



NPTEL ONLINE CERTIFICATION COURSES

Basics of Mental Health & Clinical Psychiatry

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**Dr B.C. Roy Multi-speciality Medical Research Centre
IIT KHARAGPUR**

Lecture 36: Anti-psychotic drugs

CONCEPTS COVERED

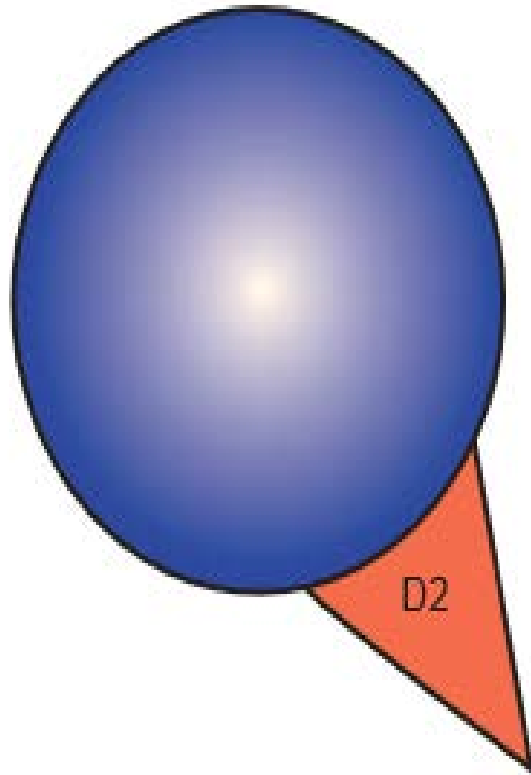
- Typical anti-psychotic drugs
- Atypical anti-psychotic drugs

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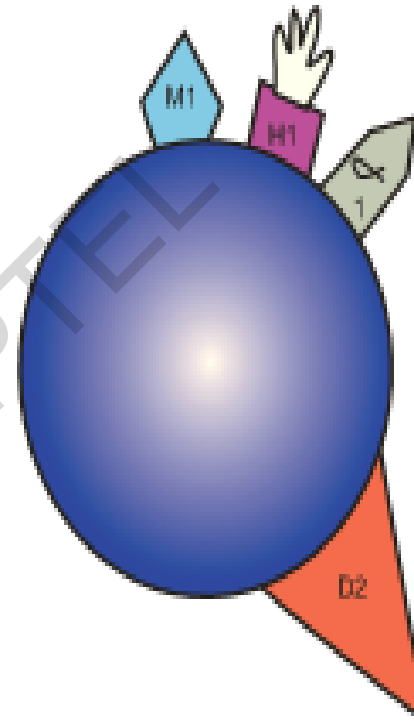
Typical Antipsychotics

What Makes an Antipsychotic Conventional?

D2 Antagonist Actions

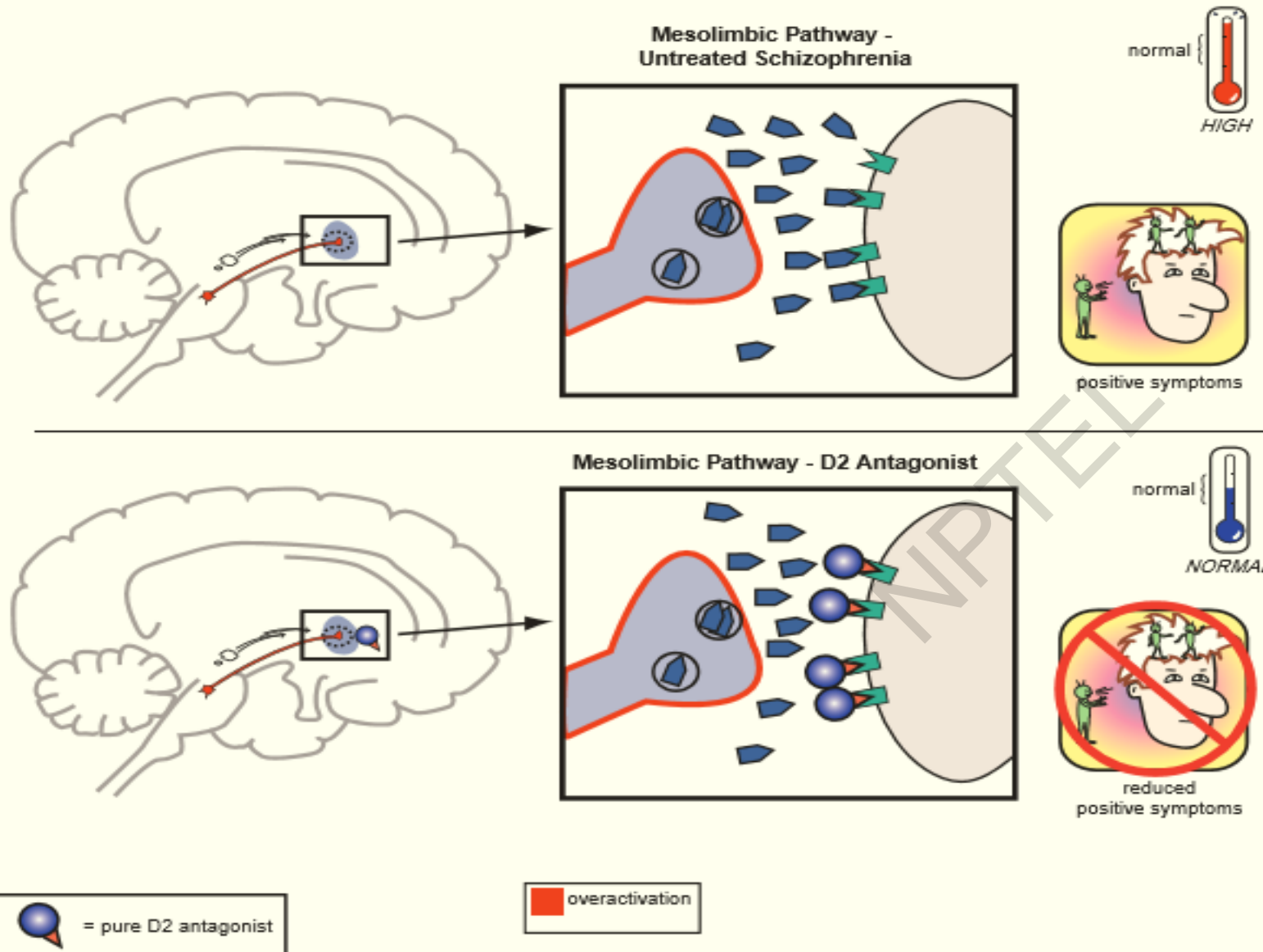


Various Binding Properties of Conventional Antipsychotics



Chlorpromazine

Mesolimbic dopamine pathway and D2 antagonists



Side effects

- ✓ Acute blockade of dopamine 2 receptors in the striatum can cause **drug-induced parkinsonism, dystonia, or akathisia.**
- ✓ Chronic blockade of dopamine 2 receptors in the striatum can cause **tardive dyskinesia.**
- ✓ By blocking dopamine 2 receptors excessively in the meso-cortical and mesolimbic dopamine pathways, especially at high doses, it can cause **worsening of negative and cognitive symptoms (neuroleptic induced deficit syndrome).**
- ✓ Blocking muscarinic cholinergic receptors can **cause dry mouth, blurred vision, urinary retention, constipation, and paralytic ileus.**
- ✓ Antihistaminic actions may cause **sedation, weight gain.**

- ✓ **Priapism** Cholinergic receptor antagonism may induce erectile dysfunction by reducing peripheral vasodilation
- ✓ **Galactorrhea , amenorrhea** Blockade of dopamine D2 receptors in the tuberoinfundibular pathway by antipsychotics may decrease the libido, impair arousal, and impair orgasm indirectly, by leading to elevated prolactin levels.
- ✓ **Neuroleptic malignant disease**

- ✓ Should be used during pregnancy if benefits outweigh risks.
- ✓ Should not be prescribed for breast feeding mother because secreted in milk.
- ✓ **How does muscarinic cholinergic receptor blockade reduce the EPS caused by dopamine D2 receptor blockade in the nigrostriatal pathway?**
 - Dopamine and acetylcholine have a reciprocal relationship in the nigrostriatal dopamine pathway. Dopamine neurons here make postsynaptic connections with the dendrite of a cholinergic neuron. Normally, dopamine suppresses acetylcholine activity.
 - If anticholinergic properties are present in the same drug with D2 blocking properties, they will tend to mitigate the effects of D2 blockade in the nigrostriatal dopamine pathway.

Haloperidol

Dopamine receptor antagonist - Conventional antipsychotic

Indications (FDA approved)

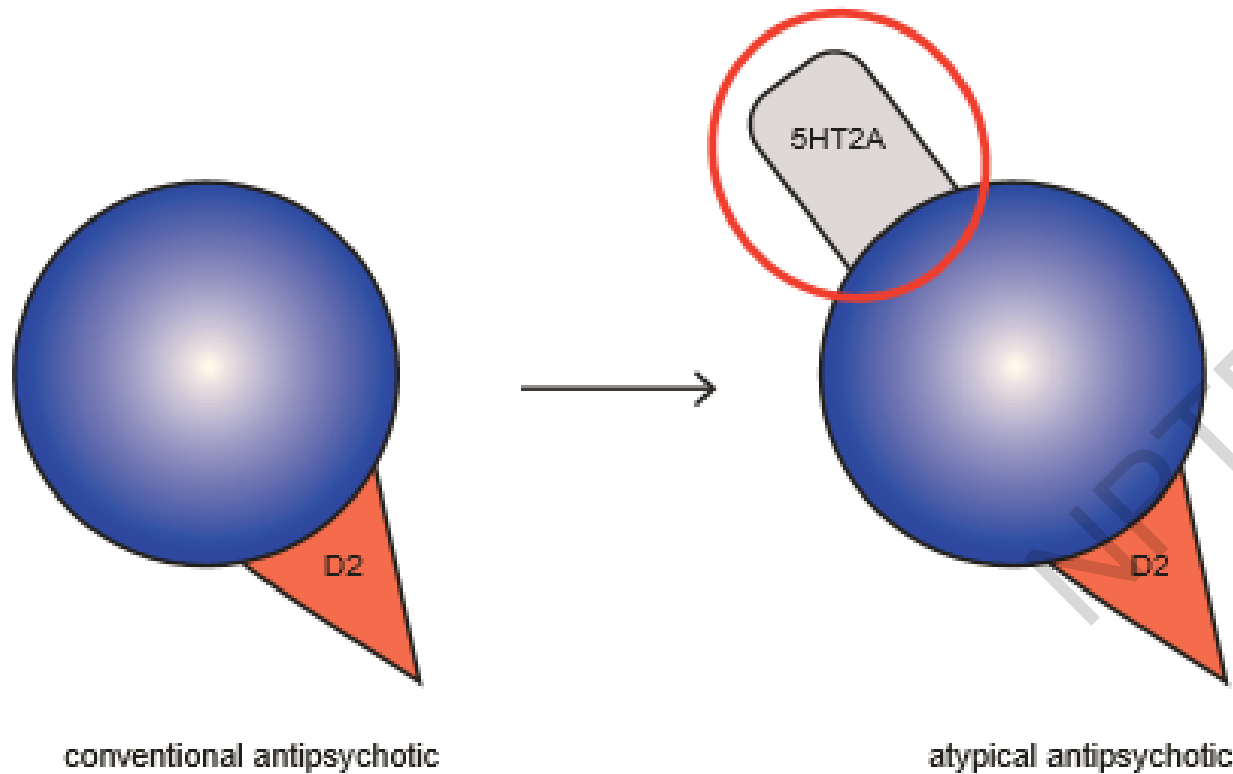
- ✓ **Manifestations of psychotic disorders (oral, immediate-release injection , depot intramuscular decanoate injection)**
- ✓ **Tics and vocal utterances in Tourette's syndrome (oral, immediate-release injection).**
- ✓ **Second-line treatment of severe behavior problems in children of combative, explosive hyperexcitability (oral)**

