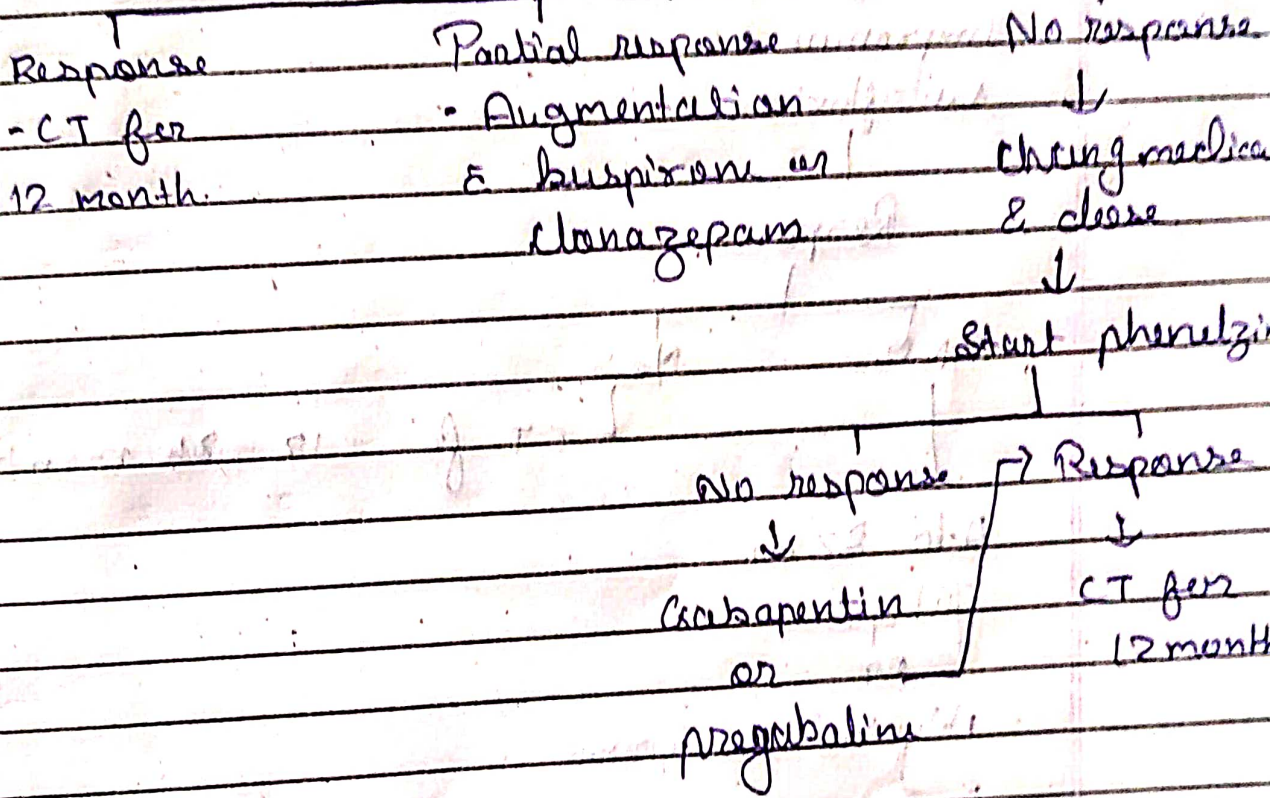
①

AND & SAD

↓

SSRI or SNRI

for 12 week

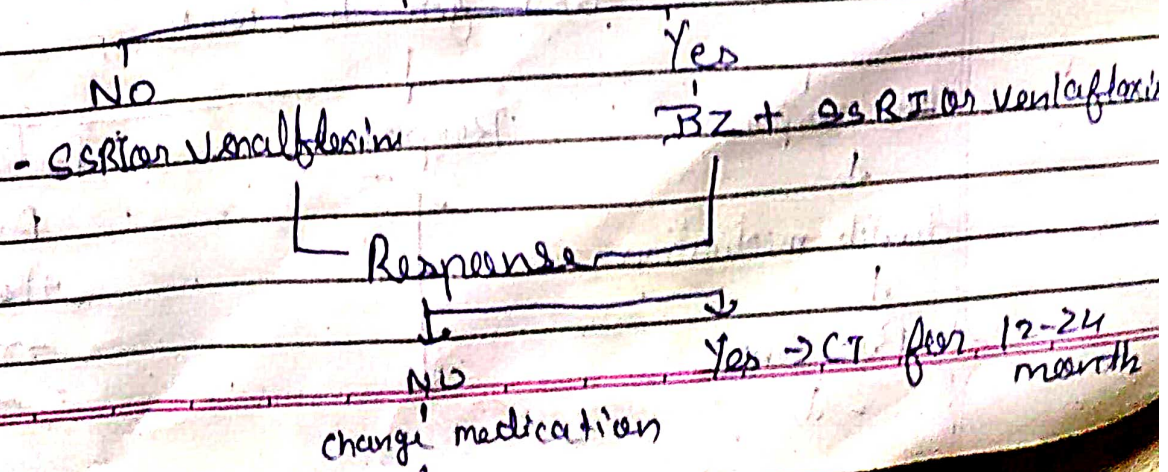


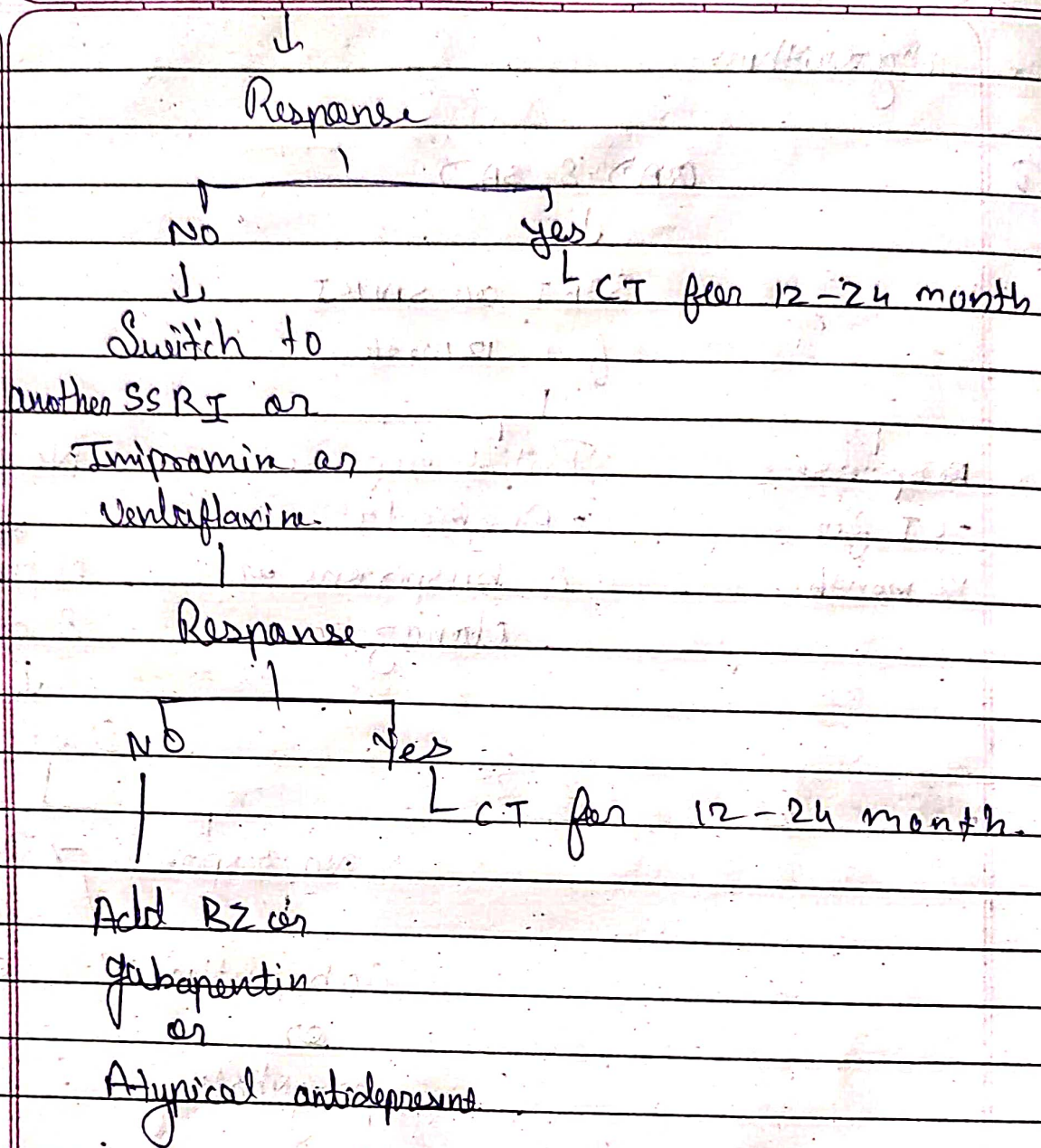
②

Panic disorder

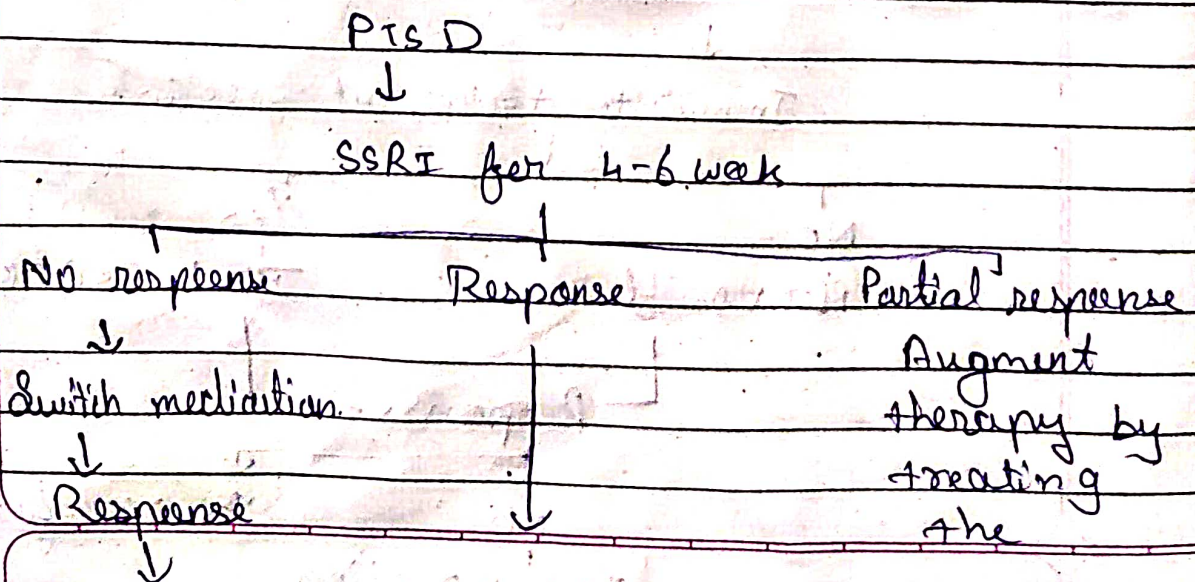
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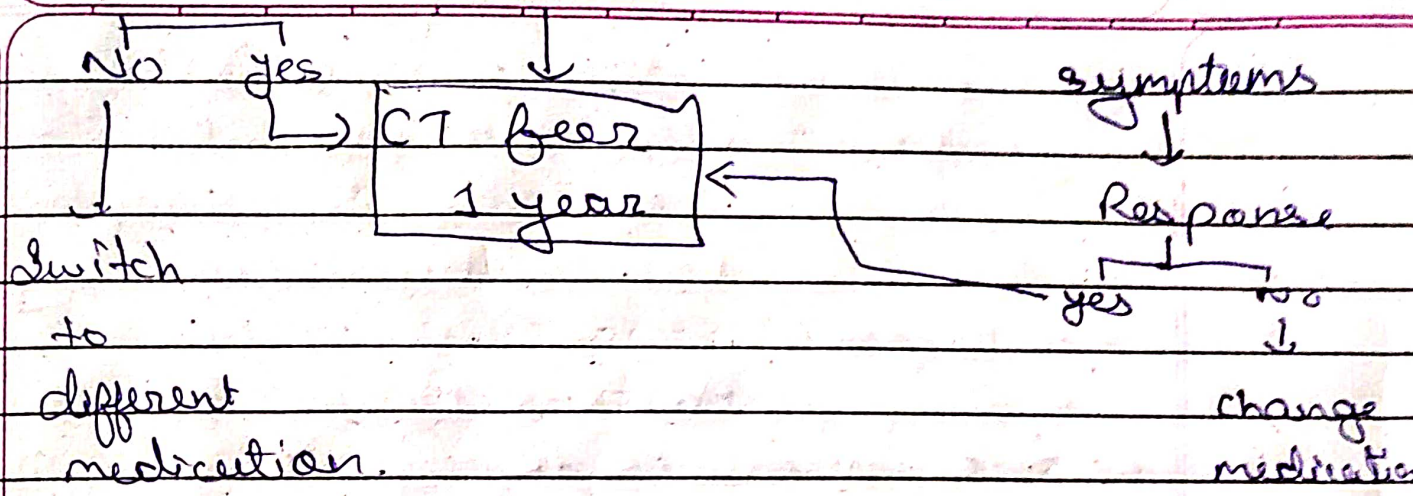
Immediate treatment require





③ PTSD





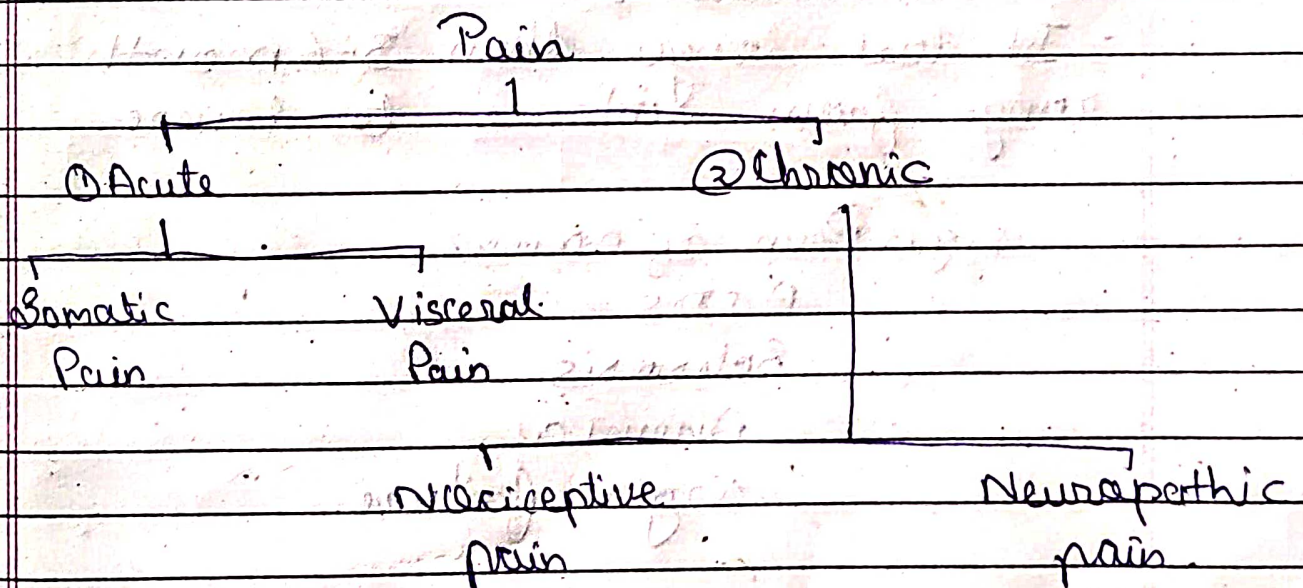
* Pain Management

Q1 what is pain define, note on types & patho physiology.

→ Pain derived from Latin word *poena* which means penalty or punishment.

→ Define: An unpleasant sensory and emotional experience associated with actual or potential tissue damage or in terms of such damage.

* Types



① Acute Pain is The pain of a shorter duration that subside healing process.
- Rang from mild to intense.

(1) Somatic pain - Results of activation of nociceptors sensitive to noxious stimuli in cutaneous or deep tissue.

- Experienced locally & constant
- e.g. Pain in cancer patient.

cb) Visceral Pain: Mediated by nociceptors

- Deep, aching & colicky experienced.
- Diffuse site which may tender
- e.g. Stretching of viscera by tumor growth.

② Chronic Pain :-

- It last longer than 3-6 month & range from intense to severe.

e.g. Pain of cancer

- AIDS
- Sclerosis
- Anemia
- Organ failure.

① Nociceptive pain :- It may be visceral or somatic.

- Arise from tissue inflammation, mechanical deformation, ongoing injury.

cb) Neuropathic pain: Involve PNS & CNS.

- It does not respond predictable as nociceptive pain to analgesic.

Pathology

Pathophysiology classified into 2 pathways

- ① Nociceptive
- ② Neuropathic

① Nociceptive pathway involves

(a) Stimulation

(b) Transmission

(c) Perception

(d) Modulation

Delta fibres - sharp, local pain

C fibres slowly acting pain

Explanation

are further



Noxious (painful) Stimuli

↓ Stimulation

Activation of peripheral nervous system.



Production of action potential (AP).

② Transmission



AP reaches dorsal horn of spinal cord. Activation of CNS at spinal cord

③ Modulation



Transmission of pain signal to brain
Nociception inhibiting neurons

Brain respond to
Stimuli

(3) perception

② Neuropathic pain

Nerve damage / persistent stimulation



Rewiring of pain circuit both
anatomically & biochemically
causing



Spontaneous
nerve stimulation

Autonomic
neural
stimulation

Increased
discharge of
dorsal horn
neurons

leads to



Neuropathic pain.

- iii) Nerve damage or persistent stimulation may cause pain circuit to rewire themselves both anatomically & biochemically. This produce
- Spontaneous nerve stimulation
 - Autonomic neural pain stimulation
 - Progressive increase in the discharge of horn neurons

Q2 Note on clinical manifestation & treatment of pain management.

Ans Clinical manifestation

① Etiology

- Drugs
- Injury
- Trauma
- "

② Symptoms & Signs

~~Signs~~

There are ~~not~~ any specific symptoms or signs but experience an sharp, dull, burning, shock, tingling, shooting type of pain

Ans Non specific symptoms like : Anxiety

- Fatigue
- Insomnia

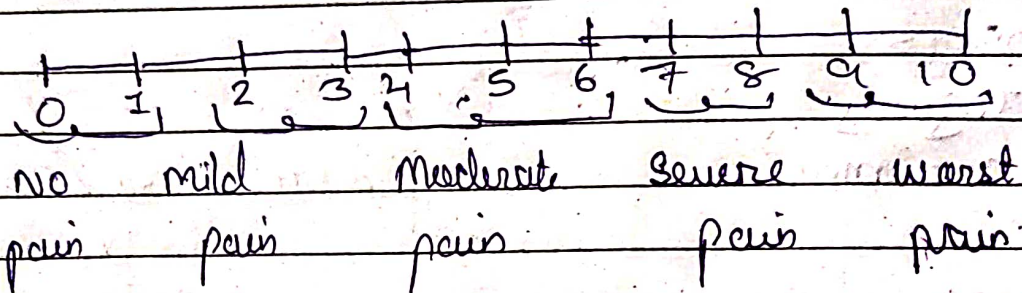
- Anger

- Irritation

- Fear

② Diagnostic test

- ↳ There are no specific test for pain but a pain scale are used to measure pain



numeric pain intensity scale

- + Patho with proper explanation

① Stimulation

- ↳ The first level to pain stimulation at free nerve ending.
- ↳ The receptors are found in skeletal & visceral structure
- ↳ Receptor activation leads

↓

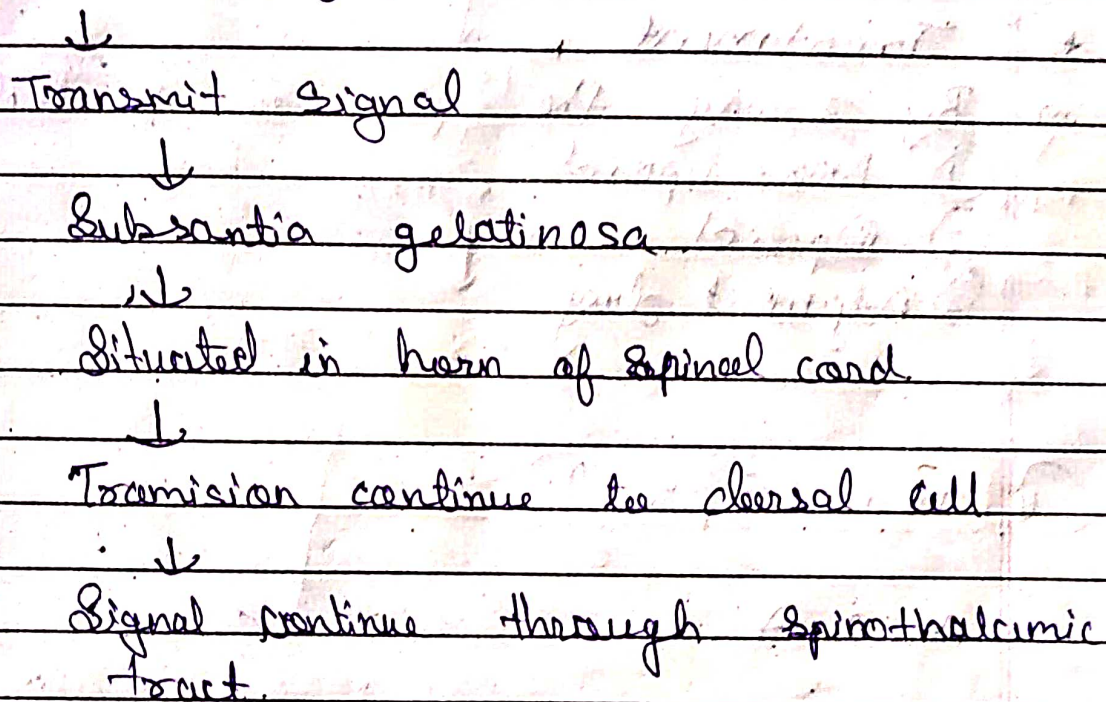
secretion of histamin,
bradykinin, PGE₂,
serotonin

↓

develop
action potential
in nerve fibre
of spinal cord

② Transmission

- Transmission take place in A delta & C afferent fibre
- Stimulation of myelinated "A" delta leads to sharp, localized pain whereas in unmyelinated C fibre cause dull, aching & poorly localized pain.
- C fibre & A fibre



② Pain perception.

- Brain accommodate limited number of signals thus cognitive & behavioural function can modify pain.

④ Modulation

- By 2 method modulation of pain done
- Inhibition of nociception impulse

① Endogenous & exogenous opioid that help in ~~moder~~ inhibiting pain impulse

* Treatment *

⇒ By 3 way the pain is treated

- ① Non-Opioid
- ② Opioid
- ③ Adjuvant drug

① Non-Opioid

M.O.A

NSAIDs



Prevent formation of
P & except acetaminophen



Decrease no. of pain impulse



Relieve pain.

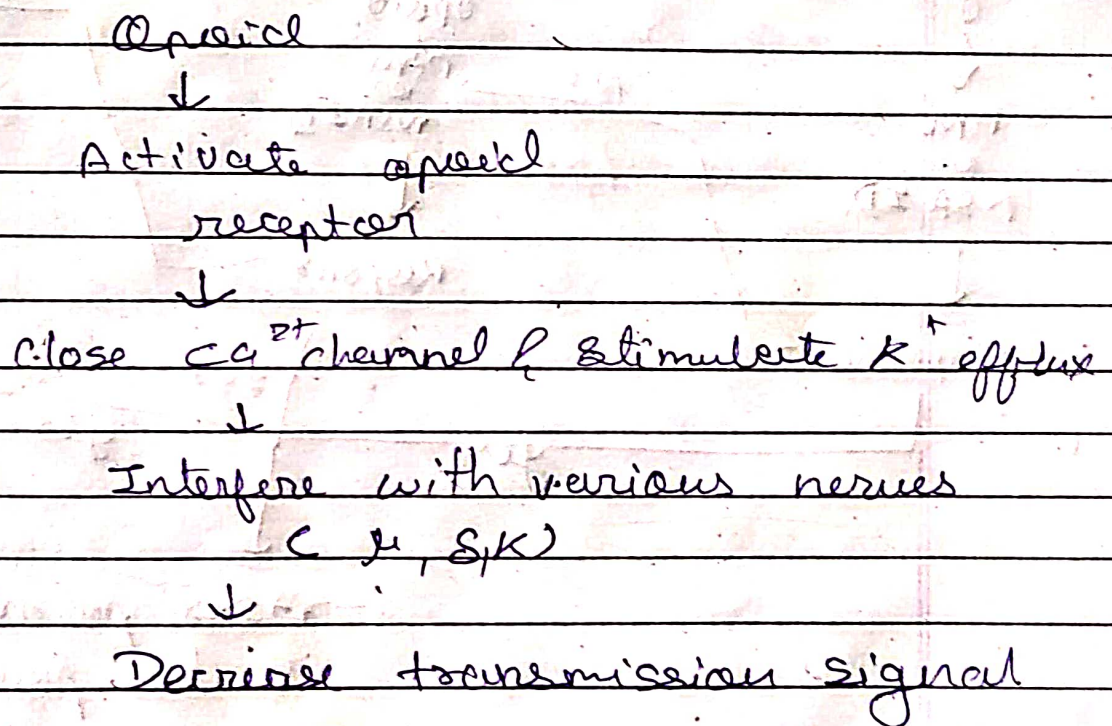
e.g. Paracetamol 325-600mg

Meclofenamate 25-100mg

Diclo : 50-100mg
Neyentamo A : 250-500mg

(2) Oprioid

MoA.