Other indications

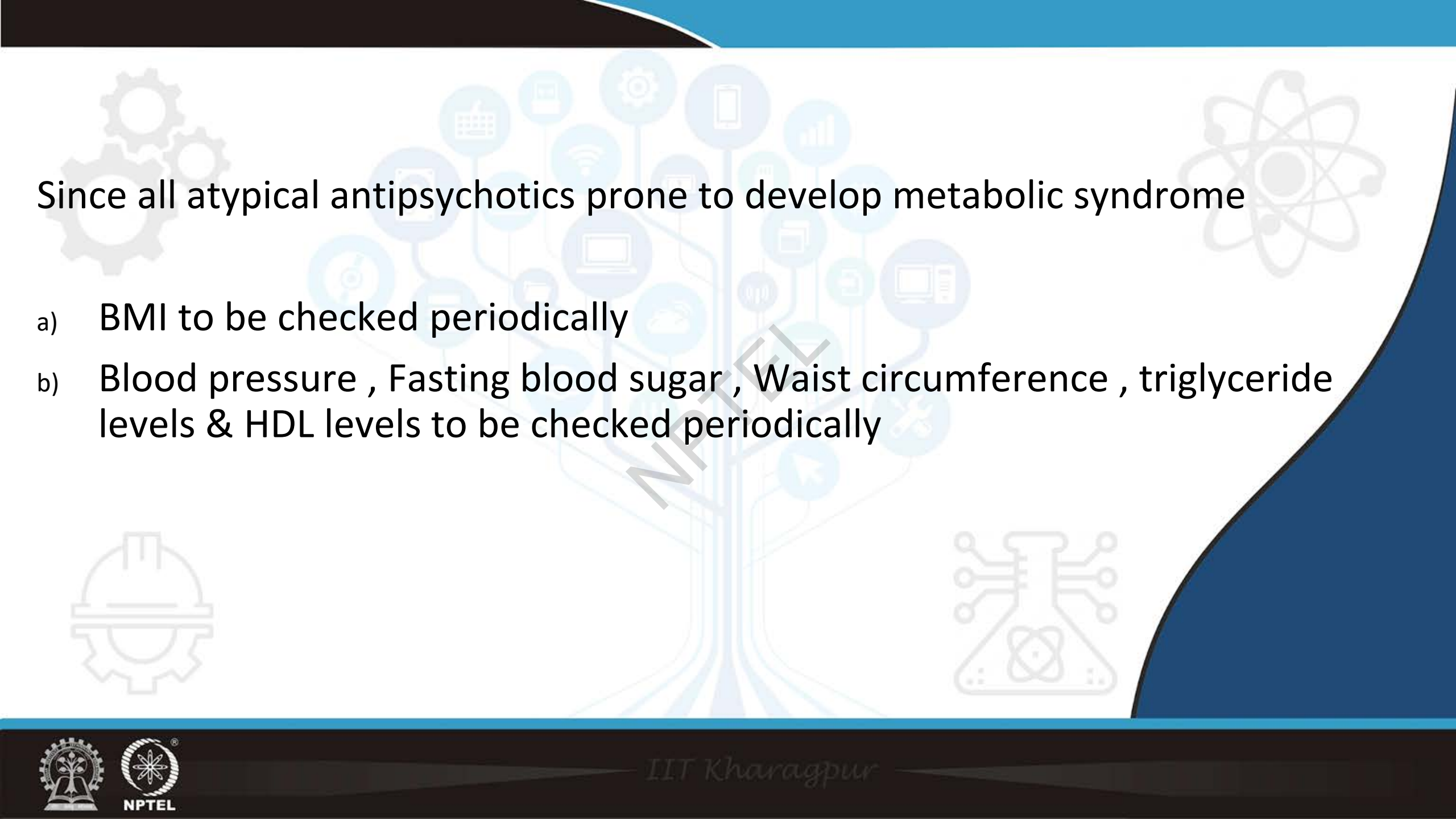
- ✓ Treatment of schizophrenic patients who require prolonged
- ✓ Bipolar disorder
- ✓ Behavioural disturbances in dementias
- ✓ Delirium (with lorazepam)

What Makes an Antipsychotic Atypical?

Adding 5HT_{2A} Antagonist / Inverse Agonist Actions



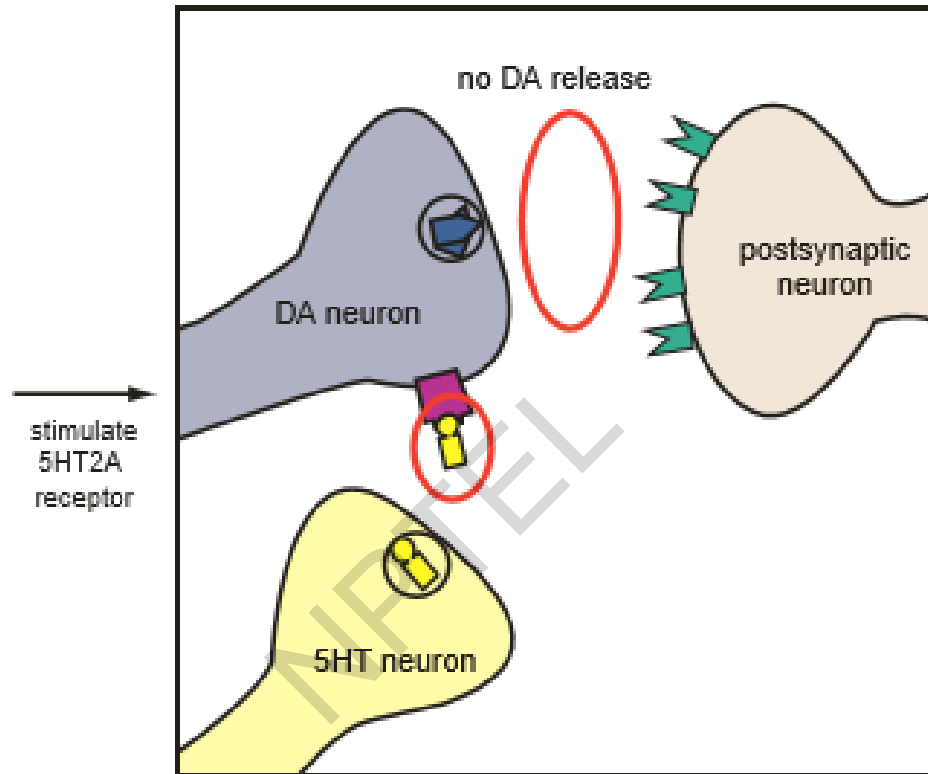
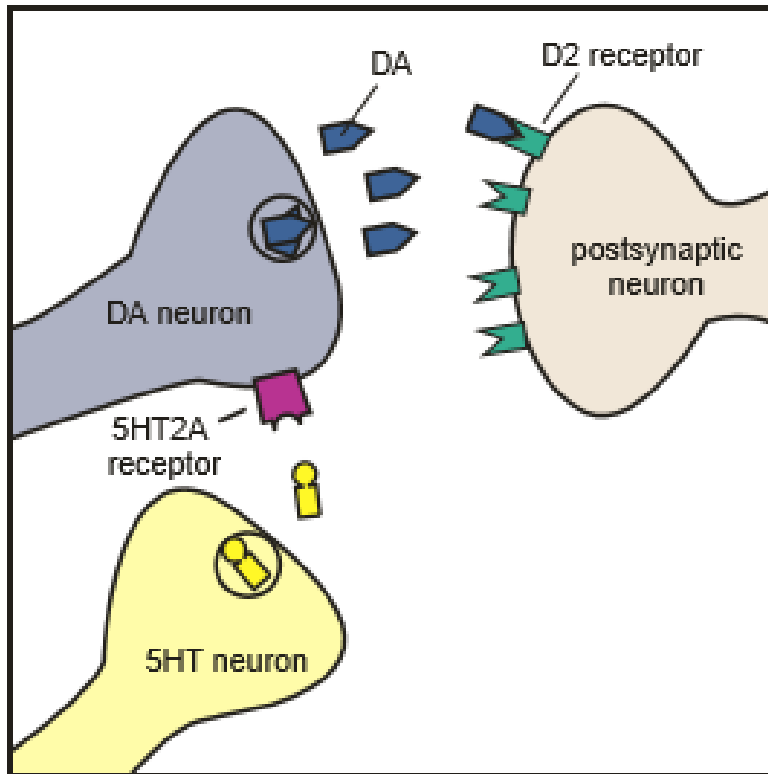
Atypical Antipsychotics



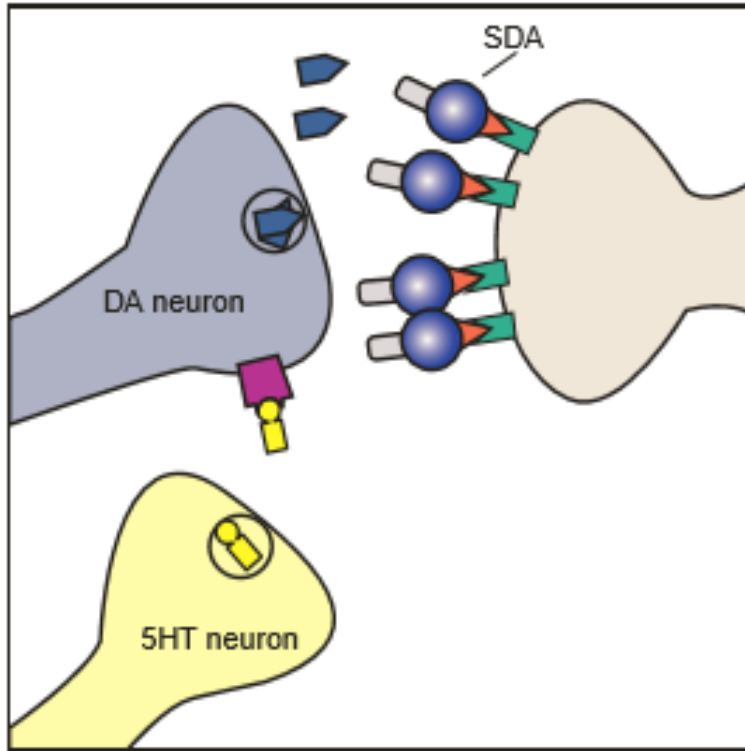
Since all atypical antipsychotics prone to develop metabolic syndrome

- a) BMI to be checked periodically
- b) Blood pressure , Fasting blood sugar , Waist circumference , triglyceride levels & HDL levels to be checked periodically

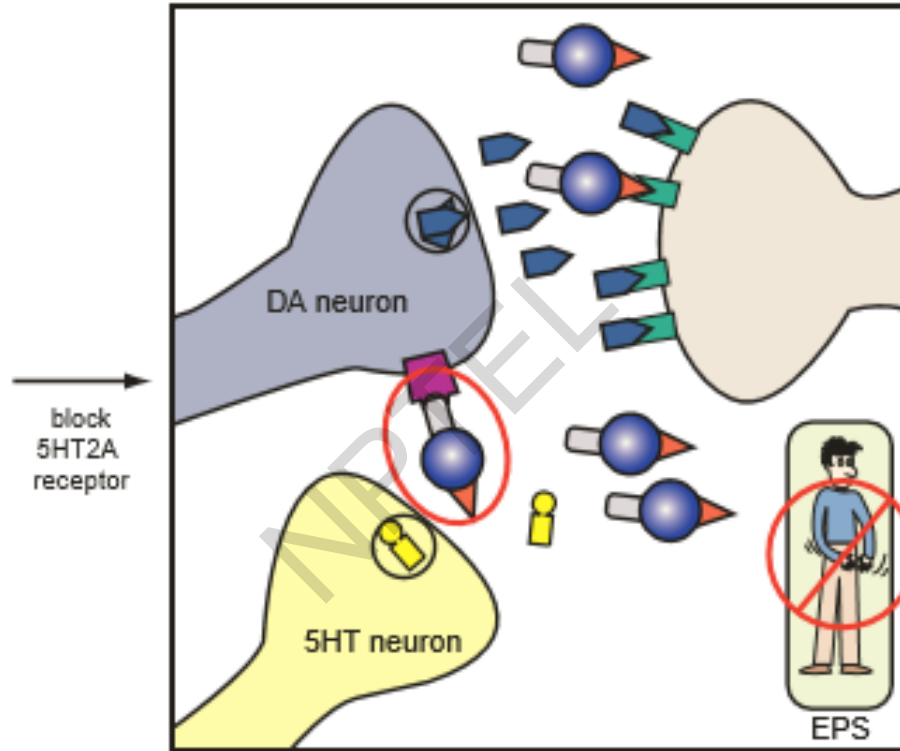
Nigrostriatal Pathway



**Nigrostriatal Pathway:
Blocking D2 Receptors**



**Nigrostriatal Pathway: Blocking 5HT2A Receptor
Disinhibits DA Release and
Reduces D2 Blockade**

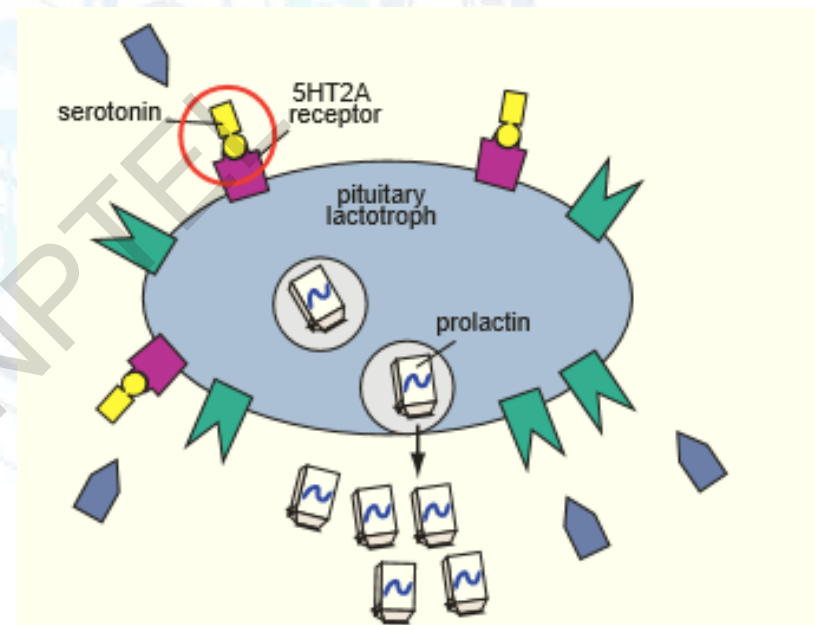
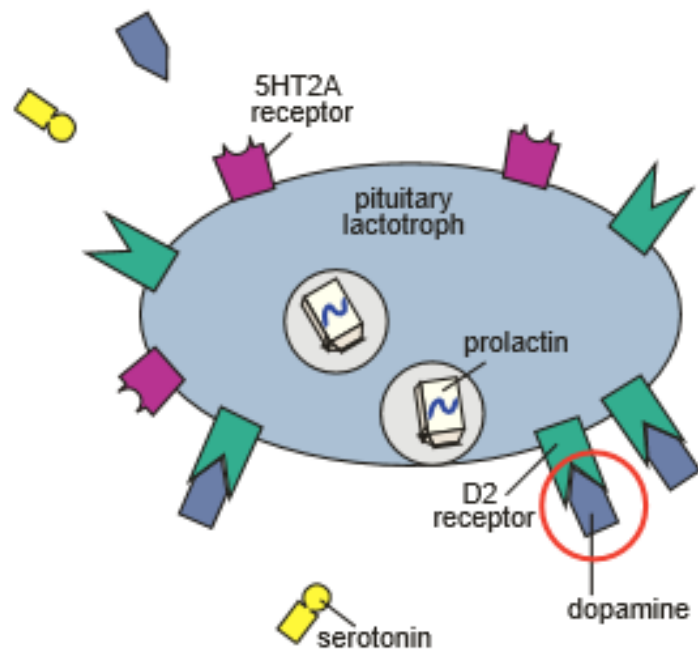


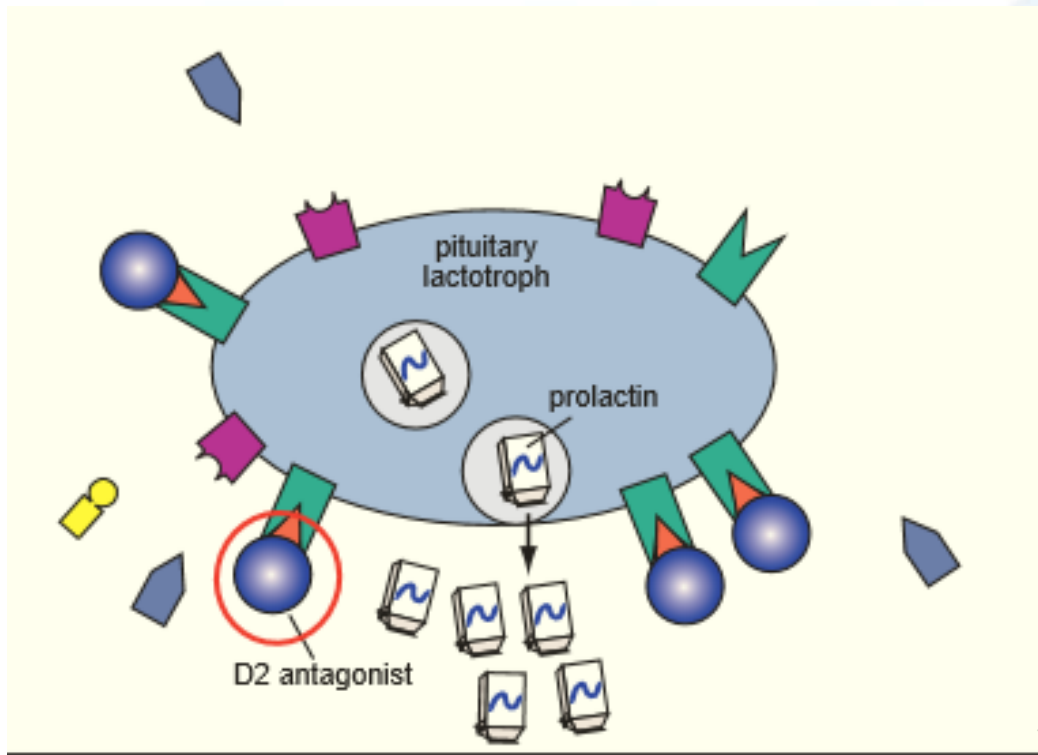
The consequence of this is that dopamine can then compete with the SDA for the D2 receptor and reverse the inhibition there. As D2 blockade is thereby reversed, SDAs cause little or no extrapyramidal symptoms (EPS) or tardive dyskinesia.

Serotonin 2A antagonists in the nigrostriatal pathway

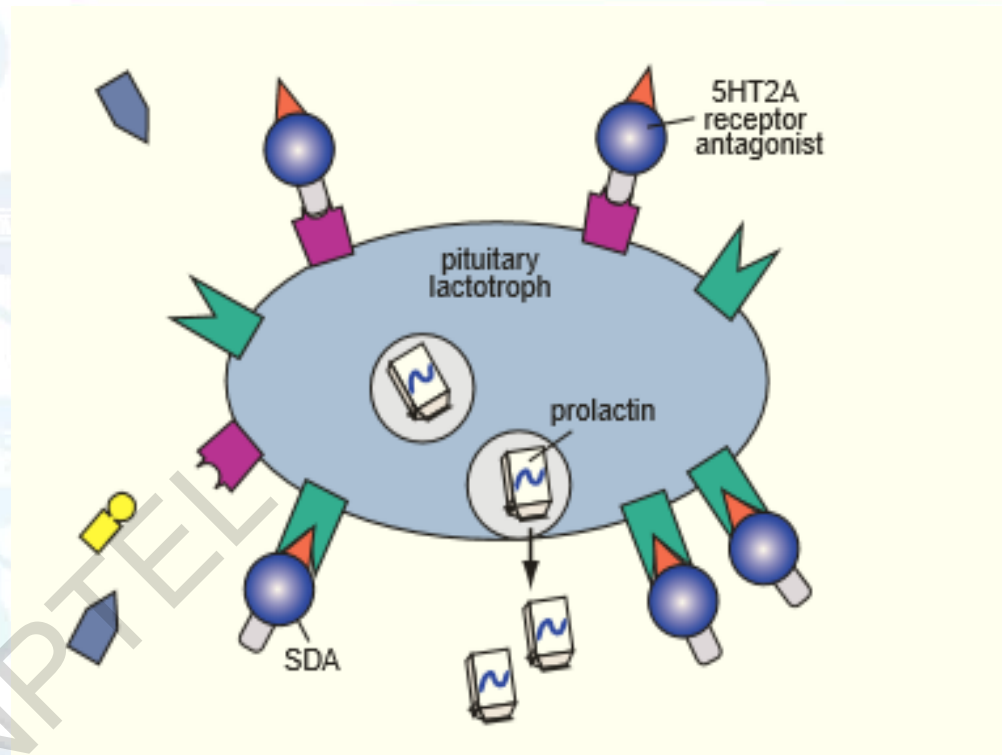
Dopamine inhibits prolactin.

Serotonin stimulates prolactin.





Conventional antipsychotics and prolactin.



Atypical antipsychotics and prolactin.

Risperidone

Atypical antipsychotic (serotonin-dopamine antagonist; second generation antipsychotic.

Active metabolite – 9 hydroxy-risperidone

Half-life upto 20 hours

Works as an Atypical antipsychotic at low doses and conventional antipsychotic at high doses.

Indications (FDA approved)

1. Schizophrenia (Acute & Maintenance)
2. Mania
3. Autistic Spectrum Disorder
4. Intellectual Disability
5. Delaying relapse in schizophrenia
6. Acute mania/mixed mania
7. Autism-related irritability in children ages 5–16 & Bipolar maintenance

Clozapine

Atypical antipsychotic (serotonin-dopamine antagonist; second generation antipsychotic.

Indications (FDA approved)

- 1. Treatment-resistant schizophrenia**
- 2 . Reduction in risk of recurrent suicidal behavior in patients with schizophrenia or schizoaffective disorder.**

Other indications

- ✓ Treatment-resistant bipolar disorder
- ✓ Violent aggressive patients with psychosis and other brain disorders not responsive.
- ✓ Parkinsons disease dementia related psychosis

Olanzapine

Atypical antipsychotic (serotonin-dopamine antagonist) , Second generation antipsychotic

Indications (FDA approved)

- **Schizophrenia (ages 13 and older)**
- **Maintaining response in schizophrenia (long-acting injectable)**
- **Acute agitation associated with schizophrenia (intramuscular)**
- **Acute mania/mixed mania (monotherapy, ages 13 and older) and adjunct to lithium or valproate (adults)**

- Bipolar maintenance
- Acute agitation associated with bipolar I mania (intramuscular)
- Bipolar depression in combination with fluoxetine , ages 10 and older
- Treatment-resistant depression in combination with fluoxetine
- Behavioural disturbances in dementias
- Borderline personality disorder

Quetiapine

Atypical antipsychotic serotonin-dopamine antagonist , Second generation antipsychotic

Indications (FDA approved)

- 1. Acute schizophrenia**
- 2 . Schizophrenia maintenance**
- 3. Acute mania**
- 4. Bipolar maintenance**
- 5. Bipolar depression**

Aripiprazole

Dopamine partial agonist (dopamine-serotonin partial agonist, dopamine stabilizer, atypical antipsychotic, third-generation antipsychotic).

Indications (FDA approved)

- **Schizophrenia (ages 13 to 17) & adult**
- **Maintaining stability in schizophrenia**
- **Acute mania/mixed mania**
- **Bipolar maintenance**
- **Depression**
- **Autism-related irritability in children ages 6 to 17**
- **Tourette's disorder in children ages 6 to 18**
- **Acute agitation associated with schizophrenia or bipolar disorder**

Mechanism Of Action

- 1 . Partial agonism at dopamine 2 receptors
- 2 . Actions at dopamine 3 receptors could theoretically contribute to aripiprazole's efficacy.
3. Blockade of serotonin type 2C and 7 receptors as well as partial agonist actions at 5HT1A receptors may contribute to antidepressant actions.
4. Partial agonism at 5HT1A receptors may be relevant at clinical doses
5. Blocks serotonin 2A receptors, causing enhancement of dopamine release in certain brain regions and thus reducing motor side effects.

Lumateperone

Lumateperone is a newly approved 2nd generation antipsychotic .

It has a unique receptor binding profile and differs from other antipsychotics in that it modulates glutamate, serotonin and dopamine, which are all neurotransmitters that contribute to the pathophysiology of schizophrenia.

Mechanism Of Action

- It acts as a dopamine phosphoprotein modulator (DPPM), with pre-synaptic D2 receptor partial-agonist and post-synaptic antagonist activities.
- Post-synaptic antagonism of D2 receptors by lumateperone increases GSK-3 signaling via phosphorylation.

- D2 receptor activity shows regioselectivity for both mesocortical and mesolimbic circuits with a lower affinity for nigrostriatal dopamine pathways.
- A unique feature of lumateperone is its indirect activity on glutaminergic signaling.
- It exhibits phosphorylative capacity at the GluN2B receptor, which may result from downstream signaling of the D1 receptor.
- Lumateperone also has additional serotonergic modulatory activity, acting as an inhibitor of the SERT, resulting in potential antidepressant activity and efficacy against the negative symptoms of schizophrenia , depression



Most common side effects are

- ✓ Somnolence
- ✓ Sedation
- ✓ Fatigue
- ✓ Constipation

Appetite loss - 5HT_{2A} stimulation (antihistaminics can increase appetite)

GI discomfort, nausea, vomiting- 5HT₃ stimulation

Weight gain - 5HT_{2C} antagonism and antihistaminic effects

Retrograde ejaculation - due to α 1 block, anticholinergic and antihistaminic effects.

Tardive dyskinesia - Supersensitivity of dopamine receptors, which develops because of prolonged therapy with dopamine blocking drugs

CONCLUSIONS

-In this lecture we have discussed regarding concepts of antipsychotics both typical & atypical preferentially haloperidol , chlorpromazine , risperidone , olanzapine , quetiapine , clozapine , aripiprazole & lurasidone

REFERENCES

- 1 Oxford Test book Of Psychiatry
- 2 Comprehensive Text book Of Psychiatry (Kaplan & Sadock)
- 3 Text book Of psychiatry (Tasman & Leiber mann)
4. Stephan stahl's psychopharmacology.



**THANK
YOU !**



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Lecture 37: Antidepressants

CONCEPTS COVERED

- MAO-Inhibitors
- Tricyclic Antidepressants
- Selective serotonin reuptake inhibitor
- Serotonergic Noradrenergic Reuptake Inhibitor
- Serotonin partial agonist reuptake inhibitor (SPARI)

Classic antidepressants: MAO inhibitors

- ✓ The first clinically effective antidepressants to be discovered were inhibitors of the enzyme monoamine oxidase (MAO).

MAO subtypes

MAO-A

Substrates Serotonin Norepinephrine Dopamine
Tyramine
Tissue distribution Brain, gut, liver, placenta, skin

MAO-B

Dopamine Tyramine
Phenylethylamine
Brain, platelets, lymphocytes

MAO-A inhibition increases 5HT, NE, and DA but that the increase in DA is not as great as that of 5HT and NE because MAO-B can continue to destroy DA .

Isoniazide, phenelzine were non selective and irreversible inhibitors used previously but not used now due to drug food interactions

Tyramine is an amine present in various foods, including cheese. A high-tyramine meal acts to increase the release of norepinephrine (NE).

However, in normal circumstances the enzyme monoamine oxidase A (MAO-A) readily destroys the excess NE released by tyramine.

When MAO-A inhibition is taking place in the presence of tyramine, the combination can lead to a very large accumulation of NE.

Such a great NE accumulation can lead to excessive stimulation of postsynaptic adrenergic receptors and therefore dangerous vasoconstriction and elevation of blood pressure.