e.g. Codeine : 30-60mg
methadone : 2.5-5mg
Tramadol : 50-100mg

(3) Adjuvant : use to increase potency of drug

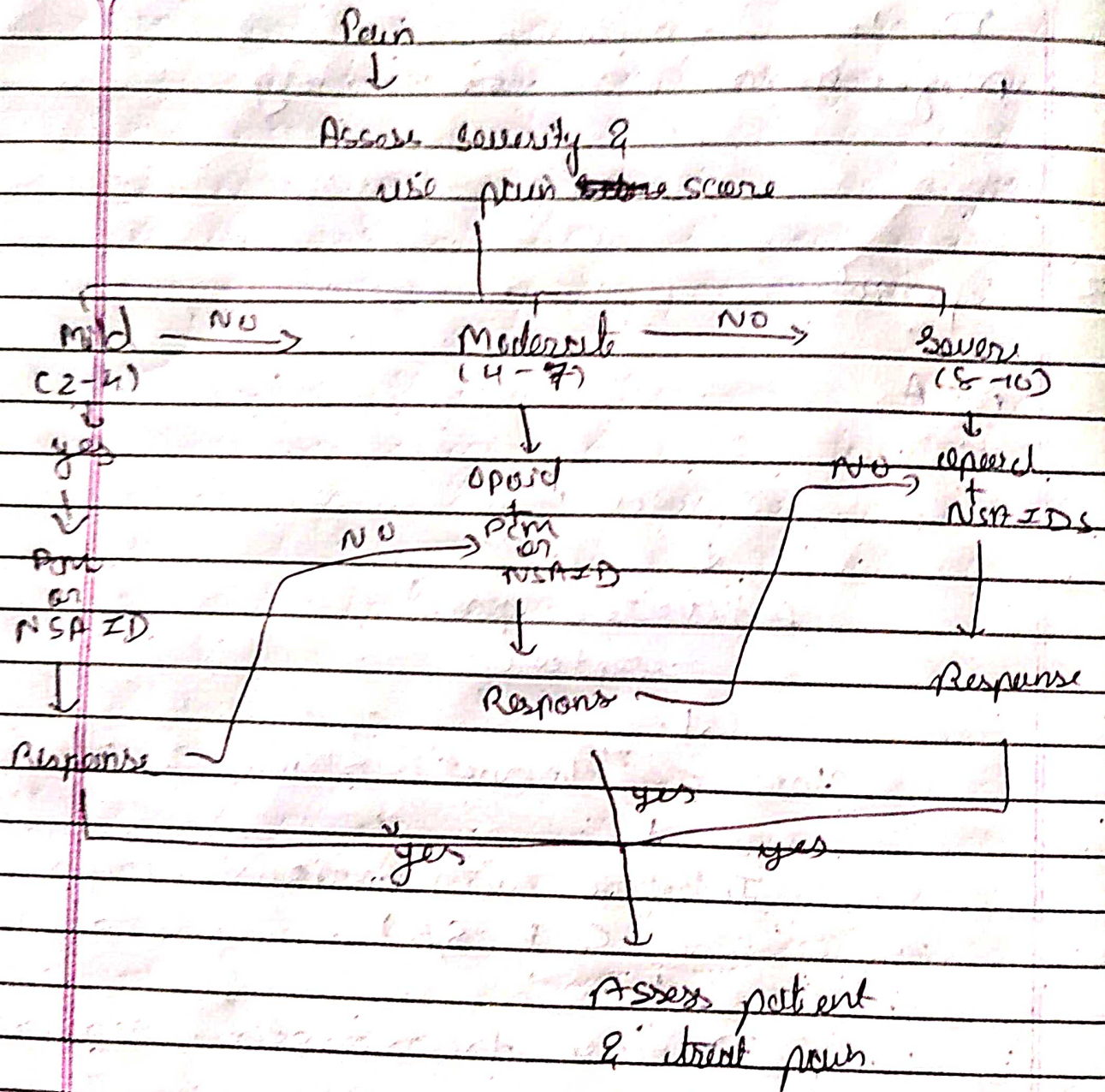
(1) T&A : Amitriptyline

(2) Anticonvulsant : Carbamazepine, Valproate

(3) Steroid : Prednisolone, Dexamethasone

(4) Anxiolytic : Diazepam, Cerevizepam

(5) muscle relaxant : Baclofen.



Q1) Define, types & patho of neuralgia

Ans) 1st Neuralgia refers to severe, shooting or stabbing pain that occurs along the course of a nerve or group of nerve.

It is a type of ~~acute~~ chronic pain that can be caused by various factors such as nerve damage, inflammation, or pressure on nerve.

The pain associated with neuralgia is usually severe, sharp, and sudden and can trigger by even minor stimulation.

* Types of neuralgia

1) Trigeminal Neuralgia: It is a type of neuralgia that affects the trigeminal nerve, which is responsible for sensation in the face. Sudden and severe pain on face by minor triggering stimuli like touching, eating or speaking, chewing.

2) Occipital neuralgia: It affects the occipital nerve, which run from the top of the spinal cord up through the scalp. It is characterized by severe pain in the back of head accompanied by sensitivity of light, sound or movement.

- 3) Glossopharyngeal neuralgia : That affect the glossopharyngeal nerve responsible for sensation and movement in the throat and tongue. It is characterized by sudden and intense pain in the back of throat that can be triggered by swallowing, speaking, or even breathing.
- 4) Postherpetic Neuralgia : That occurs after an episode of shingles. It is characterized by persistent pain and sensitivity in the area where the shingles rash occurred, even after the rash has healed.
- 5) Neuropathic Pain Syndrome : Neuropathic pain syndrome is a type of chronic pain that cause by nerve damage or dysfunction. It can occur in any part of body and characterized as burning, tingling or shooting pain cause numbness or weakness.
- 6) Phantom Limb Pain : That occur after amputation of a limb. Characterized by pain and other symptom in missing limb.
- 7) Meralgia Paresthetica :- It affect the lateral femoral cutaneous nerve runs from spinal cord to thigh.

• Treatment

• Pharmacological

1. Anticonvulsant

(a) Gabapentin :- 300mg/day Tds

(b) Pregabalin :- 75mg/day Tds

(c) Carbamazepine :- 100mg Bds

2. T.T.C

(a) Amitriptylline :- 10-25mg OD

(b) Nortriptylline :- 10-25mg OD

~~(c) SSRI~~

3. SSRI

(a) Duloxetine :- 30-60mg OD

(b) Venlaflexine :- ~~30~~ 37.5mg OD

4. Opioids

(a) Oxycodone :- 5mg @ 6 hr

(b) Morphine :- 10mg @ 4-6 hr

5. Topical Medication

(a) Lidocaine patches :- 1-3 patches @ 12 hr

(b) Capsaicin cream :- 4-6 time a day

6. Corticosteroid

(a) Prednisolone :- 20-60mg (qd)

(b) Methylprednisolone :- 16-48mg (qd)

(c) Dexamethasone :- 4-24mg (qd)

* Trigeminal nerve Hz.

* Nonpharmacological [CBMP, CSR]

- ① Glyceral rhizolysis
- ② Balloon micro compression
- ③ microvascular decompression
- ④ Peripheral neurectomy
- ⑤ Linear coagulator radiosurgery
- ⑥ Cryotherapy
- ⑦ Stereotaxic radiosurgery
- ⑧ Radiofrequency gangliolysis

ham Bao bing

Date _____
Page _____

1st line : Carbamazepine :- 100-300 mg/day

2nd line :- phenylephrine :- 200-300 mg
50 60

- Barlefen :- ~~200~~ ^{50 60 8} ~~(300)~~ mg

- lumetri gine 25-50mg

3rd line Clonazepam :- 4-8mg.

Valproic acid! 20-80mg

Охарактеризуй: Зое - Гого.

Q1) Definition, types & patho of headache.

Headache is a pain or discomfort felt in the head or neck region, and can range from mild to severe in intensity.

* OCD *

Q1) What are definition, types & patho of OCD.

OCD is a mental health disorder characterized by recurrent, unwanted, and intrusive thoughts, image, or impulse that cause distress, and repetitive behaviour or mental act that aimed at reducing anxiety or preventing harm.

* Types [AH MOD SBT

① Aggressive & sexual thought

② Harm to close ones

③ Germ and contamination

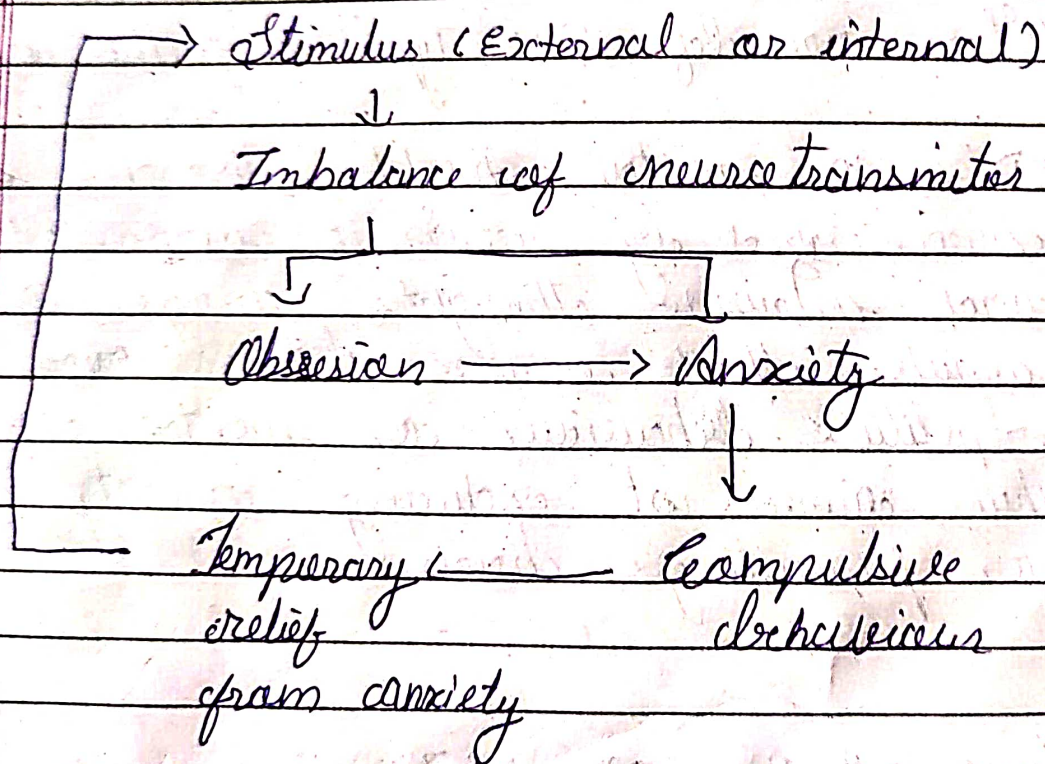
④ Order and symmetry

⑤ Doubt and incompleteness

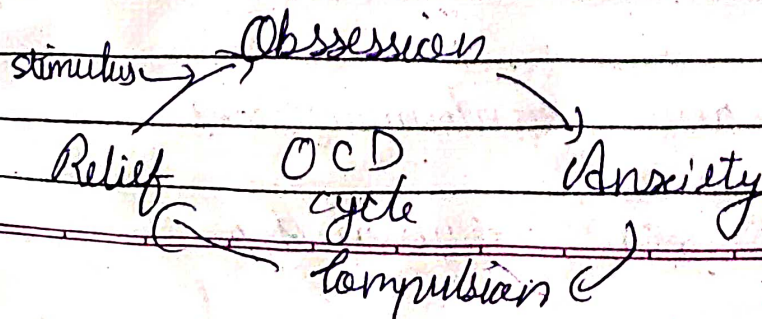
⑥ Self Control

⑦ Religion & mortality

* Pathogenesis



* Cycle repeats.



② Diagnosis

① Psychological evaluation

② DSM-5 (Diagnostic and Statistical Manual of Mental Disorder)

③

* Treatment

+ N. pharmacology

① Psychological

② CBT

③ Neuromodulator

④ Social Support

⑤ TMS :- Transcranial magnetic stimulation

+ Pharmacologicals

① SSRI

(a) Fluoxetine :- 40-80mg / day

(b) Fluvoxamine :- upto 300mg / day

(c) Escitalopram :- 10-20

(d) Citalopram :- 20-40

Sertraline : 50-100 mg

② TCA :- Amitriptyline :- 75-150 mg
Imipramine :- 11
Tukomipramine :- 11

③ SNRI :-

Venlafaxine :- 75-375 mg
Duloxetine :- 60-120 mg

③ Atypical antipsychotic

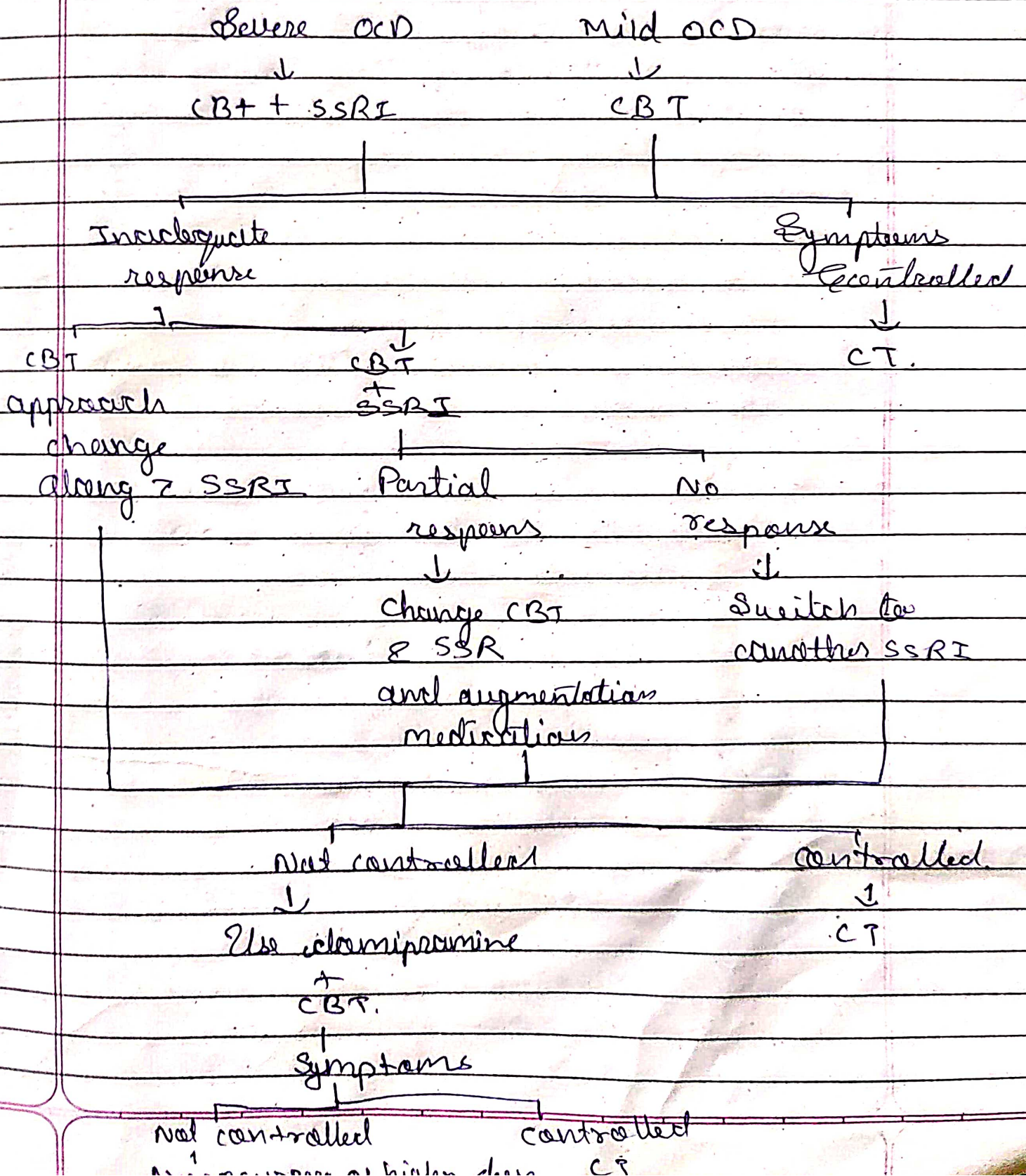
① Aripiprazole - 10-15 mg

② Quetiapine :- 50 mg BD

③ Risperidone :- 0.5 - 1 mg BD

④ Chlorzypin :- 12.5 - 50 mg

Algorithm



Q14 Alzheimer

patho

Etiology

accumulated protein

↓ toxicity

change of nerve cell functioning

↓ accumulation of neurofibrillary tangle & plaques

↓ loss of cholinergic nerve

↓ loss of memory

AD

management

i) cholinesterase

ca) Rivastigmine 1-3-6

cb) Donepezil ~ 5-10

cc) Galantamine 8-12

cii) Alimem, D-aspartate

memantine - 10mg -> 28mg

ciii) Antipsychotic

AD

↓ cholinesterase

↓ Response

yes
CT

no
↓

change mechanism
Response

yes
CT

no
↓

2 cholinesterase
1 NMDA

↓ Response

yes
CT

no
↓

Increase dose

or change combination

* Parkinson *

Date _____
Page _____

* Describe patho, clinical manifestation, neurochemical basis & treatment of Parkinsonism.