Moclobemide-

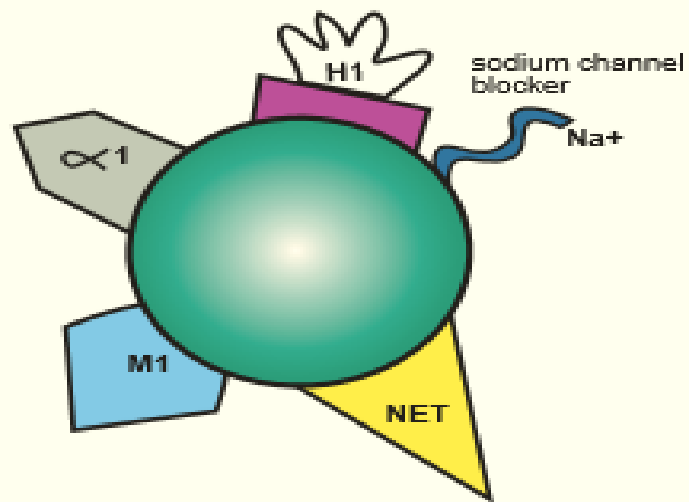
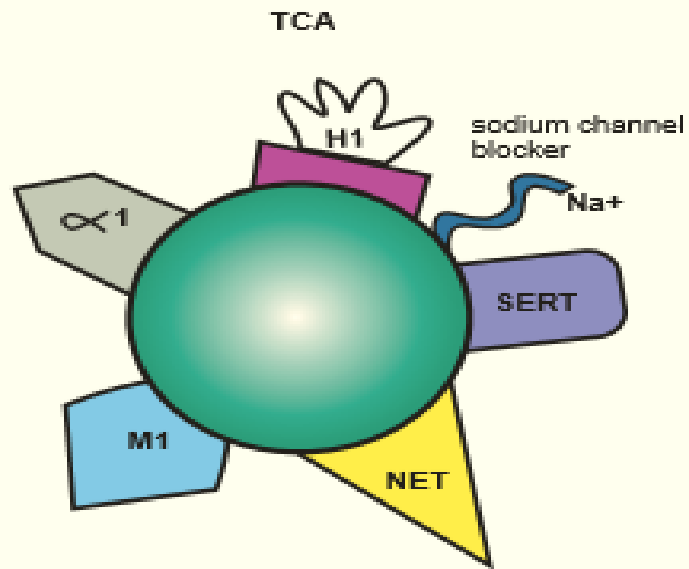
- ✓ **Reversible and selective MAO-A inhibitor**
- ✓ **Short duration of action**
- ✓ **Competitive enzyme inhibition**

Tricyclic Antidepressants

Predominantly NA reuptake inhibitor
Desipramine, Nortriptyline
Amoxapine, Reboxetine

NA + 5 HT reuptake inhibitor

Imipramine, Amitriptyline
Trimipramine, Doxepin
Dothiepin, Clomipramine



Amitriptyline

Serotonin and norepinephrine/noradrenaline reuptake inhibitor

Indications (FDA approved)

- ✓ **Depression**
- ✓ **Endogenous depression**

Mechanism Of Action

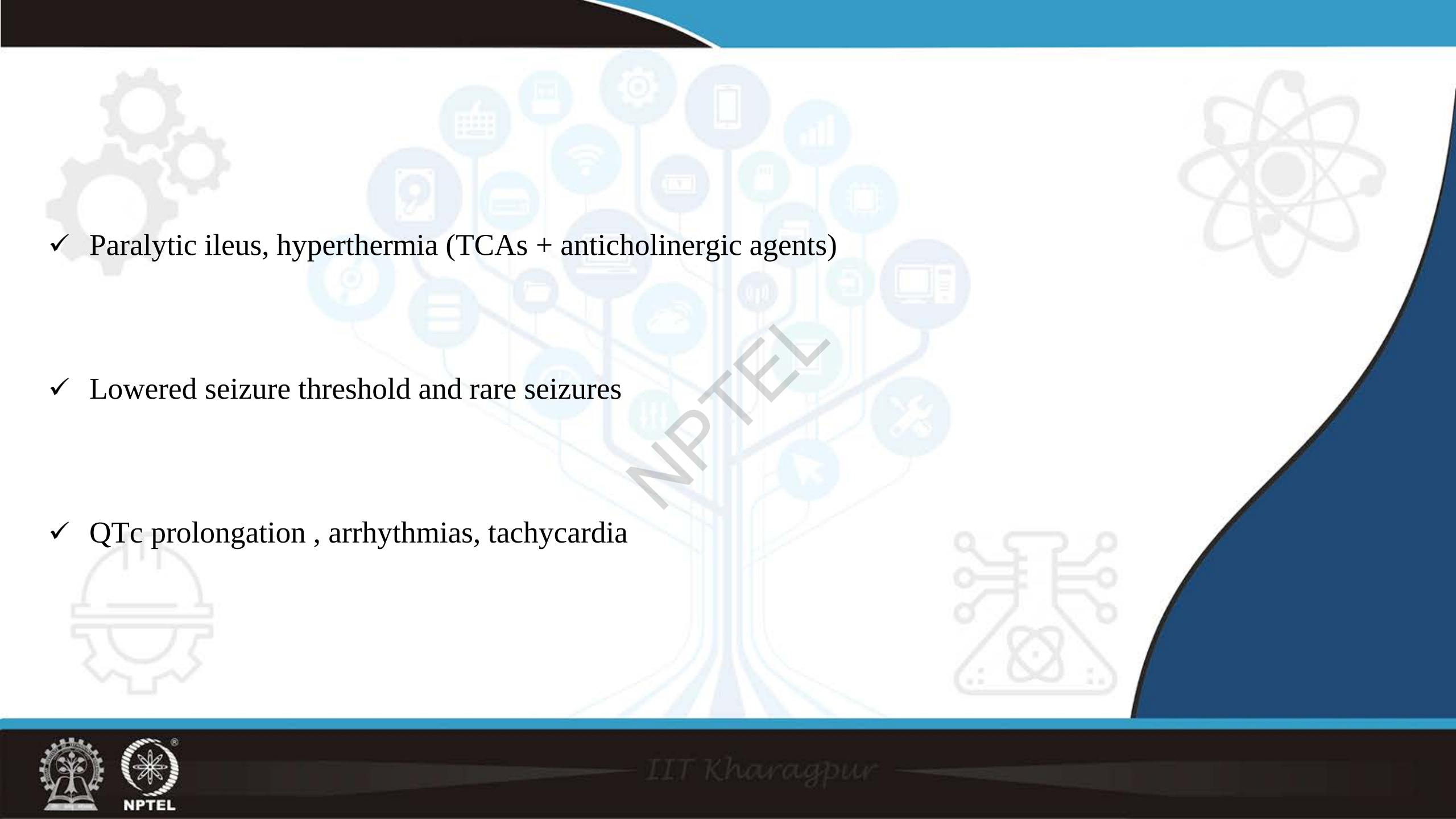
Blocks serotonin reuptake pump (serotonin SERT transporter), increasing serotonergic neurotransmission.

Desensitizes both serotonin 1A receptors and beta adrenergic receptors.

Blocks norepinephrine reuptake pump (norepinephrine transporter), increasing noradrenergic neurotransmission

Side Effects

- ✓ Anticholinergic activity - sedative effects, dry mouth, constipation, and blurred vision
- ✓ Sedative effects and weight gain may be due to antihistamine properties
- ✓ Blockade of alpha adrenergic 1 receptors - dizziness, sedation, and hypotension
- ✓ Fatigue, weakness, dizziness, sedation, headache, anxiety, nervousness.



✓ Paralytic ileus, hyperthermia (TCAs + anticholinergic agents)

✓ Lowered seizure threshold and rare seizures

✓ QTc prolongation , arrhythmias, tachycardia

Dothiepin/dosulepin

Tricyclic antidepressant (TCA) - Serotonin and norepinephrine/noradrenaline reuptake inhibitor

Indications (FDA approved)

- ✓ Major depressive disorder
- ✓ Anxiety
- ✓ Neuropathic pain/chronic pain
- ✓ Treatment-resistant depression

Inhibition of nerve terminal NE neuronal uptake system

Uptake blockade occurs quickly but antidepressant action occurs after months

Increase in synaptic concentrations of NE

Desensitization of inhibitory Alpha2-adrenoceptors in the terminal

Increase in neuronal NE release

desensitize and down regulation of these receptors and induce adaptive changes in the number

Further increase in synaptic concentrations of NE

Desensitization of postsynaptic Beta adrenoceptors without affecting postsynaptic Alpha1-adrenoceptor sensitivity

Mechanism of TCA Action

- ✓ Since **tricyclic and tetracyclic antidepressants** are **frequently associated with weight gain**, before starting treatment, **weigh all patients and determine** if the patient is already overweight (BMI 25.0– 29.9) or obese (BMI >30).

- ✓ **ECGs may be useful for selected patients those with**
 - a) Personal or family history of QTc prolongation
 - b) Cardiac arrhythmia
 - c) Recent myocardial infarction
 - d) Uncompensated heart failure
 - e) Taking agents that prolong QTc interval

Selective Serotonin Reuptake Inhibitor

Mechanism of action

In depression, the 5HT neuron is conceptualized as having a relative deficiency of the neurotransmitter 5HT.

When an SSRI is administered, it immediately blocks the serotonin reuptake pump

this causes serotonin to increase initially only in the somatodendritic area

The increase in 5HT causes the autoreceptors to desensitize / downregulate

there is no longer inhibition of impulse flow in the serotonin (5HT) neuron. Thus, neuronal impulse flow is turned on.

The downregulation of the autoreceptors causes the neuron to release more 5HT at the axon

The increase of 5HT at the axon causes the postsynaptic receptors to desensitize / downregulate, reducing side effects

Fluoxetine

Selective Serotonin Reuptake Inhibitor, also has 5HT_{2C} antagonist actions

Indications (FDA approved)

- ✓ Major depressive disorder
- ✓ Obsessive-compulsive disorder.
- ✓ Premenstrual dysphoric disorder (PMDD)
- ✓ Bulimia nervosa
- ✓ Panic disorder
- ✓ Bipolar depression in combination with olanzapine



Treatment resistant depression

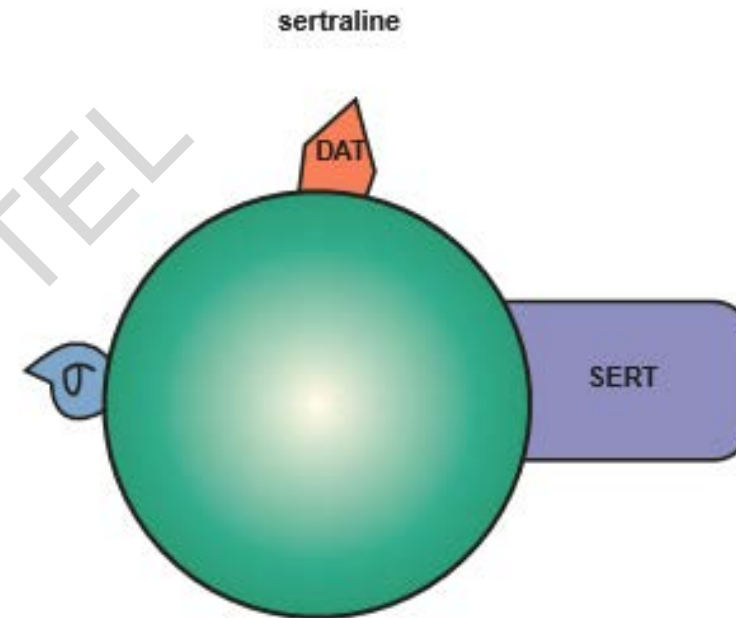
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Sertraline

SSRI - Selective Serotonin Reuptake Inhibitor

Indications (FDA approved)

- ✓ Major depressive disorder
- ✓ Premenstrual dysphoric disorder (PMDD)
- ✓ Panic disorder
- ✓ Posttraumatic stress disorder (PTSD)
- ✓ Social anxiety disorder (social phobia)
- ✓ Obsessive-compulsive disorder (OCD)



Mechanism Of Action

- ✓ Blocks serotonin reuptake pump (serotonin transporter)
- ✓ Desensitizes serotonin receptors, especially serotonin 1A receptors
- ✓ Sertraline also binds at sigma 1 receptors
- ✓ Sertraline also has some ability to block dopamine reuptake pump (dopamine transporter), which could increase dopamine neurotransmission and contribute to its therapeutic actions.

Side Effects

- ✓ **Sexual dysfunction** - Dose-Dependent
men: delayed ejaculation, erectile dysfunction; men and women: decreased sexual desire, anorgasmia
- ✓ **Gastrointestinal** -decreased appetite, nausea, diarrhoea, constipation, dry mouth.
- ✓ Mostly CNS - insomnia but also sedation, agitation, tremors, headache, dizziness.
- ✓ Patients with diagnosed or undiagnosed bipolar or psychotic disorders may be more vulnerable to CNS-activating actions of SSRIs Autonomic (sweating).
- ✓ **Rare bleeding**- Decreases platelet activation.
- ✓ **Rare hyponatremia** Mostly in elderly patients and generally reversible on discontinuation of sertraline.

Selective Norepinephrine Reuptake Inhibitor

Venlafaxine

SNRI dual serotonin and norepinephrine reuptake inhibitor

Indications (FDA approved)

- ✓ **Depression**
- ✓ **Generalized anxiety disorder (GAD)**
- ✓ **Social anxiety disorder (social phobia)**
- ✓ **Panic disorder**

Mechanism Of action

- ✓ Blocks serotonin reuptake pump (serotonin transporter), presumably increasing serotonergic neurotransmission.
- ✓ Blocks norepinephrine reuptake pump (norepinephrine transporter), presumably increasing noradrenergic neurotransmission
- ✓ Presumably desensitizes both serotonin 1A receptors and beta adrenergic receptors.
- ✓ Since dopamine is inactivated by norepinephrine reuptake in frontal cortex, which largely lacks dopamine transporters, venlafaxine can increase dopamine neurotransmission in this part of the brain.

Although there are abundant serotonin transporters (SERTs) and norepinephrine transporters (NETs) in the prefrontal cortex (PFC), there are very few dopamine transporters (DATs).

Dopamine's actions are terminated at norepinephrine axon terminals, because DA is taken up by NET

NET blockade in the prefrontal cortex leads to an increase in synaptic norepinephrine, thus increasing norepinephrine's diffusion radius

Because NET takes up dopamine as well as norepinephrine, NET blockade also leads to an increase in synaptic dopamine.

agents that block NET increase norepinephrine throughout the brain and both norepinephrine and dopamine in the prefrontal cortex

Duloxetine

Serotonin Noradrenergic Reuptake Inhibitor

Indications (FDA approved)

- ✓ Major depressive disorder
- ✓ Diabetic peripheral neuropathic pain (DPNP)
- ✓ Fibromyalgia
- ✓ Generalized anxiety disorder, acute and maintenance
- ✓ Chronic musculoskeletal pain

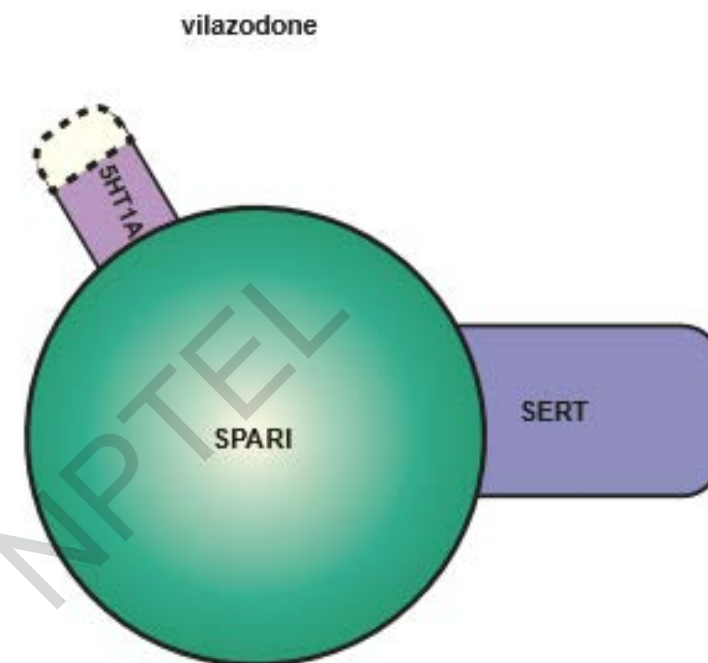
Side Effects

- ✓ Nausea, diarrhea, decreased appetite, dry mouth, constipation (dose dependent)
- ✓ Insomnia, sedation, dizziness
- ✓ Sexual dysfunction -men: abnormal ejaculation/orgasm, impotence
- ✓ Decreased libido; women: abnormal orgasm)
- ✓ Sweating
- ✓ Rare seizures

Serotonergic partial agonist reuptake inhibitor (SPARI)

Indications (FDA approved)

- **Major depressive disorder**
- Anxiety
- Obsessive-compulsive disorder



serotonin partial agonist/reuptake inhibitor

Mechanism Of Action

- ✓ Dual-acting serotonin reuptake inhibitor plus 5HT_{1A} partial agonist.
- ✓ Blocks serotonin reuptake pump (serotonin transporter)
- ✓ Desensitizes serotonin receptors, especially serotonin 1A autoreceptors increases serotonergic neurotransmission
- ✓ Partial agonist actions at presynaptic somatodendritic serotonin 1A autoreceptors, thereby enhance serotonergic activity and contribute to antidepressant actions
- ✓ Partial agonist actions at postsynaptic serotonin 1A receptors .

Side Effects

- ✓ Nausea, diarrhoea, vomiting
- ✓ Insomnia, dizziness
- ✓ Bleeding , Seizures , Suicidality in adolescents
- ✓ Hyponatremia (mostly in elderly patients and generally reversible on discontinuation of vilazodone)
- ✓ Sexual dysfunction (men: delayed ejaculation; men and women:
- ✓ Decreased sexual desire, anorgasmia
- ✓ SIADH -syndrome of inappropriate antidiuretic hormone secretion

Mirtazapine

- ✓ Noradrenergic and specific serotonergic antidepressant
- ✓ Blocks **alpha 2 auto receptor** (on NA neuron) and hetero- (on 5-HT neuron) receptors increasing both NA and serotonin release.

Bupropion

- ✓ Inhibits **DA** and NA uptake has excitant effect
- ✓ Used to reduce smoking

CONCLUSIONS

-In this lecture we have discussed regarding concepts of selective serotonin reuptake inhibitor , serotonergic noradrenergic reuptake inhibitor, noradrenergic serotonergic agent, tricyclic antidepressants , serotonergic partial agonist reuptake inhibitor its mechanism of action indications & side effects.

REFERENCES

- 1 Oxford Test book Of Psychiatry
- 2 Comprehensive Text book Of Psychiatry (Kaplan & Sadock)
- 3 Text book Of psychiatry (Tasman & Leiber mann)
4. Stephan stahl's psychopharmacology.



**THANK
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Course Name Basics Of Mental Health & Clinical Psychiatry

Faculty Name Dr Sumit Kumar

Department Name Psychiatry

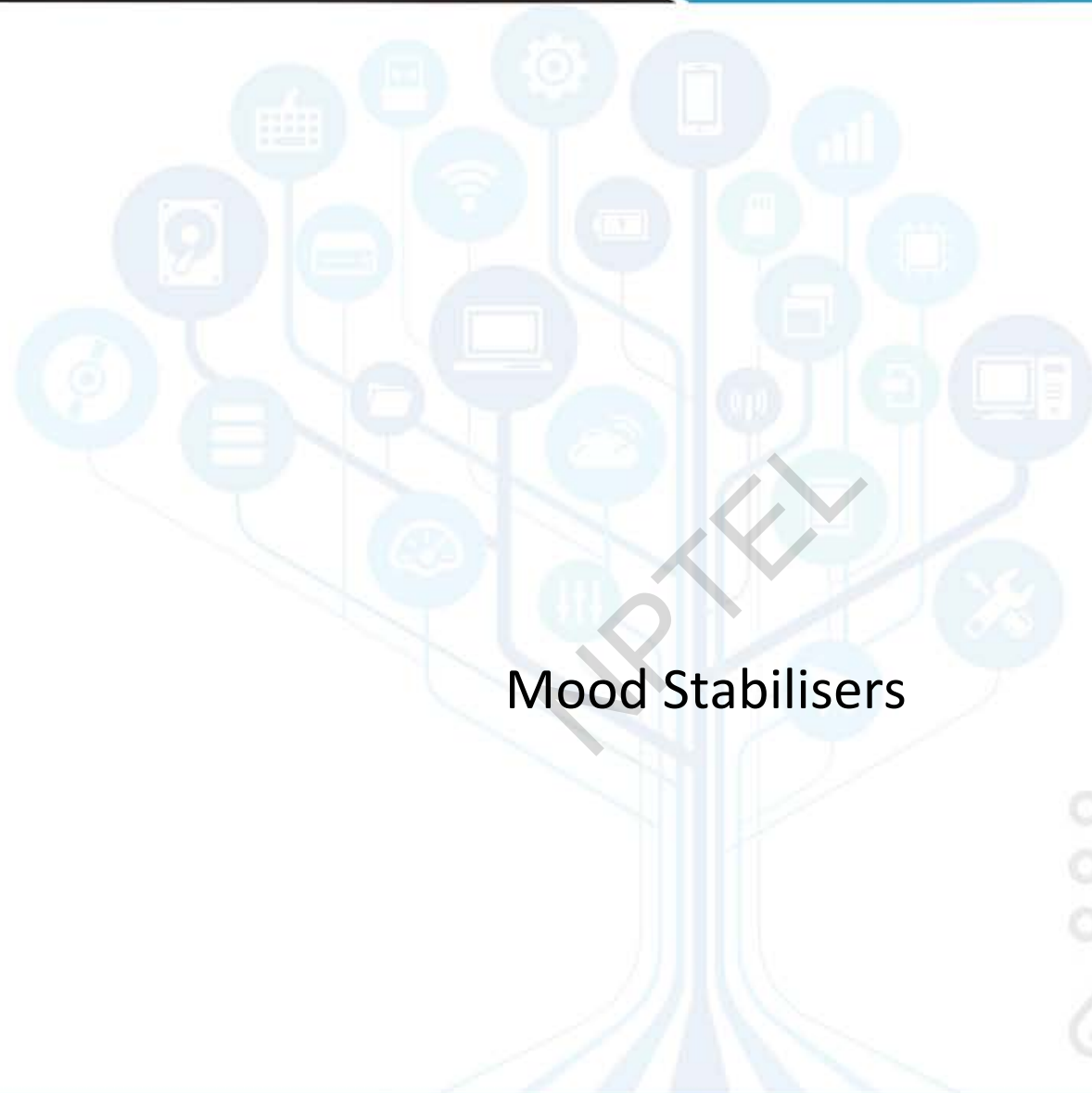
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Lecture 38 : Mood Stabilizer

CONCEPTS COVERED

- Lithium
- Valproate
- Carbamazepine
- Topiramate
- Lamotrigine

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Mood Stabilisers



Lithium

Lithium is the third element of the periodic table, in the same column as hydrogen and sodium.

Indications

- ✓ Maintenance of Bipolar Disorder
- ✓ Augmentation of Major depressive disorder (Unipolar depression)
- ✓ Acute treatment of Mania
- ✓ Prophylaxis of Unipolar Depression

Other uses of Lithium (Not FDA approved)

- ✓ Lithium is also used to treat aggressive and self-mutilating behaviour, and recent studies
- ✓ raise the white blood cell (WBC) count in patients receiving clozapine.
- ✓ Used to prevent & treat steroid induced psychosis
- ✓ Antisuicidal Properties

Neuroprotective

- 1 .Lithium may have neuroprotective effects that preserve the function of neurons and neuronal circuits.
- 2 .Lithium also promotes **the creation of new neurons (neurogenesis) in the hippocampus,** which **is potentially important for learning, memory and stress responses.**

- ✓ Lithium is neither metabolized nor protein-bound, thus its bioavailability usually is close to 100%.
- ✓ Lithium is more susceptible to accumulation in the liver, bone, muscle, or thyroid with brain and kidney showing the highest level.
- ✓ Lithium freely crosses the placenta and is also excreted in breast milk. It is labeled as pregnancy class D.
- ✓ Lithium was first introduced as a mania and gout treatment approved by FDA in 1974.
- ✓ **It is worth noting that lithium toxicity signs do not often conform to the measured lithium level.**

- ✓ Producing brain inositol depletion, leading to reduced responsiveness to alpha-adrenergic stimulation.
- ✓ Reducing neuronal responsiveness to neurotransmitters due to inhibitory effects on adenylate cyclase and G proteins vital for ion channel opening.
- ✓ Stimulating serotonin release from the hippocampus.

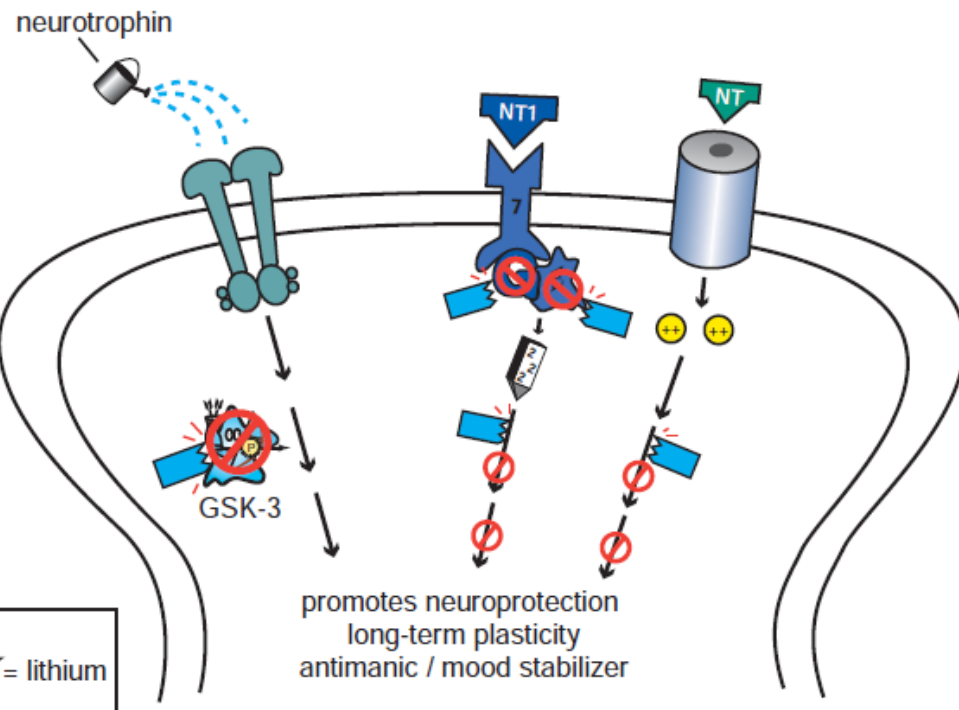
Therapeutic Level:

.6- 1.2 mEq/L

Maintenance Level: .5 – 1 mEq/L

Mechanism Of Action: Lithium

Possible Mechanism of Lithium Action on Downstream
Signal Transduction Cascades



1. Signal transduction sites which includes second messengers, phosphatidylinositol system, where lithium inhibits the enzyme inositol monophosphatase
2. Modulation of G proteins
3. Regulation of gene expression for growth factors and neuronal plasticity by interaction with downstream signal transduction cascades, including inhibition of GSK-3 (glycogen synthase kinase 3) and protein kinase C.