Ans. It is progressive degenerative disorder.
* Patho

Environmental factors
and other causative
agent.

↓
Affect the region of
brain called substantia
nigra

↓
Destruction of dopamine producing
neuron within the basal
ganglia.

↓
Reduce the amount of available
dopamine

↓
This cause increase in ach
concentration.

↓
Result in imbalance concentration
of ach.

↓
This affect difficulty in controlling
and initiating the voluntary
movement.

Clinical manifestation

① Sign & Symptom

- General

- Speech abnormality
- Stooped posture
- Impaired postural reflex

- Gait

- Shuffling walking
- Motion arm deterioration
- Impaired balance

- Tremor

- Pill rolling

- Leg, jaw & chin affected

- On stretching out arms

- Rigidity

- Akinesia

- Bradykinesia

②

Causes

- Drug & toxin
 - Antipsychotic
 - Metoclopramide
 - Tetrabenazine
 - Lithium, Mg.
- Genetic
 - Wilson diseases
 - X-tremor ataxia
 - Huntington's diseases
- Other
 - Dementia & Lewy body
 - Atrophy
 - Alzheimer

③

Investigation or Diagnosis

- Physical Examination

- ① Classic rest-tremor
- ② Muscular rigidity
- ③ Postural movement
- ④ Response to stimuli

- History :- Repeated stroke or Head injury

- Imaginative technique :- MRI or CT scan
or SPECT-scan.

Treatment

Diagnosis of P.D.

Non pharmacological

- Education
- Exercise
- Nutrition
- Psychological support

Pharmacological

Rasagiline

Symptomatic management

Anticholinergics $\xleftarrow{<65y}$ Tremor $\xrightarrow{>65}$ Amantadine

Amantadine, Dopamine agonist or carbidopa

Bradykinesia, rigidity \rightarrow same

Motor fluctuation management

- Res dose of levodopa
- AChE MAO-B inhibitor
- Dopamine agonist

Dyskinesia Management

- Res Dopamine agonist dose

+ Amantadine

manage symptom

Surgery

Drugs

① Anticholinergic

- Benztropine 1-6 mg/day

② Carbidopa / Levodopa ~~100/100~~ 10/100 - 25/250 mg

③ Dopamine agonist

- Bromocriptine 15 - 40 mg/day

- Pramipexole 1.5 - 4.5 mg/day

~~Ropinirole~~

- Ropinirole 9 - 24 mg/day

④ COMT / inhibitor

- Entacapone 200 - 600 mg/day

- Tolcapone 300 - 600 mg/day

⑤ MAO-B Inhibitor

- Rasagiline 0.5 - 1 mg/day

- Selegiline 5 - 10 mg/day

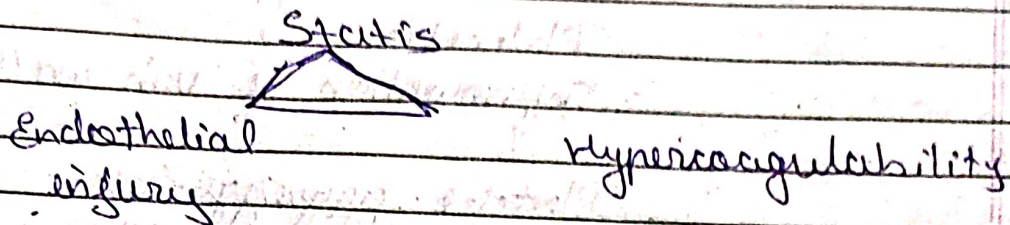
⑥ Amantadine 200 - 300 mg/day



* Venous thromboembolism *

Ans → Pathogenesis, clinical manifestations & Treatment
Also called Deep Vein thrombosis.

→ There are 3 conditions that predispose thrombus formation by Virchow's triad.



Virchow's triad.

Endothelial injury

- Smoking
- Hypertension
- Surgery
- Trauma

Hypercoagulability

- Genetic
 - Factor V Leiden
 - Prothrombin
 - Protein C & S deficiency
- Acquired
 - Cancer
 - Drug
 - Obesity

Stasis

- Immobility

* Pathophysiology

Venous stasis and skeletal muscle contraction reduce



Damage to intimal lining of blood vessels



Phlebitis

- Inflammation of vein wall.



Platelet aggregation

- contain tail like fibrin, WBC & other cells



This tail grows & form layer of thrombus



Fragmentation



Recanalization

* Clinical Manifestation *

① Sign & Symptoms

- Local Swelling or Edema
- Tenderness
- Pain, erythema (skin redness)
- Pulmonary embolism
- Skin colour changes

- Palpitation
- Chest pain
- Cough & S.O.B

② Causes

- Immobility
- Surgery
- Obesity
- Injury
- Pregnancy

③ Risk factor

- Age
- Cancer
- Surgery
- Heredity
- Smoking
- Protein C & S deficiency

④ Investigation or Diagnosis

- Physical Examination:- Swelling
- Tenderness
- Skin colour
- Pain
- Laboratory report - CBC
- D-dimer test

- Imaging technique

- USG

- Venography

- MRI

- Chest - X-ray

- CT Angiography

- Other - Well's criteria

- Paucla score

* Treatment.

VTE diagnosis



Anticoagulant Yes → Vena cava filter
contraindications

↓ No

Thrombolytic Yes Pulmonary Embolism
therapy, if bleeding
risk acceptable

diagnosed

↓ No

↑ Yes

Proximal DVT

↓ No

→ start anticoagulant =
unfractionated Heparin

or

LMWH or fondaparinux
= warfarine



Warfarin or rivaroxaban
for 3 month

VTE provoked by Yes → stop anticoagulation
Risk factor
↓
No

Continue therapy

Drug

~~① Unfractionated heparin :-~~

① Warfarin :- 5mg/day

② Enoxaparin - 1mg/kg S.C. @ 12h

1.5mg/kg SC OD

③ Dalteparin :- ^{initial} 200 U/kg OD x 30 day

Maintenance 150 U/kg OD

④ Dabigatran :- 150mg p/o BD 5-10 day

⑤ Apixaben :- 10mg BD - 7 D.

⑥ Rivaroxaban - 15mg p/o BD x 21 day

⑦ Unfractionated heparin :- 5000 - 10000 U

* Drug induced Blood disorders

Date _____
Page _____

Q1. Write note on management of drug induced blood disorders or write classification and describe them.

1) Mainly drug cause the bleeding risk 2, that lead to severe condition.

3) Drug like Anticoagulant :- Heparin, warfarin, desirudin

Antiplatelet :- Aspirin, dipyridamol, clopidogrel

Oral anticoagulant :- rivaroxaban, Apixaban

NSAID :- ^{low risk} celecoxib, ibuprofen, meloxicam

^{High risk} - Indomethacin, naproxen, ketorolac

selective norepinephrine reuptake inhibitor SNRIs - duloxetine, Venlafaxine

SSRIs - citalopram etc

- This are mainly responsible for bleeding.

Classification

1) Thrombocytopenia

2) Thromboembolic disorder

3) Anemia

- Drug induced

Aplastic anemia

- " "

Hemolytic anemia

- " "

Neutropenia - agranulocytosis

- " "

megalooblastic anemia

1) Thrombocytopenia.

↳ It is a condition in which platelet count fall below $1,50,000 \text{ cell/mm}^3$.

↳ Normal - $1,50,000 - 4,50,000 \text{ cell/mm}^3$

↳ There are 2 type of thrombocytopenia.

(I) Immune :- Body produce antibodies, which destroy platelet e.g. Heparine

(II) Non Immune :- Medication prevent formation of platelet ~~from~~ by suppressing bone marrow e.g. chemotherapy drug & valproic acid

• Drug cause thrombocytopenia

[Q, N, S, A, P, R, F, Q]

- Quinine

- NSAID

- Sulphonamide - Cefaclor

- Penicillin

- Ranitidine

- Furosemide

- Quinine

* Clinical manifestation

1) Signs & Symptom

- Abnormal bleeding
- Bleeding during brush
- Pin point red spot on skin
- Fatigue
- Lethargy
- Jaundice
- Heavy menstrual flow

↓
ITP

* Cause :- Drugs are above

* Diagnostic or investigation

- Physical examination

- Fatigue
- weight loss
- Skin red spots
- Weakness

- Laboratory :-

CBC

~~Prothrombin time~~

- Bleeding time
- Bone marrow test

2 Treatment

1) Stop heparin (if patient are taking)

2) Stop drugs that causes

3) IVIC therapy • Intravenous Immune Globulin therapy
400-800mg/kg

4) Plasma exchange - HSE/T

5) Platelet transfusion

→ Low Cyclosporine - 4-6mg/kg 3-4 days

6) Corticosteroid medication - methylprednisolone

1000mg/kg - 4 weeks

2) Drug Induced Thromboembolic Disorder

→ Same as VTE or DVT.

3) Drug induced Aplastic Anemia

→ Aplastic anemia is a rare disease in which the bone marrow and hematopoietic stem cell are damaged.

→ This lead to failure of production of RBC, WBC, platelet.

→ Pluripotential hematopoietic stem cell are damage before their differentiation.

1) Clinical manifestation

1) Sign Symptom

- Fatigue
- Headache
- Lethargy
- Neutropenia
- Fever
- Chills

2) Infection

- Tachycardia
- Weakness
- Pale skin

2) Cause - Drug like [CCI APAC]

- Chloramphenicol
- Cefixime
- Indomethacin
- Amodiaquine
- Phenytoin
- Carbimazole

3) Diagnosis

- Physical examination
- Lab report = CBC
- LFT
- Bone marrow biopsy

+ Treatment.

- ① Blood transfusion
- ② Corticosteroid *methylprednisolone* : 1mg/kg x 1 week
- ③ Bone marrow transplant
- ④ Immunosuppressive agent - cyclosporin 4-10 mg/kg
- ⑤ HSCT
- ⑥ Anti thymocyte globulin - 120mg/kg x 1 day
- ⑦ Hematopoietic stem cell transplant

4) Drug induced hemolytic anemia.

→ when medicine trigger the immune system to attack its own red blood cell.
thus called hemolysis

* Clinical manifestations.

+ Sign Symptom

- Dark urine
- Fatigue
- Pale skin, color
- S.O.B
- Jaundice

+ Cause :- Drug like

- Antibiotic
- Dapsone
- levofloxa
- NSAID
- Penicillin

Diagnosis

- HbU
- Auto h. l. test
- Reticulocyte
- Hemoglobin

Non-Drug is :-

- Oxidant injury
- Methemoglobinemia

+ Diagnosis

- Physical examination :- Skin color
- Fatigue
- Urine
- Lab report :- CBC
- LFT
- Urine test

- Coombs' test

+ Treatment

- ① Stop drugs
- ② Immune suppression - Cyclosporine 4-6 mg/kg
- ③ Blood transfusion
- ④ G6P deficiency should be managed
- ⑤ Corticosteroid - Methyl prednisolone 1 mg/kg
- ⑥ IVIG - 100-200 mg/kg

5) Drug induced agranulocytosis & Neutropenia

↳ Neutrophil count $< 1500 / \text{mm}^3$ - 2

↳ Non-cytotoxic drug lower neutrophil count are called agranulocytosis.

↳ More in female.

* M.O.A.

↳ Disorder or defect of pluripotent myeloid stem cell & committed myeloid progenitor cell that cause decrease neutrophil production include neutropenia, anemia & myelodysplastic Syndrome.

* Clinical manifestation

- Sign & Symptom

- Sore throat

- Pharyngitis

- Mouth ulcer

- Fever

- Weakness

- Pallor

- Sinusitis

Neutropenia

phil count

hyaloid
genitor
production
elastic

Cause: Drug like

- Diclofenac, Ibuprofen
- Quindine
- Cephalosporin
- Penicillin
- Ampicillin
- Infliximab
- Clopidogrel
- Fluoxetine

DC Pta
IQ

* Diagnosis or Investigation

- Physical
- Lab report :- CBC
- Bone marrow biopsy
- Other symptomatic report
- CRP, ESR, Urine

* Treatment

- ① Removal of drug causes
- ② Sargramostim - 250 mcg/kg/day @ 4 hr
- ③ Filgrastim - 5 mcg/kg/day

@ Supportive care

- Symptomatic
- Airway
- B.P.

B] Drug induced megaloblastic anemia

- Abnormal development of RBC in bone marrow
- It develop by disorders of DNA synthesis, due deficiency of B_{12} & folic acid

* M.O.A

① Cotrimoxazole & MxT bind to dihydrofolate reductase & inhibit their activity result in this type of anemia.