Drug interactions with lithium

✓ Angiotensin-converting enzyme (ACE) inhibitors can

a) Reduce thirst leading to mild dehydration

b) Increase renal sodium loss leading to increased Na re-absorption by the kidney, resulting in an increase in lithium plasma levels.

✓ Diuretics can reduce the renal clearance of lithium, the magnitude of this effect being greater with thiazide than loop diuretics. Lithium levels usually rise within 10 days of a **thiazide diuretic** being prescribed

Pre treatment work up for Lithium

- ✓ Pre-lithium workup eGFR and TFTs. ECG recommended in patients who have risk factors for, or existing cardiovascular disease.
- ✓ Baseline measure of weight desirable.
- ✓ Blood should be taken 12 hours after the last dose.
- ✓ Monitoring Plasma lithium every 6 months
- ✓ GFR and TFTs every 6 months. Weight (or BMI) should also be monitored

Signs and Symptoms Of Lithium toxicity

❑ Mild & Moderate Intoxication (1.5-2.0mEQ/L)

Gastrointestinal – Vomiting

Abdominal Pain

Dryness of Mouth

Neurologic-

Ataxia

Dizziness

Slurred of Speech

Nystagmus

Muscle Weakness

☐ Moderate & Severe Intoxication (2.0-2.5 mEQ/L)

Gastrointestinal - Anorexia

Persistent nausea & Vomiting

Neurologic - Blurred Vision

Fasciculations

Hyperactive Deep Tendon reflexes

Convulsions

Delirium

Stupor

Coma

Circulatory Failure (lowered BP , Cardiac Arrhythmia

☐ Severe Lithium Intoxication (Lithium Level >2.5 mEQ/L)

Generalised Convulsions

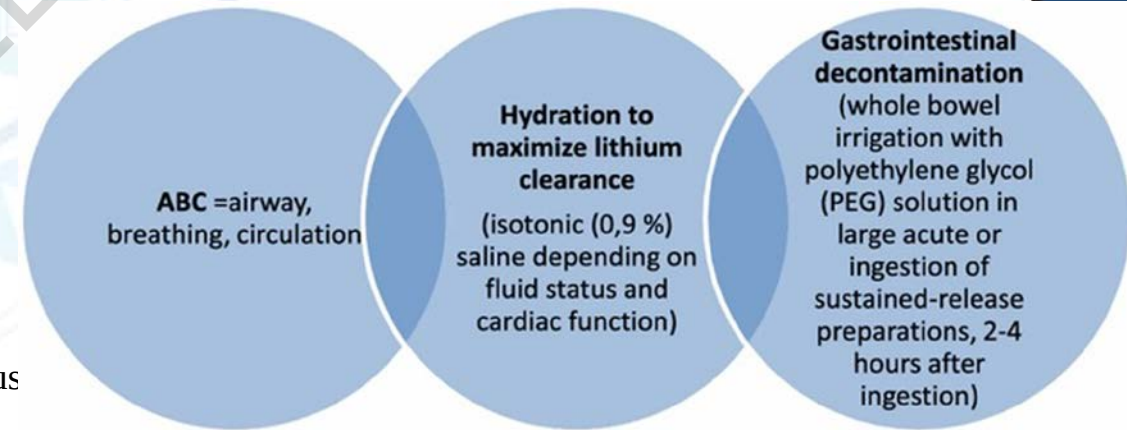
Oliguria and Renal Failure

Death

Signs
and
Symptoms
Of
Lithium toxicity

Treatment

- Lithium has a very narrow therapeutic index, and toxic levels are when the drug is above **2 mEq/L**.
- **Lithium toxicity has no antidote.**
- Treatment for lithium toxicity is **primarily hydration** and to stop the drug.
- Give hydration with normal saline, which will also enhance lithium excretion.
- **Avoid all diuretics. Gastrointestinal Decontamination**
- If the patient has severe renal dysfunction or failure, or severely altered mental status **hemodialysis**.
- 20 to 30 mg of propranolol given 2 to 3 times per day may help reduce tremors.



Valproate



Valproate

- ✓ Most widely prescribed mood stabiliser in psychiatry.
- ✓ Its called valproic acid because it is converted into acidic form in the stomach.
- ✓ Metabolized by hepatic glucuronidation & mitochondrial beta oxidation.

Plasma half – life- 10 -16 hrs:

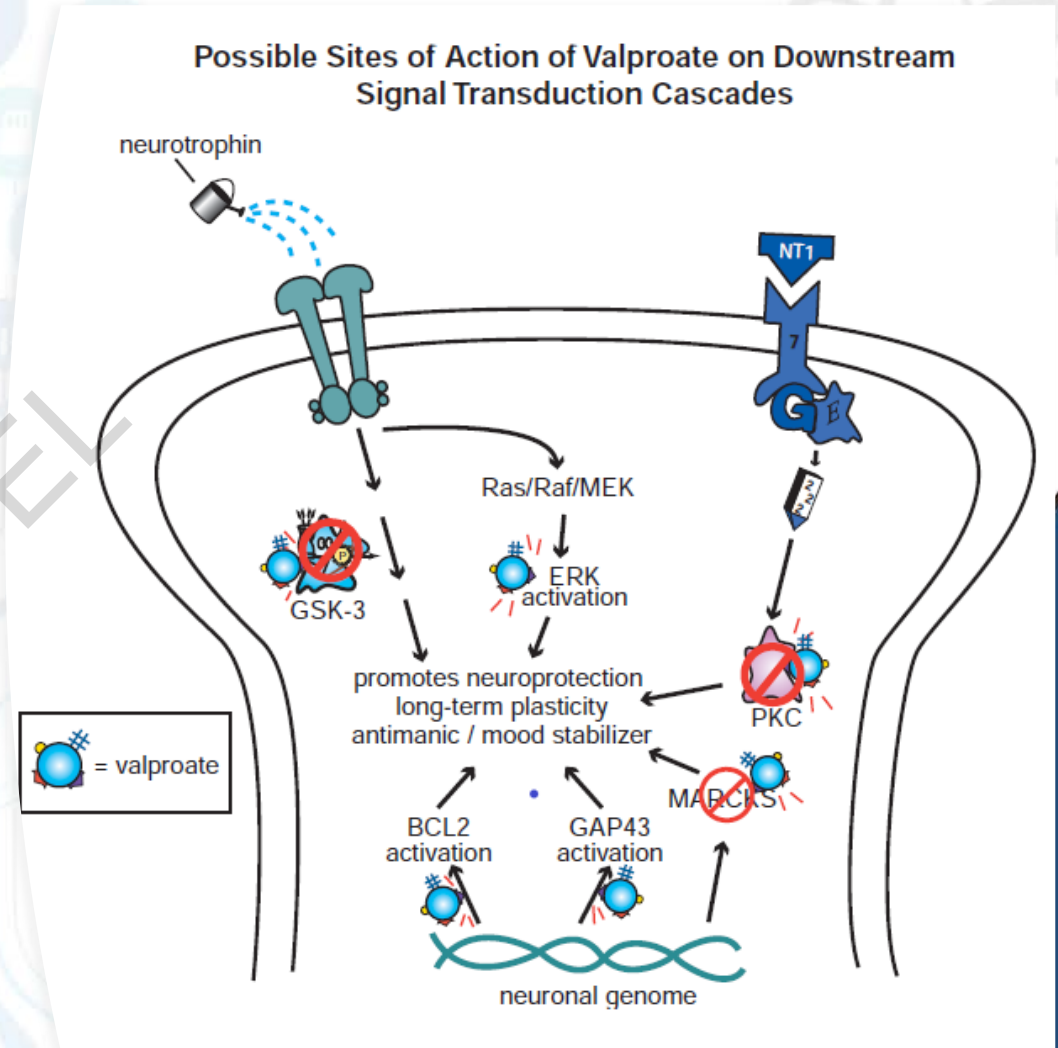
Therapeutic dose range- 20-30 mg/kg body weight

Therapeutic Indications

- Acute Mania
- Acute bipolar depression
- Prophylaxis of depression both(unipolar & bipolar)
- Augmentation of antipsychotics in treatment of schizophrenia & Schizoaffective disorder
- Behavioural agitation , Personality disorder , dementia with Behavioural & psychological symptoms .

Mechanism Of Action: Valproate

- ✓ Enhancement of GABA activity.
- ✓ Modulation of voltage sensitive sodium channels multiple downstream effects on signal transduction cascades, which may be involved in its antimanic effects.
- ✓ Valproate inhibits glycogen synthase kinase 3 (GSK- 3), protein kinase C (PKC) and myristolated alanine-rich C kinase substrate (MARCKS).



Common adverse effects

Nausea , vomiting , dyspepsia , diarrhoea , sedation , ataxia , tremors , dysarthria , weight gain , hair loss.

Neuroprotective

Valproate activates signals that promote neuroprotection and long-term plasticity, through

- a) Extracellular signal-regulated kinase (ERK)
- b) Cytoprotective protein B-cell lymphoma/leukemia-2 gene (BCL2), and GAP43

Serious side effects

i) **Hepatotoxicity-**

Risk factors- young age (less than 3 years), presence of neurologic disorders , use of phenobarbital
Pain abdomen , anorexia , malaise , nausea , oedema suspect hepatic issues

ii) **Pancreatitis-** observed during first 6 months of use.

iii) **Hyperammonemia-** more commonly observed with carbamazepine Associated with coarse tremors and respond with L-carnitine.

iv) **Teratogenicity-** spina bifida (Neural tube defects 1 -4 %) during first trimester of pregnancy

v) **Thrombocytopenia-** bleeding gums , bruising

vi) **Metabolic Syndrome** due to Insulin resistance & hyperinsulinemia

Lamotrigine

- ✓ Was developed as antiepileptic drug for adjunctive treatment of partial Seizures.
- ✓ Approved for maintenance treatment for bipolar disorder (FDA,2003).
- ✓ No approval for Acute Bipolar Depression

Half-life- 25 hours

Mechanism Of action-

- i) Blockade of Voltage sensitive sodium channels
- ii) Increases serotonin concentration through inhibition of serotonin uptake (weak inhibitor of 5HT₃ receptors).

Side Effects

Common side effects- dizziness , somnolence , headache , diplopia , nausea are mild in nature.

Severe side effect

Rash- it can appear within first 4 months of starting the drug.

If the drug is titrated too fast with rapid hike in the doses, rashes can occur which may represent early manifestations of Steven-Johnson syndrome/Toxic Epidermal Necrolysis. (Incidence of Rash- 0.08%)

Children less than 16 years appear to be more susceptible to rash

Drug Interactions

- i) Serious interaction with Valproate (Enzyme Inhibitor) which doubles the concentration of Lamotrigine.
- ii) Increases concentration of lamotrigine when given along with sertraline ,paroxetine).

Carbamazepine

- ✓ Initially developed as an Antidepressant
- ✓ Approved for use in trigeminal Neuralgia & Complex Partial Seizures

Half-life – 26 hours

(with chronic administration its half-life reduces to 12 hours due to its enzyme inducing properties.(autoinducer).

Mechanism Of Action-

Therapeutic dose range-

10-12mg/kg body weight

- i) Binding of voltage dependant sodium channels in inactive state thereby prolonging their inactive state.
- ii) Reduces voltage dependant calcium channel activation.
- iii) Reduction of N methyl-D aspartate (NMDA) glutamate receptor channels.

Indications

- i) Acute Mania
- ii) Prophylaxis of Bipolar disorder II
- iii) Help to control acute alcohol withdrawal.
- iv) Controls agitation , impulsive aggressive behaviour.

Adverse effects-

Common side effects

Nausea, vomiting , gastric distress , constipation , diarrhoea , anorexia , ataxia drowsiness.

Serious side effects

- i) **Blood dyscrasias-** Effects are dose related

incidence- 1 in 125000 treated with carbamazepine (pancytopenia , agranulocytosis , aplastic anaemia).

Symptoms of sore throat, rash , petechiae , bruising and easy bleeding should seek medical evaluation immediately

ii) Hepatitis-

- a) Within the first few weeks of starting carbamazepine both hepatitis and increase in serum transaminases is seen.
- b) Persistent elevation of more than 3 times the upper limit indicates need to discontinue the drug.

iii) Dermatologic effects-

Rash is observed within 3 weeks of starting treatment

Some patients experience life threatening dermatologic syndromes namely , exfoliative dermatitis, stevens-johnson syndrome , toxic epidermal necrolysis.

Patients who are good responders to the drug can be continued with prednisolone 40 mg a day without having to stop the carbamazepine.

iv) Renal Effects-

Carbamazepine activates vasopressin receptor function which results in a condition resembling the syndrome of inappropriate antidiuretic hormone characterized by hyponatremia(water intoxication).

Maternal use of carbamazepine during pregnancy

Cleft palate, fingernail hypoplasia , microcephaly , spina bifida in infants

Carbamazepine treatment should be discontinued

a) When WBC count $< 3000/\text{mm}^3$

b) Neutrophils below $< 1500/\text{mm}^3$

c) Serum iron $< 150 \text{ mg/ml}$

a) Platelet $< 100,000 \text{ mm}^3$

b) Haemoglobin $< 11\text{g}\%$



Topiramate

Developed as an anticonvulsant useful in neurologic and psychiatric illness.

Indications-

- i) FDA approved for seizure as monotherapy or augmentation with other antiepileptics
- ii) Useful in prevention of migraine (FDA 2012).
- iii) Treatment of obesity , bulimia nervosa off label use
- iv) Useful in decreasing tremors.

Mechanism Of action

- a) Blocks voltage gated – sodium channels.
- b) Reduces membrane depolarization by AMPA/Kianate receptors.
- c) Enhances GABA (A) receptor activity

Most common adverse effects

- a) Paresthesias , weight loss , somnolence , anorexia , dizziness ,memory problems.
- a) Low serum bicarbonate – formation of renal stones.

REFERENCES

- 1 Oxford Text book Of Psychiatry
- 2 Comprehensive Text book Of Psychiatry (Kaplan & Sadock)
- 3 Text book Of psychiatry (Tasman & Leiber mann)
4. Stephan stahl's psychopharmacology

CONCLUSIONS

-In this lecture we have discussed regarding mood stabilizer used in clinical practice for treating various psychiatric illness , its mechanism of action , indications & related side effects.



**THANK
YOU !**



NPTEL ONLINE CERTIFICATION COURSES

Basics of Mental Health & Clinical Psychiatry

Dr. Arijita Banerjee

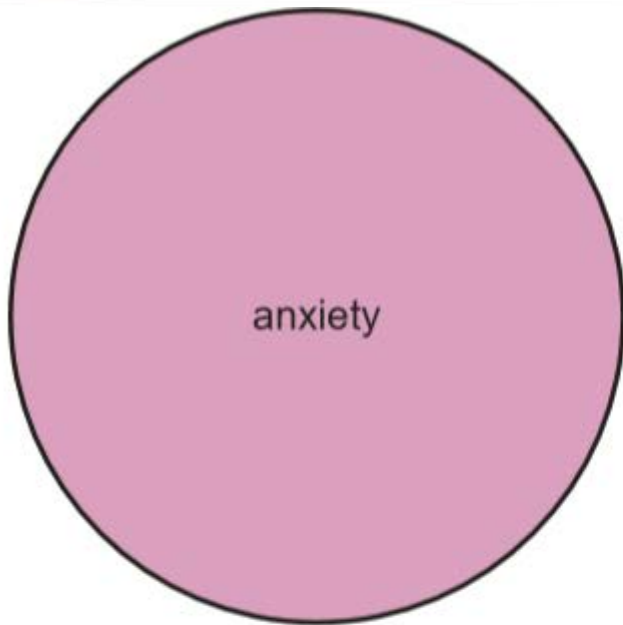
**Dr B.C. Roy Multi-speciality Medical Research Centre
IIT KHARAGPUR**

Lecture 39: Anti-anxiety drugs

CONCEPTS COVERED

1. Spectrum of anxiety.
2. Neurotransmitters involved.
3. Role of drugs

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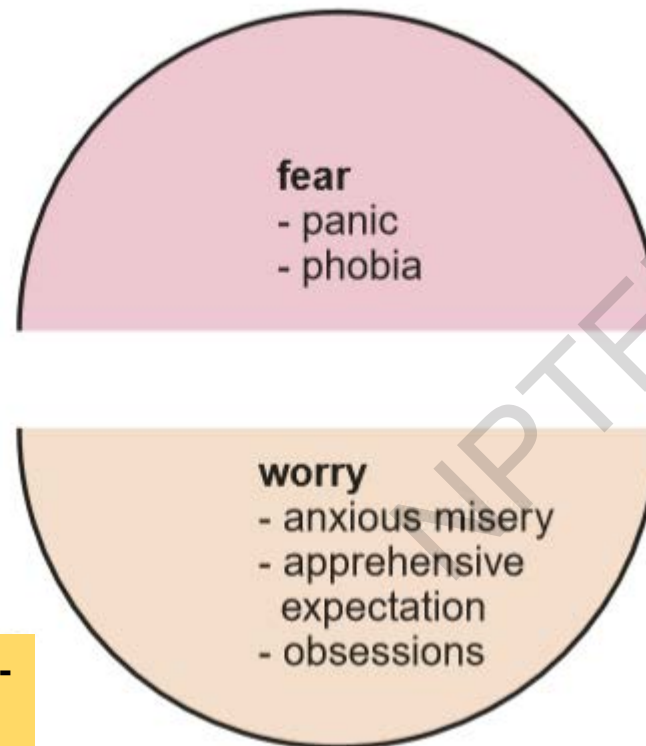


deconstruct the syndrome...