② Zidovudine disrupt the DNA synthesis

* Clinical manifestation

• Sign & Symptom

- Fatigue
- Headache
- Tachycardia
- weakness
- Lethargy

② Cause :- Drug like

IRAM SN of ~~D~~

- | | |
|-----------------|------------------|
| - Ribavirin | - Sulfonamide |
| - Ascorbic acid | - Nitrofurantoin |
| - Metformin | - Dapsone |

Diagnosis

- Physical - weakness, fatigue, no mood
- Lab report - ~~WBC~~ CBC
- Vit. B12
- Folic acid

Treatment

- ① Folic acid - 5-10mg @ Qhr
- ② Stop drug causes anemia
- ③ Blood transfusion - ~~plasma~~ RBC
- ④ Supportive care - Oxygen, vitamin supplement
- ⑤ Symptomatic management
 - B12 deficiency
 - Iron deficiency
 - Folate deficiency
- ⑥ Corticosteroids - 60mg / day / 10 days

* Schizophrenia *

Q. What is Schizophrenia describe its ^{Types} ~~etiology~~, pathogenesis of it.

- 1) Schizophrenia means fragmented mind
- 2) Schizophrenia characterized by hallucination, disorganized thinking & speech, abnormal motor behaviour & negative symptom.

* Types

1) Paranoid Schizophrenia :-

- Common form
- Prominent hallucination or delusion
- Exaggerated in movies & by media
- Violent behaviour or suicidal

2) Disorganised

- Behaviour disorganised & purpose
- Speech is disorganised
- Pranks, giggling, health complaints
- Inappropriate emotional response

3) Catatonic

- Rare type
- Self injury or malnutrition risk
- Unusual physical movements
- No react to stimuli, odd & stiffy movement
- Negative symptom of Schizophrenia.

4) Undifferentiated

- ~~People~~ People who may not fit in any other classes.

- Exhibit +ve & -ve symptom

- characteristic of paranoid schizophrenia but differ in exact class.

5) Residual

- People have past history of -ve symptom of schizophrenia.

- Poor attention, emotional withdrawal

* Pathogenesis

By 3 ~~the~~ hypothesis this are give

1) Dopamine hypothesis

PET [Positron Emission Tomography] study shows



~~D₂ specific agent~~

D₂ receptor density increase in nucleus accumbens

&

D₁ receptor density decrease in prefrontal cortex



Causes



↓
+ve symptom due overactivity in
mesolimbic dopaminergic pathway
at D_2 receptor

↓
-ve symptom due to decrease activity
in mesocortical dopaminergic pathway
at D_1 receptor

② Glutamate hypothesis

Hypofunction of NMDA
receptor

↓
Increase activity
in mesolimbic

↓
Reduce GABA inhibitor

↓
Increase DA release
in limbic areas
such as nucleus
accumbens.

↓
Decrease ~~the~~ activity in
mesocortical neuron

↓
decrease dopamine release
in prefrontal cortex

③ 5-HT hypothesis

- On stimuli the amount of DA rises in
prefrontal cortex.

- In this schizophrenia higher 5HT concentration
& this correlate to increase ventricular size

Q2 Write a note on Clinical manifestation & treatment of Schizophrenia.

W. Clinical manifestation

- Sign & Symptom.

- Delusion
- Hallucination
- Disorganized speech
- Violent behaviour
- Poor functioning of execution
- Memory poor
- Flat affect
- Alogia
- Avolition.
- Asocialist.

→ Cause :-

- Genetic
- Neurobiology
- Infection
- Immune disorder
- Pregnant women getting flu which cause Schizophrenia to baby

Investigation

- Physical

- Laboratory : DSM IV

- History of Medicine

- CBC

- CT Scan

- MRI

- PET

Treatment

→ Non - P.

↳ Psychological therapy

- Electroconvulsive therapy

- Cognitive behavioural therapy

→ Pharmacological

① Phenothiazines : chlorpromazine : 50-150mg
Trifluoperazine 2-5mg

② Piperidine chain : Thioridazine 50-150mg

③ Piperazine chain : Trifluoperazine - 2-5mg

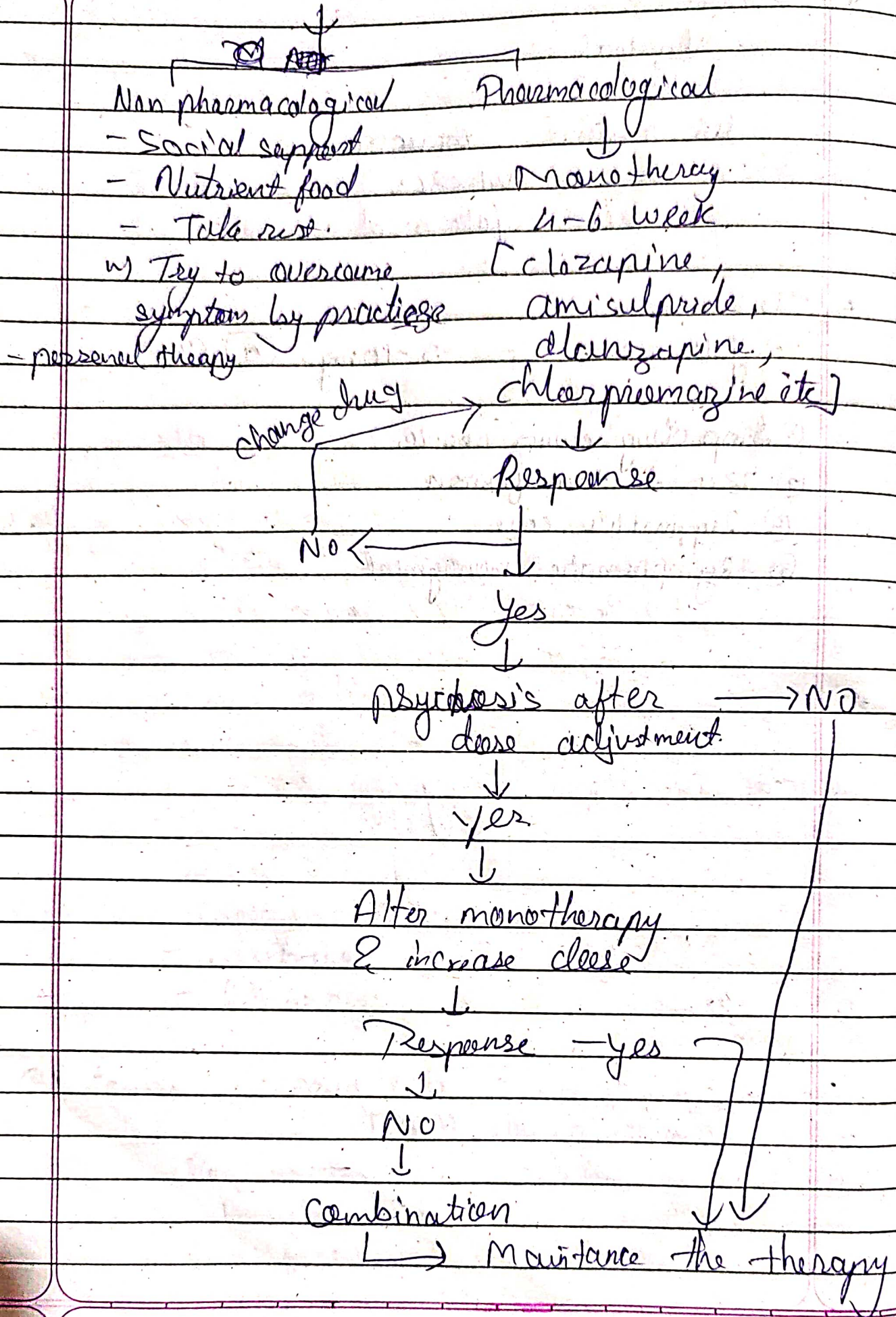
④ Thiohexane : Thiothixene 4-10mg

⑤ Other heterocyclic : loxapine 20mg

- ~~chlor~~

- Haloperidol 2-8mg

- Clozapine 25mg



* Sleep disorder *

Date _____
Page _____

Q1 Define & ~~note~~ write note on risk factor, classification & pathology of sleep disorder

Ans Sleep disorders are a group of syndromes characterized by disturbance in the pattern amount of sleep; quality or timing of sleep or in behaviour or physiological condition associated with sleep.

Ans To different sleep disorders.

Ans Sleep disorders may occur due to occupation or emotional stress.

Ans There are risk factors that may cause or enhance sleep disorders.

(i) Age:- More than 60-65 years old persons are more likely to sleep disorders due to any medication that interrupts their sleep.

(ii) Chronic diseases:- The chronic diseases cause insomnia due to induced pain of diseases.

Diseases like:- Diabetes

- Renal diseases

- Arthritis

- Heart diseases

- Fibromyalgia

(iii) Medication :- Medications increase risk of sleeping problem

e.g. Steroids

- Theophylline

- Phenytoin

- Verapamil

- SSRI

(iv) Gender :- Women more affected by insomnia than men

(v) Psychological factors :- Stress is the most affecting factor to sleep.

(vi) Lifestyle ~~Factor~~ Behaviour :-
- Drinking alcohol
- Smoking
- Exercising before immediate bed time
- Irregular morning & night schedule

(vii) Night shift work

(viii) Long-range travel

(ix) Poor Sleep Environment

Classification

1) ~~Tasomoria~~

2) Sleep apnea

3) Parasomnia

4) Dysomnia

5) ~~Disorder~~ ~~Sleep~~ ~~Disorder~~ Circadian rhythm disorder

6) Tet leg

7) Pathology

1) No concluded pathology is till now known.

2) There are many model but not explain real cause.

3) Researchers suggest due to Psychiatric, physiological & pathological condition the sleep ~~and~~ disorders occur.

4) Most precise theory is of deficiency of hypocretin ligand due to postnatal loss of neuron.

5) Dysfunction of supraspinal inhibitory triggering RLS (Restless leg syndrome) and PLMD (Periodic limb movement disorder).

Q2 Describe the sleep disorders with clinical manifestations & Treatment.

Ans (1) Insomnia

It is a type of sleep disorder & individual has difficulty in falling asleep.

There are 2 type of insomnia.

(i) primary :- Not directly associated with any other health condition.

(ii) Secondary :- Due to any other cause as health condition or pain, medication etc.

* Clinical manifestation

(2) Cause :-

- (a) Illness
- (b) Emotional stress
- (c) Medication
- (d) Depression
- (e) Chronic diseases
- (f) Alcohol, Smoking
- (g) Travelling
- (h) Occupation
- (i) Abil cramp
- (j) SOB
- (k) Pain

② Signs & Symptoms

- (a) Waking too early
- (b) Breaking sleep
- (c) Stay asleep
- (d) fatigue
- (e) Poor concentrations
- (f) social life disturbance
- (g) headache
- (h) Mood disorders
- (i) Irritability

③ Prognosis

- No certain or specific
- Sleep logs
- Symptoms monitoring for weeks

* Treatment

- Non pharmacological
 - Improve sleep hygiene
 - Stimulus Control Therapy
 - Restrictive Sleep Therapy

* Pharmacological treatment

① Benzodiazepines

- ~~Estazolam~~ lorazepam :- 0.5mg
- Flurazepam
- Triazolam - 0.125mg

(2) Benzodiazepine Receptor Agonists
 - Zolpidem 6-12 mg OD
 - Eszopiclone

(3) Melatonin Receptor
 Ramelteon 8mg before bed

(4) Serotonine precursors :-
 L-Tryptophan - 500mg

(5) Antipsychotic / Antidepressant

(2) Sleep apnea

m) Central or Biphase apnea during sleep, associated with frequent awakening and often with daytime sleepiness.

- Patient with this sleep disorder shows a high risk of morbidity and mortality

+ clinical manifestation

(1) Cause :- same.

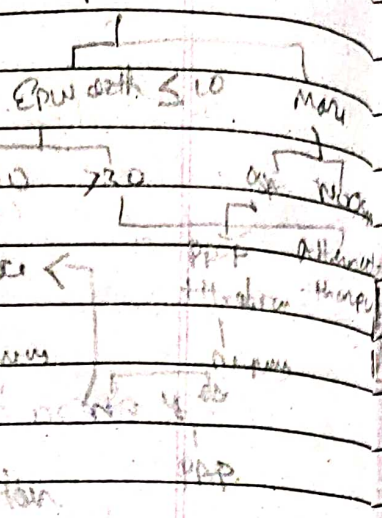
Sign Symptom

- Daytime sleepiness
- Loud snoring
- Awakening with choking
- Lack of concentration
- Change in mood
- LAEPD
- Nocturia

OSTA

↓

Ep work



Diagnosis

- Polysomnography
- Patient Counselling
- Overnight Oximetry
- Multiple sleep latency
- Sleep Diary

medication

FAP: Positive Airway Pressure
CPAP: Continuous Positive Airway Pressure
BiPAP: Bilevel Positive Airway Pressure
Ventilation: Mechanical Ventilation

Treatment

Non pharmacological

- ↳ Weight loss
- ↳ Nasal continuous positive airway pressure
- ↳ ~~Therapeutic pharyngoplasty~~
- ↳ Surgical

Pharmacological

- ↳ Tricyclic antidepressant e.g. Imipramine - 75mg
- protriptyline - 10mg OD

② clonidine :- 0.05 - 0.1 mg @ 12 hr

③ SSRI :- Paroxetine :- 20 mg OD
- Fluoxetine :- 20 mg OD

→ Pathway

Normal breath of air

↓
Enter to nasal passage

↓
Soft palate & uvula

↓
Pass through tongue base

↓
Throat muscle & vocal cord

↓
lung

Any obstruction into flow of this passage
lead to apnea

③ Parasomnia

→ It involve abnormal and unwanted movement, behaviour, emotion, perception and dream that occur while falling asleep or sleeping.

(iii) It is a dissociated sleep states which are partial arousal during the transition between wakefulness & NREM sleep or wakefull.

iv) There are 5 type of parasomnia

- | | |
|-------------------------|-----|
| (a) Restleg Syndrome | (3) |
| (b) Night Terror | (1) |
| (c) Somnambulism | (4) |
| (d) Sleep Enuresis | (2) |
| (e) REM sleep behaviour | (5) |

(a) Restleg Syndrome :- Wicentail nerve

cause :- Main cause unknown but may be

- Intense dyesthesia
- Worse bedtime
- Repetitive jerking
- Progressive age

Treatment :-

- Carbidoopa - levodopa - 25mg / 100mg qds.

- clonazepam :- 0.5 - 2mg qds.

(b) Night terrors

∴ Sleep terrors

- Emerge from stage 3-4 sleep
- Autonomic arousal is interpreted

u) primarily in children

u) Sudden arousal from slow wave sleep

- cry
- automatic behavioural manifestation of intense fear
- last for few minutes, sleep resumes

+ Treatment: Reassurance

- Diazepam

- Imipramine

(c) Sleep walking

u) Emerge out of stage 3 & 4 sleep

→ Overlap with night terrors

→ Involve complex behaviour

- sitting up in bed, walking, dressing, eating, etc.
- Mostly in children

Treatment: Reassurance

- Diazepam

- safety restraint

- (c) Sleep Enuresis
- Involuntary micturition during sleep following attainment of control while awake
 - usually idiopathic
 - Cause by urogenital disease, or other medical problem
 - Treatment :-
 - Behavior training
 - Imipramine

(d) REM sleep behaviour

- A rare neurological disorder in which a person does not become paralyzed during REM sleep, & thus act out his dream

- Lack of REM atonia allow patient to ~~act~~ ^{enact} his dream

- Motor activity may be harmful
- Neurological cause in 1/3rd

Treatment :- sleep study, clonazepam

(1) Dyssomnia

- Primary disorder of initiating or maintenance sleep or excessive sleepiness.
- It consist a part called hypersomnia
- The duration of major sleep episode varies from 8 to 12 hr.
- Excessive sleepiness during waking hours
- Unintentional sleep episode typically occur at low activity situation e.g. lectures, reading, driving long etc.
- Prolonged nocturnal sleep & difficulty in waking.

* Diagnosis

- A: Polysonnography :-
- Eye movement
 - Sleep recording
 - EEG
 - ECG
 - Ear oximetry
 - Thoracic & abcd wall monitor

② Multiple sleep latency test (MSLT) - Day time sleepiness

③ Actigraphy - Monitor rest & activity cycle

④ Preliminary complaint of excessive sleep

⑤ Cataplexy - Sudden bilateral loss of muscle tone,

⑥ Recurrent intrusion of element of REM sleep into transition bet.

[5] ⑤

Circadian rhythm disorder

① The sleep-wake cycle is under the circadian control of oscillator and can be disrupted by ~~misalignment~~ misalignment b/w an individual's biological clock.

② Circadian rhythm sleep disorder usually present with either insomnia or hypersomnia

③ May due to occupational work also known as shift work sleep disorder

→ Treatment

Tab - Benzodiazepam

- melatonin = 0.5 to 5mg

Q Jet lag.

It occurs when a person travel across time zone, & the external environment time is mismatch with circadian clock.

Symp :-

- Malaise
- Insomnia
- hypersomnia
- Fatigue
- Poor performance

Treatment :-

- ~~Trazodolone~~ ~~5mg~~
- Melatonin - 0.5 - 5mg
- Regular sleep cycle
- Avoid day time sleeping

Anemia

(1) Anemia is a condition in which there is a decrease in number of red blood cell or hemoglobin in blood.

(2) When the body does not have enough red cell or haemoglobin, ~~this condition~~ ~~is~~ it unable to get enough oxygen.

Types of anemia

- (1) Iron deficiency
- (2) Anemia of chronic diseases
- (2) folic acid deficiency
- (2) Vit B₁₂ deficiency
- (3) Sickle cell anemia
- (4) Aplastic anemia.

General patho

Various causes affect RBC & Hb level

↓
↓ level of RBC & Hb

↓
↓ less O₂ carrying capacity

↓
Hypoxia

↓ as Organ function

Organ failure

↓
cell death

Symptoms
of anemia

① Iron deficiency anemia.

→ It is a condition in which body is lack of enough iron in blood

→ Iron help in formation of RBC

BFU-E

↓

CFU-E

↓

Proerythroblast ← Iron

↓

RBC

→ The RBC form in deficiency of iron have abnormal carrying capacity of O_2 .

* Clinical manifestation.

• Sign & Symptom

① brittle nail

② Fatigue

- Date _____
Page _____
- (2) chest pain
 - (1) pale yellow skin
 - (5) Dizziness
 - (1) Headache
 - (5) Fast HR
 - (1) cold hand & feet
 - (4) S.O.B
 - (5) Swelling of tongue

(2) Cause

- Blood loss
- Poor diet
- Insufficient consumption of iron rich food
- Indigestion
- Genetic
- Haemorrhage

(3) Diagnostic test

- CBC
- Peripheral blood smear
- Iron studies
- MCV reduced

➤ Iron supplement

- ① Ferrous sulphate :- ~~11mg/day~~
• 325 - 600mg BD
- ② Ferrous gluconate - 320mg
- ③ Ferrous fumarate 325mg
- ④ Iron supplement with vitamin C
- ⑤ IV Iron infusion 5mg
- ⑥ Food source rich in iron are
 - kidney bean
 - Turkey leg
 - Egg
 - Peanut butter
 - Brown rice
 - Raisin bran
- ⑦ Blood transfusion

③ Anemia chronic diseases

→ Mild to moderate severe anemia associated with chronic infection & inflammatory disorders.

- Pathophysiology

→ Inadequate erythrocyte production or increased red cell destruction.

→ Inhibition of intestinal absorption of iron

- Clinical manifestations

④ Sign & symptoms:

- Anemia

- Fatigue

- Shortness of breath

- Headache

- Confusion

- Tiredness

⑤ Causes :- Infection

- HIV

- TB

- Malaria

- Cancer

- Rheumatoid arthritis

- SLE

- IBD

(3) Diagnostic

- ① MCV \rightarrow decreased
- ② Serum iron \rightarrow low
- ③ Serum ferritin \rightarrow low
- ④ MCV/RBC = High.
- ⑤ Marrow invasion

+ Treatment

- ① Blood transfusion
- ② Iron supplement.
- ③ Erythropoietin - 10,000 unit 3 times week
IV
- ④ Darboparin - 675g/kg S.C. 3 week
- ③ Folic acid deficiency & Vit B₁₂ deficiency

It is a type of Megaloblastic anemia in which the size of RBC is abnormally large

+ Clinical manifestation of

- ① Sign Symptom
 - Fatigue
 - Weakness
 - SOB

- Mouth sore
- loss of appetite
- Diarrhoea

* ~~Diagn~~ Cause

- ① Age
- ② ~~poor~~ Poor diet
- ③ Alcohol
- ④ Indigestion
- ⑤ Malabsorption
- ⑥ Pregnancy

* Diagnostic

- CBC
- Serum folate
- Vit B₁₂ level
- MCV & MCH level is low.

* Treatment *

- ① Rich in Vit B₁₂ & folic acid supplement
- ② Folic acid - 400 mcg.
- ③ Vitamin B₁₂ = 2 - 4 mcg.
- ④ Cyanocobalamin - 200 mcg as IV or SC
or
0.45 unit/kg Tds

⑤ Vitamin B₁₂ :- 1mg IM every 2-3 month.

⑥ Cynocobadamine :- 50-150 mg BD

[5] Sickle cell anemia.

↳ Inherited from parents.

↳ A condition which there is not healthy red cell to carry oxygen.

↳ Here the shape of RBC become sickle from round.

↳ Patho.

When normal cell transfer oxygen the HbS stick with tissue.

↓

This create a tension in cell.

↓

RBC formed rigid, inflexible & sickle shape.

1 Clinical manifestations

① Signs & Symptoms

- Fatigue

- Tachycardia

- Bed wetting

- Infections

- Swelling
- Pain
- Chest pain
- Paralysis
- SOB
- UT haemorrhage
- PT prolonged

② Cause

- Genetic
- High level of Hbs
- metabolic decompensation
- Decrease of pH
- longer exposure of RBC to low Oxygen level.

③ Diagnosis

- CBC
- Hbs test
- High reticulocyte
- Xray of skull
- Blood clotting
- Blood cell solubility test

• Treatment

- ① Blood transfusion
- ② Oxygen supplement
- ③ Antibiotic
- ④ Gene therapy

(E) L-glutamine 5-15 g/kg x 2 week

(C) Voxeator - 1500mg x 4 week

* ~~Polystyrene~~ Hemolytic anemia.

1) It increases RBC destruction outside bone marrow

2) Two type

(i) ~~Hereditary~~ Hereditary :- Enzyme defect (G6PD)

(ii) Acquired :-
 - hypersplenism
 - Auto immune disease
 - mechanical trauma
 - Infection
 - Toxin etc

* Clinical manifestation

(F) Signs & Symptoms

- General pallor
- Jaundice
- splenomegaly
- leg ulcer.

(2) Cause :- Toxic chemical
 - Drug

- Infection
- Hereditary disorders
- Immune system
- Haemorrhage

(3) Diagnosis:- CBC
- Peripheral smear

MCV & MCH Ix

- Serum haptoglobin
- Serum bilirubin

* Treatment

(1) Transfusion

(2) Drug bk:- Penicillin
- Ampicillin
- Quinine etc disorders

(3) Vit. folic acid:- 5mg / PO. - 2d

(4) Corticosteroids:- 1-1.5 mg / kg

(5) IVIG

iv) Aplastic Anemia.

v) Anemia due to failure of bone marrow to produce red & white blood cell as well as platelet.

vi) Patho:-

defective stem cell.

+
Number of progenitor cell

↓
Immune mediated suppression of stem cell function.

↓
Disturbance in bone marrow micro environment.

↓
Hematopoiesis interrupted.

↓
Aplastic.

* Clinical manifestations

• Signs & Symptom

- SOB

- dizziness

- Headache

- Pale skin

- Prickling PT

- Chest pain

- fever

★ Cause

- Toxicity of benzene
- ~~Dose~~ Chloramphenicol
- Elements in lead
- pregnancy
- HIV,
- Hepatitis etc.

(2) Diagnosis.

Lab finding :- ~~Bone~~

- CBC
- Severe pancytopenia
- ~~Yes~~ reticulocyte
- Platelet ~~Yes~~
- Bone marrow biopsy

★ Treatment

(1) Transfusion

(2) Treatment of infection

(3) Immunosuppressors

- cyclosporin - 1.5 - 2 mg/kg @ 12hr
- cyclophosphamide - 45 mg/kg ~~@ 12hr~~

(E) Steroid :- Methylprednisolone :- 5mg/kg
1-8 days
+ tapering 1mg on 9-14 day
further
+ tapering 1mg on 15-29 days

(C) Combination of folic acid & vitamin
Supplement

Classification

Anemia



MCV

MCV < 80 fl/cell

MCV

80-100 fl/cell

MCV > 100 fl/cell

Microcytic

Normocytic

Macrocytic



- Iron deficiency
- Thalassemia
- Anemia of chronic diseases

↓
Reticulocyte count

- Megaloblastic
 ↓
 B₁₂
 - folic acid
- Alcoholic liver disorder

High

Low

- Sickle Cell Anemia
- G6PD deficiency
- Hemolytic Anemia
- Nocturnal Hemoglobinuria

- Marrow failure
- Aplastic anemia
- Leukemia
- Renal failure
- ~~Anemia of~~

Drug induced liver

Drug induced agranulocytosis & Neutropenia

M.C.Q.

(Pcm) Centro lobular necrosis

(Tetracycline, Mx T) Steatohepatitis

(aminodantone) Phospholipidosis

(Isoniazid) Generalized hepatocellular necrosis

(Estrogen therapy) Cholestatic Jaundice

(niacin, flutamide) Mixed hepatocellular necrosis & cholestatic disease

(Hormonal-related agent) Neoplastic jaundice

Types

- ① Idiosyncrasy - sulphonyl urea glyburide
- ② Allergic - flum, mkt, salic
- ③ Toxic hepatitis :- PCM
- ④ chronic active toxic hepatitis - Isomayoid
- ⑤ Toxic cirrhosis - Mkt, vit A
- ⑥ liver vascular dis - O. C. Aza

Epilepsy.

Ques

Date _____
Page _____

Q1) What is Epilepsy, types of epilepsy, pathophysiology, etiology, types of seizure

Ans) Epilepsy defined as any group of various disorder marked by abnormal electric discharge in ~~continuous~~ ~~the~~ brain & have sudden brief period of altered consciousness, involuntary movement or convulsion.

Ans) Several type of epilepsy classified based on pattern & frequency of seizure.

(I) Focal or ~~Partial~~ ^{Partial} seizure

(A) Simple (without impairment of consciousness)

→ Affect small part of brain

→ Alter motor symptoms like taste

(B) Complex (with impairment of consciousness)

→ Impaired function of response

→ Person become confuse & dazed

(C) Secondary generalized seizure

- Beginning of spreading of seizure

- Also called pre ~~gen~~ generalized seizure

II

Generalized seizure

→ Bilaterally affected

(a) Absence

- Rapid blinking
- Blanking of ~~the~~ mind
- Starrings

(b) Myoclonic

- Jerking of body, arms or head
- Imbalance of walking & standing

(c) Tonic :-

- Increase in tone ; pale skin
- loss of awareness

(d) clonic & Grand mal

- ~~Breathing stops~~ breathing ; urine, feces
- Cortical discharges reduces

(e) Tonic - clonic

- Stiffness
- falling
- ~~Bad~~ Breathing problem
- Convulsion
- Confusion

(d) ~~Atonic~~ Atonic

- Sudden loss of muscle tone
- Drooping limb effect

III) Unclassified. \pm tonic-clonic, behavioural arrest

IV) Status epilepticus :- Continuous seizure for 30 min
can't return to consciousness

* Pathophysiology

Paroxysmal discharge in cortical
neurons ~~which~~ ^{where} seizure originate
from grey matter of cortical or
subcortical area

↓

Abnormal firing of neurons

↓

Breakdown of normal membrane conductance
& inhibitory synaptic currents

↓

locally
affected

↓

Focal
seizure

widely
affected

↓

Generalized
seizure

Etiology

① In Newborns

- lack of O_2
- Brain injury
- Abnormal brain development
- Metabolic disorders

② In childrens

- Fever
- Brain infection
- Brain tumor
- Trauma

③ In adult

- Brain tumor
- Head trauma
- Stroke
- Degenerative disorders

Q2 Write a note on clinical manifestation & Treatment of Epilepsy

Clinical manifestation

(i) Sign symptom

- Confusion
- Aura deflection
- Sudden fall
- Staring

(1) Frontal lobe :- twitching or stiffness

(2) ~~Parietal~~ Parietal lobe :- Burning, numbness

(3) Occipital lobe :- Visual disturbance / hallucinations

(4) Temporal lobe :- unusual taste/smell
Feeling fear

Etiology

- Same as before

a Diagnostic report

(1) CBC

(2) EEG

(3) CT scan

(4) MRI

~~Phen~~ Treatments

[BB IN, AD, P, CS]

① Barbiturates :- phenobarbital
Amobarbital 50-100

- 60 mg OD

② Hydramtain (Phenytoin)

100 - mg BID

③ Succinimide :- Ethosuximide

500 mg / day

③ Benzodiazepam

clonazepam :- 1.5 mg

Diazepam :- 4-40 mg

lorazepam :- 10 mg

④ Deoxybarbiturate (primidone)

250 - 500 mg BID

⑤ Iminostilbenes [carbamazepine]

200 - 400 mg / day

ox carbamazepine 300-600

② Aliphatic carboxylic acid

Valproic acid 200-800mg

③ phenyl triazine :- lamotrigine
25mg/day

25mg/day

② Cyber gubaba analogue

- Gabapentin 300mg OD

pregiballine = said too nig

1981 S. 1. ~~1981~~ P. C.

BB

20th - 1A

-HD- 730 500

P-25.

CS. - 500

300

Surgical

⑤ Lab. reserction

② Lesionectomy

③ *Garpus clathrus-komy*

④ MST

(5) Vagus nerve: stimulant.

Q1) Define, types & patho of Traumatic

x prodical of Epilepsy

0-5 minutes → Stabilize patient by monitoring (ABCD)

↓
Monitor vitals

↓
ECG monitoring

↓
Glucose level maintained if ≤ 60 mg/dl.

↓
100 mg + thiamine + some D5W

↓
Seizure continued

yes

No → Symptomatic care

5-20 min
initial
therapy

↓
Benzodiazepine
therapy

↓
IV lorazepam 0.1 mg/kg

or
IV diazepam 0.15-0.2 mg/kg

or
IV phenobarbital :- 15 mg/kg

70-40 min.

yes

৯৩

CT

Second line

therapy initiated

- IV valproic acid (40mg/kg)

~~- Iv levetiracetam (60mg/kg)~~

Seizure

yes

No -

Symptomatic management

40-60 min

CT 2nd line

with increasing
close

05

Anesthesia is given as well.

571 Anne 028-027

Jaundice

Date _____
Page _____

Define, Types, Patho of Jaundice

1) Jaundice is yellowish discoloration of skin, sclera & mucous membrane due to hyperbilirubinemia & deposition of bile pigment.

2) It is not a disease, but occurs with any other diseases.

Types

- It is of 3 types

① Prehepatic :- Before reaching liver
S. Bil :- 5-17 $\mu\text{mol/L}$
- Unconjugated

② Intrahepatic :- In liver
S. Bil :- 50-350 $\mu\text{mol/L}$
- Both.

③ Post hepatic :- Obstruction in biliary tract
S. Bil 10-750 $\mu\text{mol/L}$
- Conjugated.

Pathophysiology

Causes



Excess haemolysis of
RBC



Prehepatic, Jaundice



Unconjugated hyperbilirubinemia



→ Jaundice ←

hyperbilirubin

Hepatic Jaundice



Damage hepatocyte



cause

conjugated hyperbilirubin



Post/obstructive



cause

Q2 Clinical manifestation & H of Jaundice

Q3 Clinical Manifestation

Etiology

① prehepatic : Anemia, Malaria, RBC abnormal destruction, excessive haemolysis

- ① Intra hepatic :-
- Engine defect
 - Storage defect
 - Ics hepatic bil uptake
 - Alcohol
 - Auto Immune
 - Drugs
 - Turner
 - Pregnancy
 - other injury

- ② Post :- Infection
- Stone
 - Pancreas, gallbladder carcinoma
 - Pancreatitis
 - Drugs

- Drugs like :-
- Acetaminophen
 - Disulfiram
 - Ketorolac
 - carbimazole
 - OC
 - AKIT₄

* Signs Symptom

- Skin :- Pale yellow colour
- Itching
- Eyes :- whiteness of eye changes

light stool
dark urine

Stomach :-
- Abdominal pain
- loss of appetite
- Nausea vomiting

Brain :-
- Headache
- fever
- confusion
- weakness

Diagnostic Test

Test	Pre	Hepatic	Post
① T. Bil	② or Tes	Tes	Tes
② Conj Bil	N	Tes	Tes
③ Unconj	N	Tes	②
④ Urubilinogen	N or Tes	N	Tes
⑤ Urine Colour	②	Dark	Dark
⑥ ALP & AST	②	Tes	Tes

- ~~Imaging~~ ~~Imaging~~

PT time Test

- Urine routine micro

- Faecal test
stercobilinogen

- Chest X-ray

- USG Abd.

+ Treatment

Anionic exchange resins:-

cholestyramine :- 4-16 g

colestipol :- 2-16 g

Antihistamic :- Cetirizine 5-10 mg

~~Rifampicin~~ Rifampicin :- 600 mg

Antibiotics

N.P.

+ Phototherapy

+ Ursodeoxycholic acid :- 10-15 mg/kg

N acetylcysteine :- 100 mg

+ Prednisone :- ~~20~~ 60 mg

+ Phenobarbital :- 2-5 mg/kg

* The Peptic Ulceration Disorders *

* PUD *

Q What are the patho / clinical manifestation / treatment of PUD

Ans Define :- Peptic ulcer are sore that develop inside the lining of stomach.
The Peptic ulcers in stomach cause pain

Ans Types.

① Gastric Ulcer :- 1-2 hr of pain after meal
- Risk for malignancy
- Usually on lesser curvature

② Duodenal Ulcer :- 2-4 hr after meal Pain
- Pain wake up patient
- less risk of malignancy

Ans Cushing ulcer or Curling ulcer are the type of acute ulcer

Patho.

→ By 2 ways the pathophysiology works

① Drugs, stress, steroid etc.



Inhibition of phospholipase A_2



Decrease level of arachidonic acid formation



Decrease prostaglandin



Increase gastric secretion



Damage mucosal lining



Ulcer formation

② *H. pylori*

with Bacteria produce urease & stick protein



Release of cytokines, histamine & prostaglandins



Decrease phospholipase



Urea convert to CO_2 & ammonia which destruct mucosal lining



↓
Ulcer

* Clinical manifestation

① Etiology

① *Helicobacter pylori* bacteria

- Smoking
- Alcohol
- Excess stress intake
- Fasting for long period

② NSAIDs like - Ibuprofen

Naproxen

Ketoprofen

Aspirin

Acetaminophen

③ Other medication like - SSRIs

- Serotonins

② Sign Symptoms

- Epigastric tenderness
- Abdominal pain
- Loss of appetite
- Sharp burning pain
- Bleeding
- Dyspepsia
- Nausea

+ | Diagnostic test

- ① EGD :- Esophagogastroduodenoscopy
- ② Endoscopy
- ③ USG Abdomen
- ④ H. pylori test.
- ⑤ Biopsy

• Treatment:

- ① Antacid :- Sodium bicarbonate
- ② Antibiotics :- Amoxicillin
- clarithromycin
- Metronidazole
- ③ Promoter of healing :- PPIs - ~~P3~~
- ④ H₂ or secretion blocker :- Famotidine 40
Ranitidine 150
~~Cimetidine 800~~
- ⑤ Mucolyte protective :- Sucralfate - 1g
Sucralfate - 1g
- ⑥ P_A agonist :- Misoprostol - 0.2 to 0.5 mg
- ⑦ B¹² Sm²⁺ Subsalicylate



Root

Protocol

Patient come with complaint of ulcer like symptom

NO alarm Symptom

Alarm Symptom

On NSAIDS

Endoscopy

Yes

NO

ulcer

yes

no

Discontinue or decrease dose

History of H pylori

Test for H pylori

consider other Etiologies

Symptom resolve

NO

yes

Positive

Negative

yes NO further

NO

serology test

Evolution therapy

On NSAIDS

Start

H2 blocker or PPI

Sign / symp continue

Yes

NO

Symptom resolve

after 1-2 week?

Discontinue or change to COX2

Start PPIs

yes

no

yes

NO

Treat with PPI

Consider other diagnosis

H pylori eradication therapy

① PPI based triple therapy

PPI	+ clarithromycin	+ Amoxicillin
40mg	500mg	1g
BD	BD	BD
		or Metro 500mg BD

② Bismuth - base quadruple therapy

PPI or H ₂ RA	+ Bismuth Sub salicylate	+ Metronidazole	+ Tetracycline
BD	525mg Qds	250-500mg QDs	500mg Qds

③ Sequential therapy

PPI or H ₂ RA	+ Amoxicillin	+ Metronidazole	+ clarithro
BD	1g BD	250-500 BD	250-500 BD
1-10 days	1-5 days	6-10 day	6-10 day

① Second line (Salvage) therapy

② PPI or H_2RA + Bismuth subcitrate + Metronidazole + Tetracycline
BD 525 mg 250-500 500 mg
Qds Qds Qds

③ PPI or H_2RA + Amoxicillin + Levofloxacin
BD 1g BID 250mg BID

④ PPI

- ① Pantoprazole - 20-40 mg
- ② Rabeprazole - 40mg
- ③ Omeprazole - 20-40 mg
- ④ Esomeprazole - 30-60 mg

⑤ H_2RA

- ① Famotidine - 40mg
 - ② Ranitidine - 300mg
 - ③ Cimetidine - 800mg
- } At bed time

⑥ Surgery

- ① Gastroenterostomy
- ② Vagotomy
- ③ pyloroplasty
- ④ Antrectomy

* Stroke

① Definition, types and patho

→ Stroke is defined as neurological deficit of cerebrovascular cause persists beyond 24 hours or leading to death.

→ It is a rapid loss of brain function due to disturbance in the blood supply to brain.

* Types

① Ischemic

② Hemorrhagic

③ Transient ischemic attacks

1) Ischemic stroke : It is the most common type of stroke.

- Here, when a blockage or narrowing of arteries that provide blood to brain, result in ischemia

- It has further two type

(a) Thrombotic stroke :- Cause by blood clot in vessel of ~~brain~~ brain

(b) Embolic stroke :- clot or plaque develop in other part of body & travel in blood

vessel in brain.

② Haemorrhagic :- Bursting or leaking of arteries in brain.
- It is of 3 type.

(a) Subarachnoid hemorrhage :- When blood enters in subarachnoid space owing to trauma.

(b) Intracerebral hemorrhage :- When blood vessel rupture within the brain parenchyma itself, & result in formation of hematoma.

(c) Subdural hematomas :- Collection of blood below the dura which is covering of brain.

③ Transient ischemic stroke (TIA) :- It means focal deficit, vision loss for few seconds.

- It is also called mini stroke, which show warning of stroke in future.
- This occur due to temporary disruption of blood flow.

* ~~Patho~~

Q2 Clinical manifestation & T/t.

① Signs & symptoms

- | | |
|-----------------------------|----------|
| - Confusion | Face |
| - Vertigo | A arm |
| - loss of vision | S speech |
| - falling | T time |
| - Headache | |
| - Loss Giddiness | |
| - Hemiparesis | |
| - lack of co-ordination | |

② Etiology

① Ischemic :- Thrombus
- Embolic

- Artherosclerosis

② Haemorrhagic :- Trauma

- High B.P

- Rupture vessel

③ Diagnosis

① CBC :- protein & C deficiency
for hypercoagulable

② CT scan:-

hyperintensity in hemorrhage area
and hypointer in infarction area.

③ MRI

④ DWI (diffusion-weighted image)

⑤ Carotid Doppler

⑥ ECA

⑦ EEC

⑧ Transcranial Doppler

+ Treatment:

(a) Non-pharmacological:

(b) Pharmacological:

For ischemic stroke

(a) Alteplase :- 0.9 mg/kg IV

(b) Aspirin :- 160 - 325 mg/day

For Hemorrhagic Stroke

Nimodipine :- 60 mg @ 4hr

For cardioembolic

Vit K antagonist :- Dabigatran :- 150 mg BD.

For atherosclerosis.

Statins

atorvastatin - 80mg

② Surgical management

- (i) Carotid endarterectomy
- (ii) Craniotomy
- (iii) Extracranial - intracranial arterial bypass

Headache

02. Clinical manifestation and treatment

① Sign and Symptom.

- Not performing normal activities
- Severe headache
- Vertigo
- Recurring episode of throbbing head pain
- Sensitivity to light
- Feel of correlating pattern of headache

② Etiology

- ① Alcohol
- ② Change in sleep wake cycle
- ③ Depression
- ④ Stress
- ⑤ Head injury
- ⑥ Medication
- ⑦ Diseases condition
- ⑧ Weather change
- ⑨ Neuronal damage
- ⑩ Trauma

③ Diagnosis

- ① Medical history
- ② Medication history
- ③ Headache history by asking
 - When started?
 - How long it be
 - Single type or multiple type
 - How bad pain feel
 - Family history of headache
 - Symptoms of headache

④ Imaging techniques :- CT scan or MRI.

⑤ Other physical examination

* Treatment

* Non-pharmacological

* Pharmacological

for Tension-type headache

(i) Analgesic

- Acetaminophen :- 1g

- Aspirin :- 500-1000 mg @ 4-6 hr

(ii) NSAIDs

Ibuprofen 200-800 mg @ 6 hr

- Naproxen :- 550-825 mg qd

* For Migraine

(i) Pcm :- 500mg BD

(ii) NSAID

Ibuprofen 600-800mg

(iii) Ketorolac :- 30-60mg

(iv) Ergotamine tablet :- 2mg @ 3amin

(v) Triptans

- Sumatriptan :- 25-100mg @ 2hr

- Rizatriptan :- 5-10mg @ 2hr

(vi) Dexamethasone :- 4-8mg IV

~~(vii) Prophylactic :- Propranolol 40-60mg Tds~~

* For cluster headache

1) Abortive

- Oxygen

- Ergotamine - 2mg

- Triptans

- Sumatriptan 6mg

2) Prophylactic

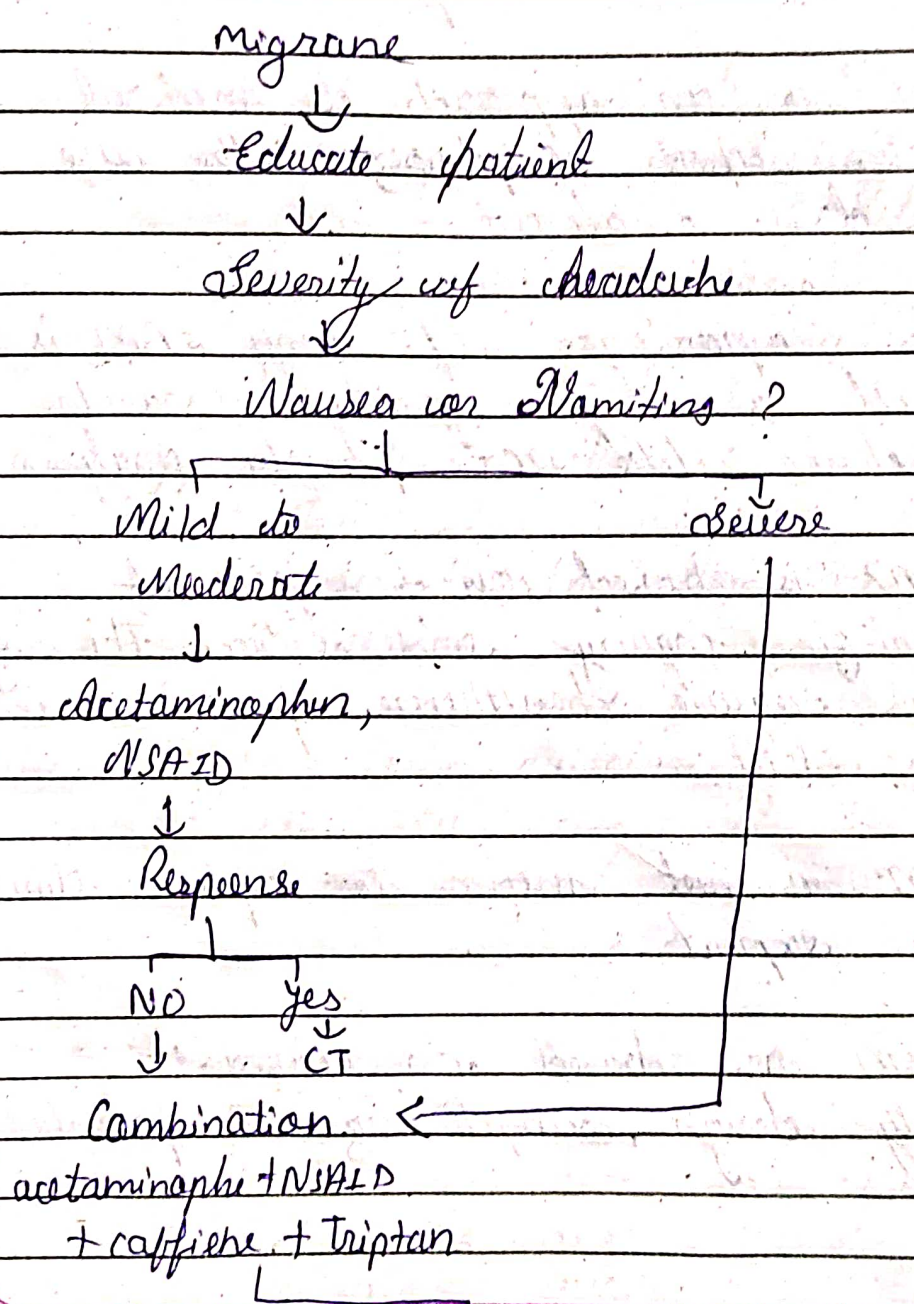
- Verapamil :-

- Lithium :- 600mg - 1.2g / day

- Corticosteroid :- 40-60mg / day

Prophylactic of migraine

- Propranolol: 40-60 mg
- Amitriptyline: 25-100mg
- Gabapentin: 900-3600 mg
- Valproate: 400-600 mg BD



Evidence based medicine

→ EBM is an approach to medical practice that use the result of patient care research and other available objective evidence for clinical decision making.

- EBM aims to provide the best possible care to patient.

- EBM is an approach to medical practice that emphasize the use of best treatment.

- The importance of using EBM is that it is high-quality research evidence like RCTs, meta analyses.

- EBM is based on principle of scientific inquiry and critical thinking and require healthcare provider in these skill.

- EBM is not meant to replace clinical ~~sign~~ expert.

- EBM are based on research study design, sample size & potential biases.

Date _____
Page _____

The process of EBM involves several steps:

(i) Formulate a clinical question :-

- It is a 1st step
- Here, a clinical question are scientific
- Questions must be specific, answerable, and relevant.

(ii) Search for the evidence :-

It involves conducting a systematic review of the literature, which involves searching for critically appraising.

(iii) Appraise the evidence :-

Here the evidence is critically appraised to determine its validity, relevance.

(iv) Integrate the evidence :- After appraisal the evidence must be integrated to ~~the~~ clinical decision-making process.

(v) Evaluate the outcome :- Final step in the EBM.

→ Involves monitoring the patient progress and adjusting the treatment plan.

Q. Write a note on Oral anticoagulant

Oral anticoagulant are medication that are used to prevent blood clot from forming or getting larger.

Oral ~~ant~~ anticoagulant are typically prescribe to individual who are at increased risk of developing blood clot due to various medical condition, such as atrial fibrillation, DVT, pulmonary embolism and Stroke.

→ They are classified into two main category

(a) Vitamin K antagonists (VKAs)

(b) Direct Oral Anticoagulants (DOACs)

(a) Vitamin K antagonists are

(i) Warfarin : It inhibit the vitamin K dependent synthesis of calcium dependent clotting factor (2, 7, 9, 10)
- It usually given in dose of 2 to 10 mg OD.

(ii) Acenocoumarol : It inhibit the vitamin K reductase enzyme.
- Dose : 4-12 mg OD.

anticoagulant

that
sheet
larger

typically
at
local
concentration,
T, pulmonary

main

MACs).

vitamin
calcium
(2, 7, 9, 10)

of

the

are

(b) Direct oral anticoagulants

(i) Dabigatran :- 150mg BD

(ii) Apixaban :- 5mg BD

(iii) Rivaroxaban :- 20mg alternate w/ Warfarine

→ Oral anticoagulant have contraindication like

- Active bleeding
- Risk of bleeding
- Severe liver diseases
- Pregnancy :- May cause fetus bleeding
- Severe kidney diseases
- Hypersensitivity
- High risk of drug-drug interaction with NSAIDs and some antibiotics.

→ VKAs should carefully monitored & required dose adjustment than DOACs

→ Patient on anticoagulant need to aware for any injury or bruising teeth

Q. Note on atypical antipsychotic drug.

- Ans. This are 2nd generation
 Atypical antipsychotic are a class of medication used to treat mental health disorders such as
- Schizophrenia
 - Bipolar disorders
 - Major depressive disorders.

→ It work by affecting the levels of certain neurotransmitter of brain, such as Serotonin (5-HT_{2A}), Dopamine & noradrenaline.

→ This are used widely than typical (first generation) antipsychotic because this are at low risk of causing movement disorders such as dyskinesia.

Ans. Example of atypical antipsychotic are

Risperidone :- 0.5 - 1mg BD

Quetiapine :- 12.5 to 25 mg at night.
 - 150 - to 300mg

Aripiprazole :- 10 - 15 mg OD

Clorazepine :- 25mg starting
 100 - 800 mg maintenance

→ The side side effect of atypical antipsychotic are

- Weight gain
- Metabolic changes
- High risk of diabetes
- Sedation
- Sexual dysfunction
- Cognitive impairment

→ Atypical antipsychotic are also use as add-on for patient ~~like~~ dementia, psychosis etc.

* These paper ans

Q. Diff b/w Chron's & UC & its management.

- Crohn's diseases

Ulcerative Colitis

Site:- Inflammation occurs anywhere in the digestive tract

Infection occurs typically in the Colon.

Pattern of inflammation - Occur in patches form

- Occur in continuous form.

Appearance - Colon wall may be thickened and appears rocky.

- Colon wall is thinner & show continuous inflammation

Ulcers deep - Ulcers are deep and extend to cell level

- Ulcers are only on mucus lining.

Symptoms - Cramps in abd

→ Bloody diarrhoea

Radiographic finding - String sign can - Barium X ray

→ Lead pipe colon can Barium X-ray

Surgery - In moderate to severe condition surgery required

- Patient does not require surgery

Cancer chances - Less common

- May be high

* Ib
* Algs

mic

Distal

- Oral Sulf
or meso
or
enema

↓ Remi

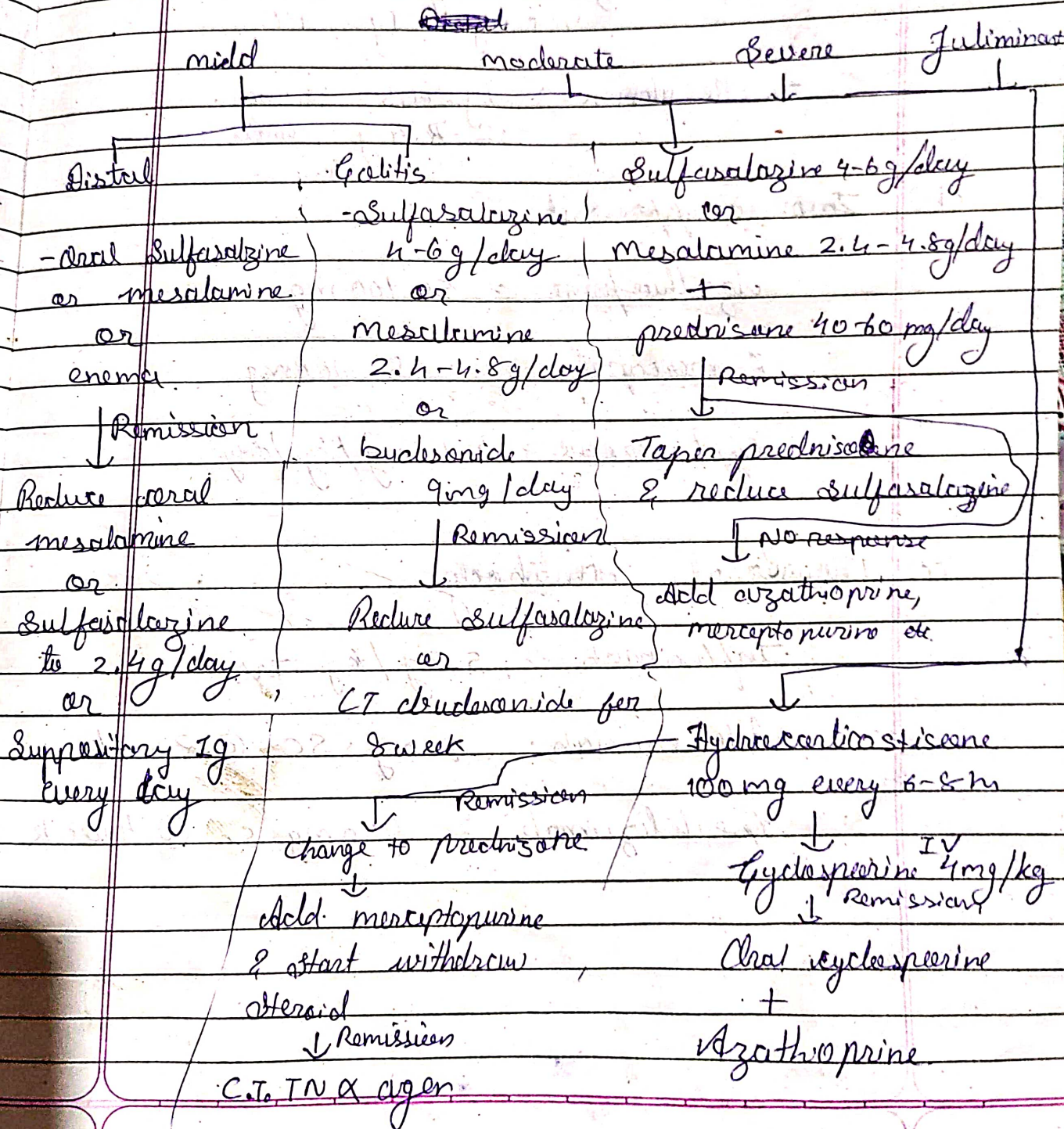
Reduce or mesolamin

or Sulfasal to 2,4 or C

Suppositories Every

Ibd Management

Algorithm for UC



(a) Amino salicylate

- Sulfasalazine :- 3g as enema.
500mg - 1g PO.

- Mesalamine :- 1g as enema
- 1.2-2.4 g oral

(b) Immunosuppressant

Azathioprine :- 50-100 mg

6 mercaptopurine :- 50-100mg

Cyclosporine :- 2-4 mg/kg/day

(c) Monoclonal antibody

Infliximab :- 5mg/kg IV

Adalimumab :- 40mg SC @ 2 week

Certolizumab :- 400mg SC @ 4 week

cd Steroid

Hydrocortisone :- 125-250 mg ^{perema}
:- 100-400 mg ^{IV fluid}

Prednisone :- 40-60 mg orally

Budesonide :- 3-9 mg orally.

Algorithm of Crohn's