...into symptoms

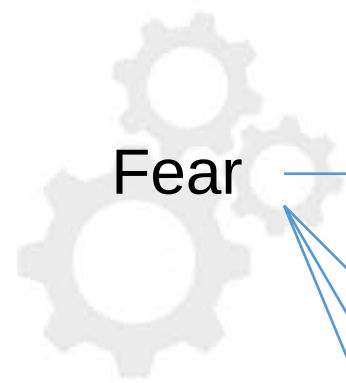




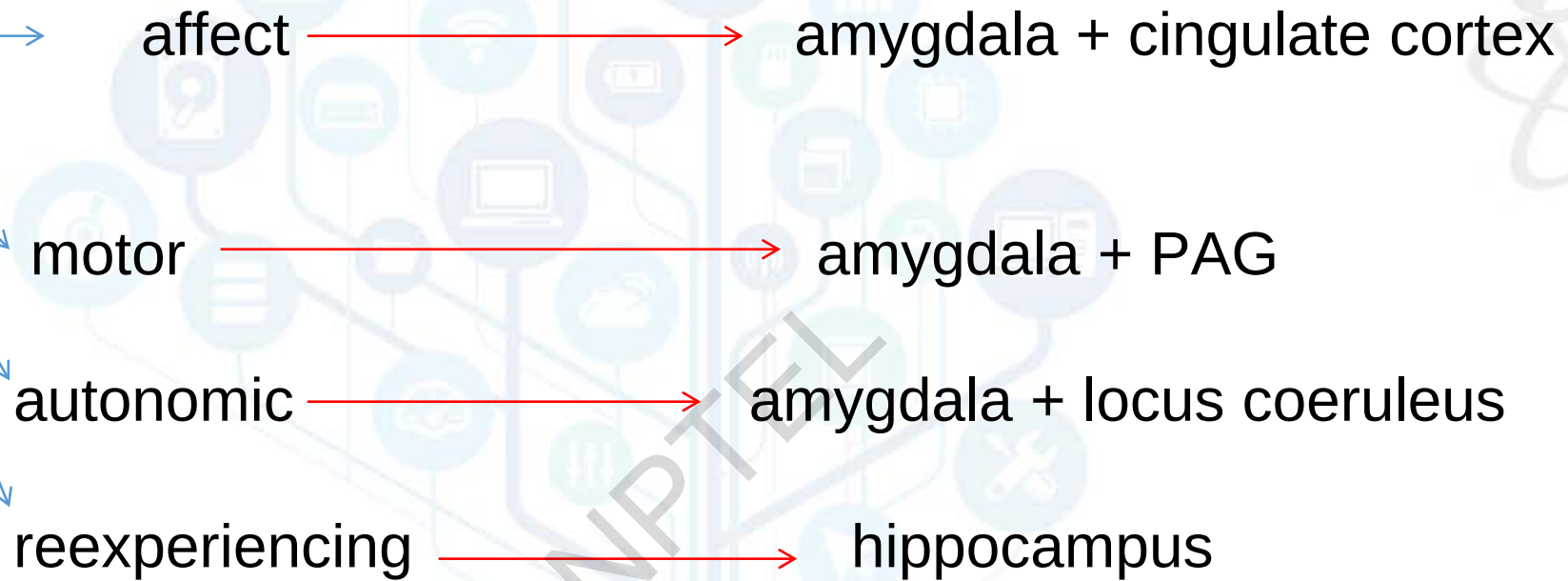
Amygdala

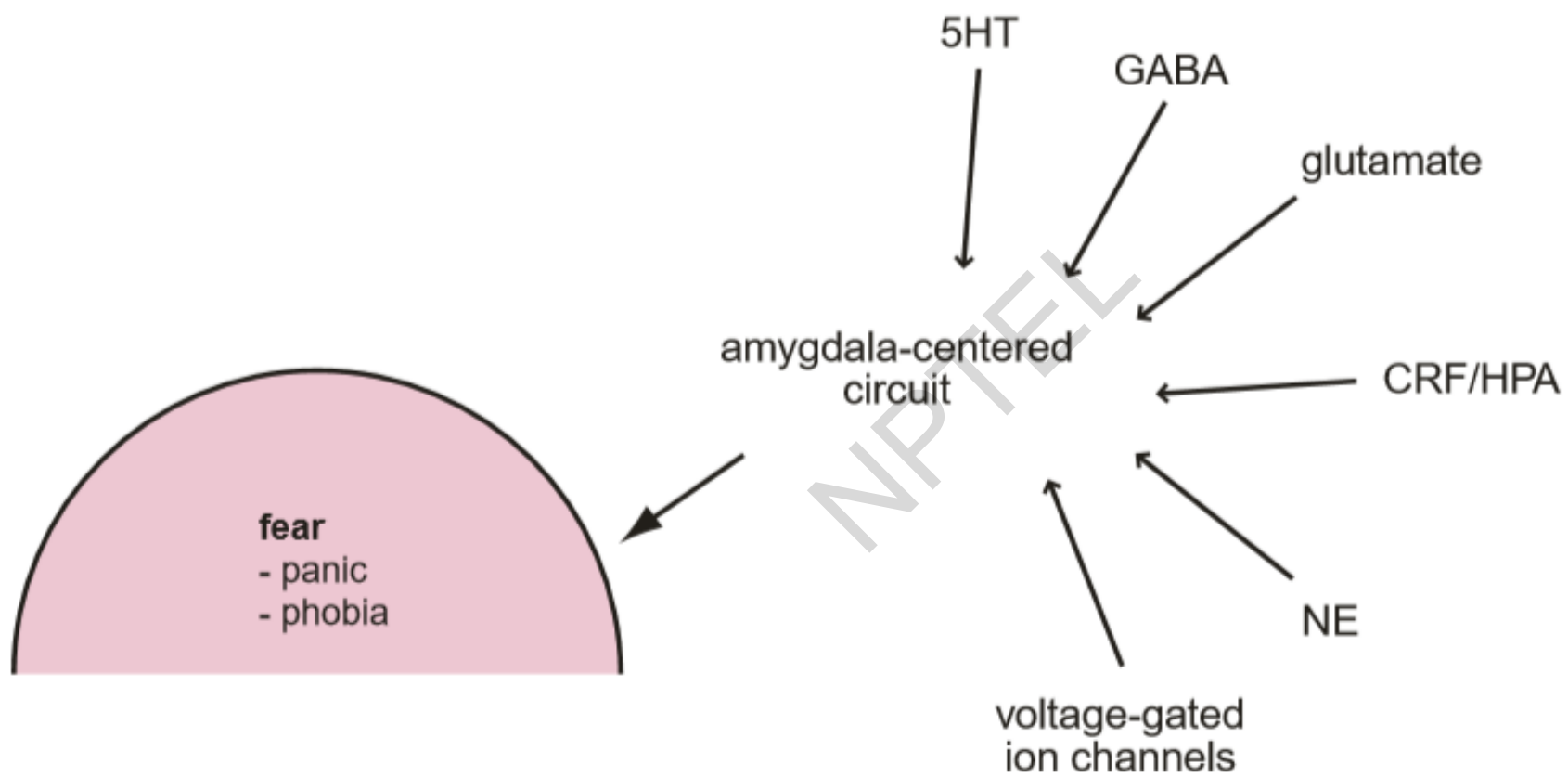


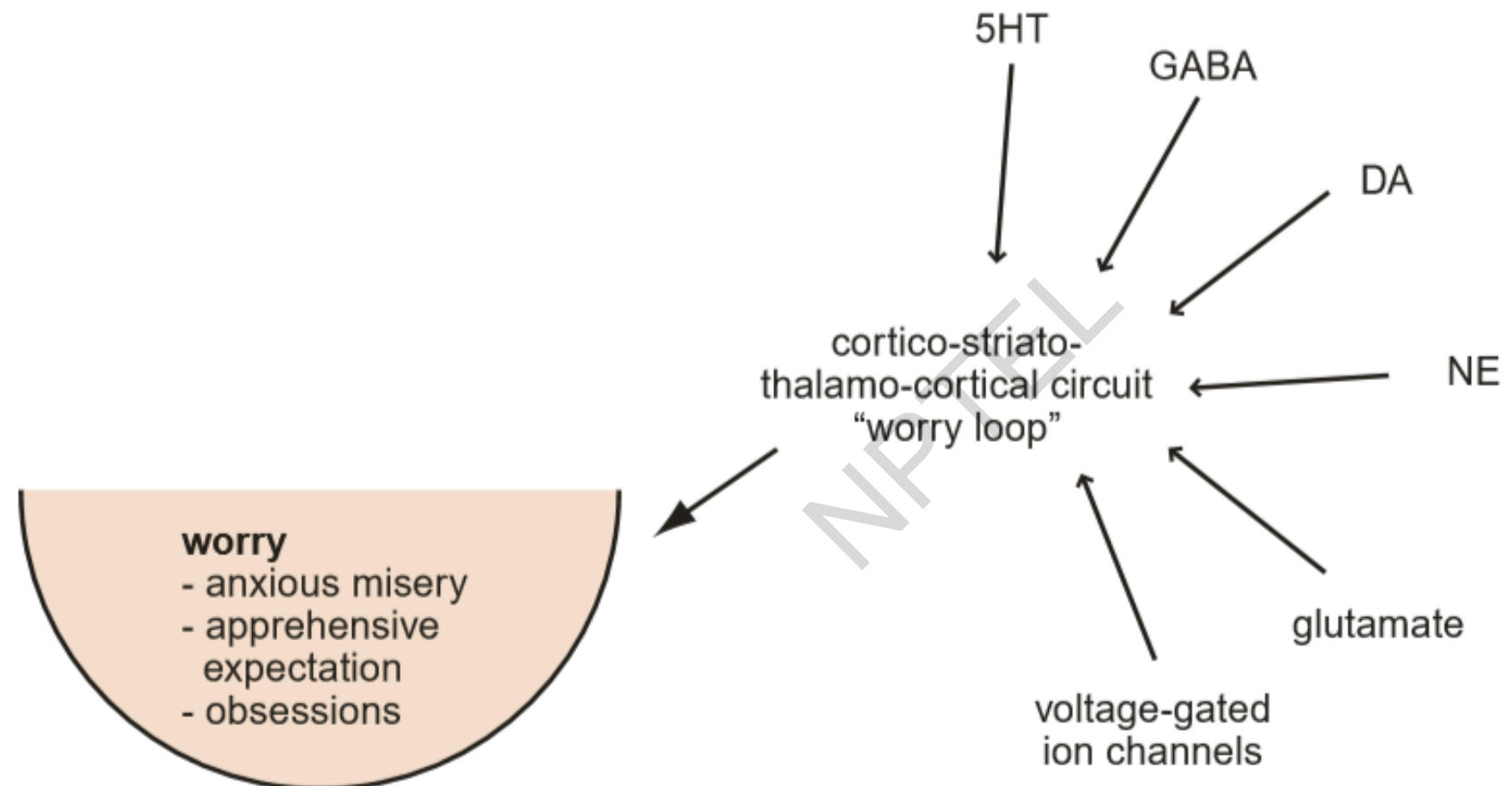
**Cortico-thalamo-
Cortico loops**



Fear

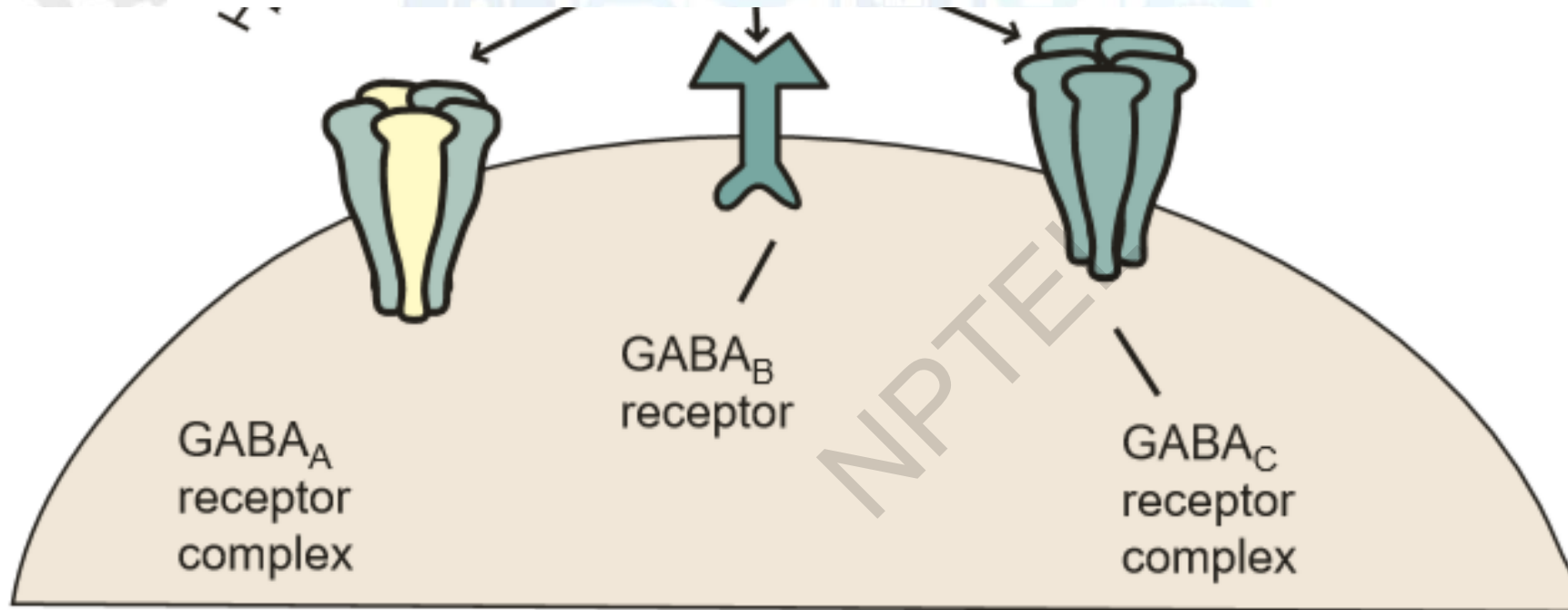


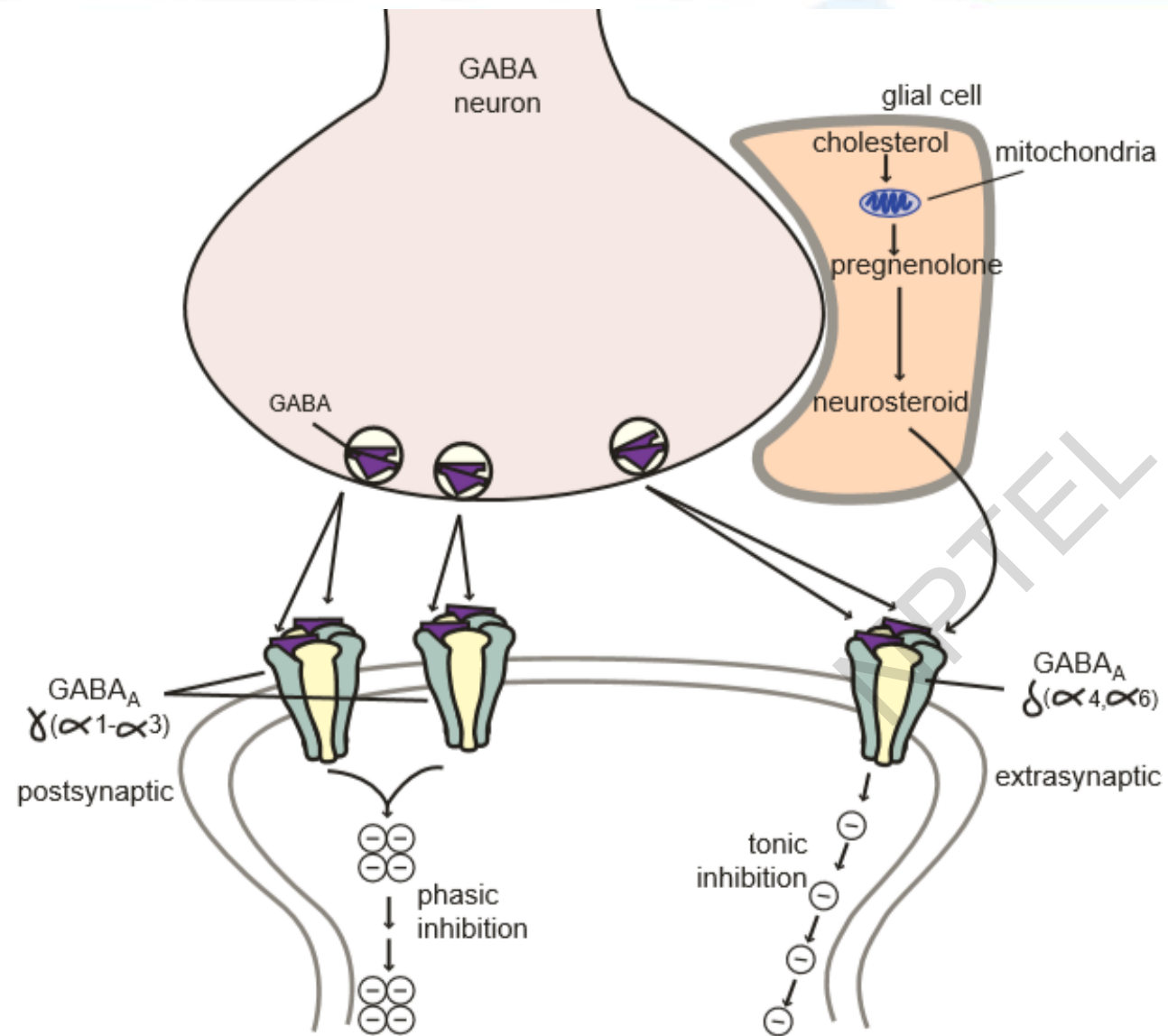




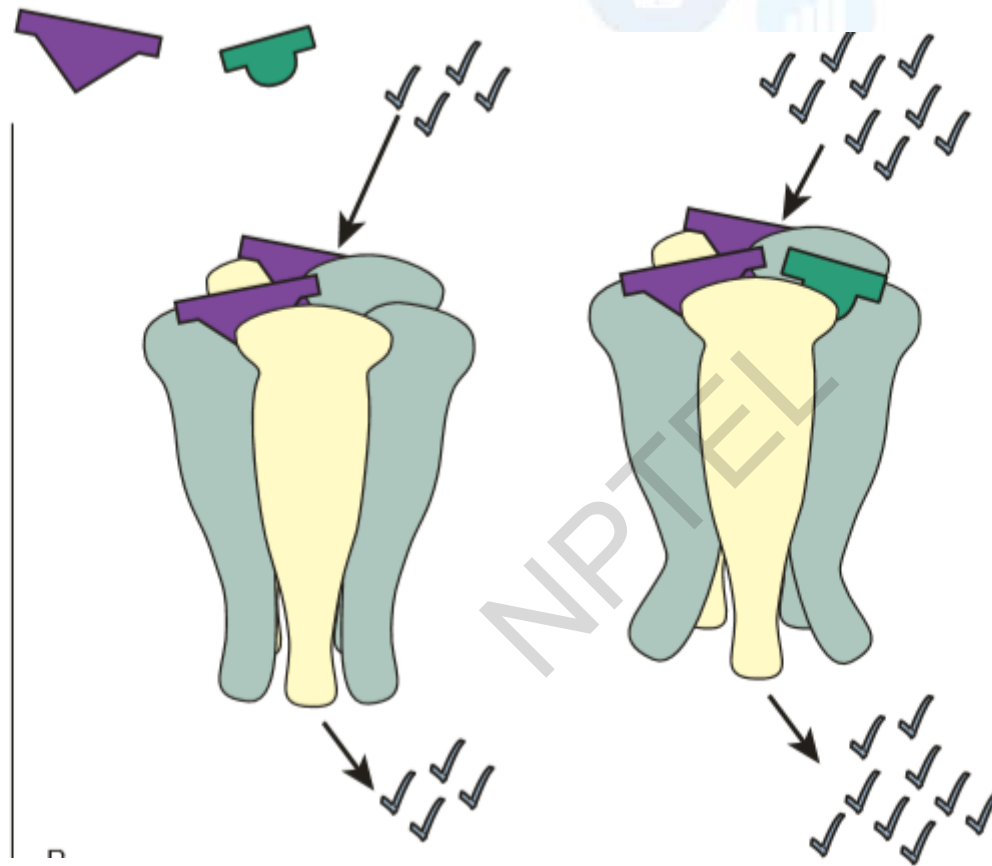
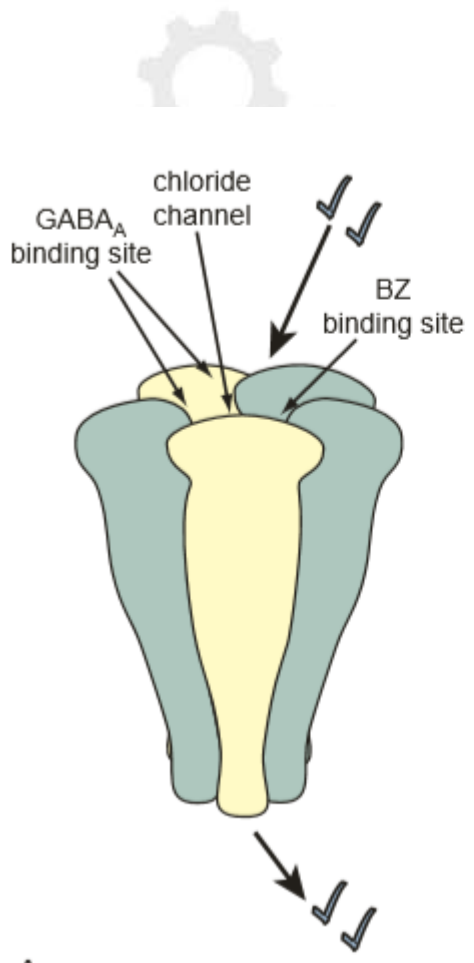
GABA and Benzodiazepines

The amino acid glutamate, a precursor to GABA, is converted to GABA by the enzyme glutamic acid decarboxylase (GAD).



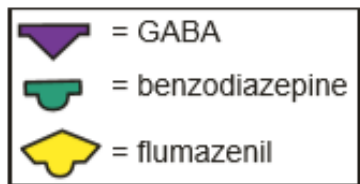
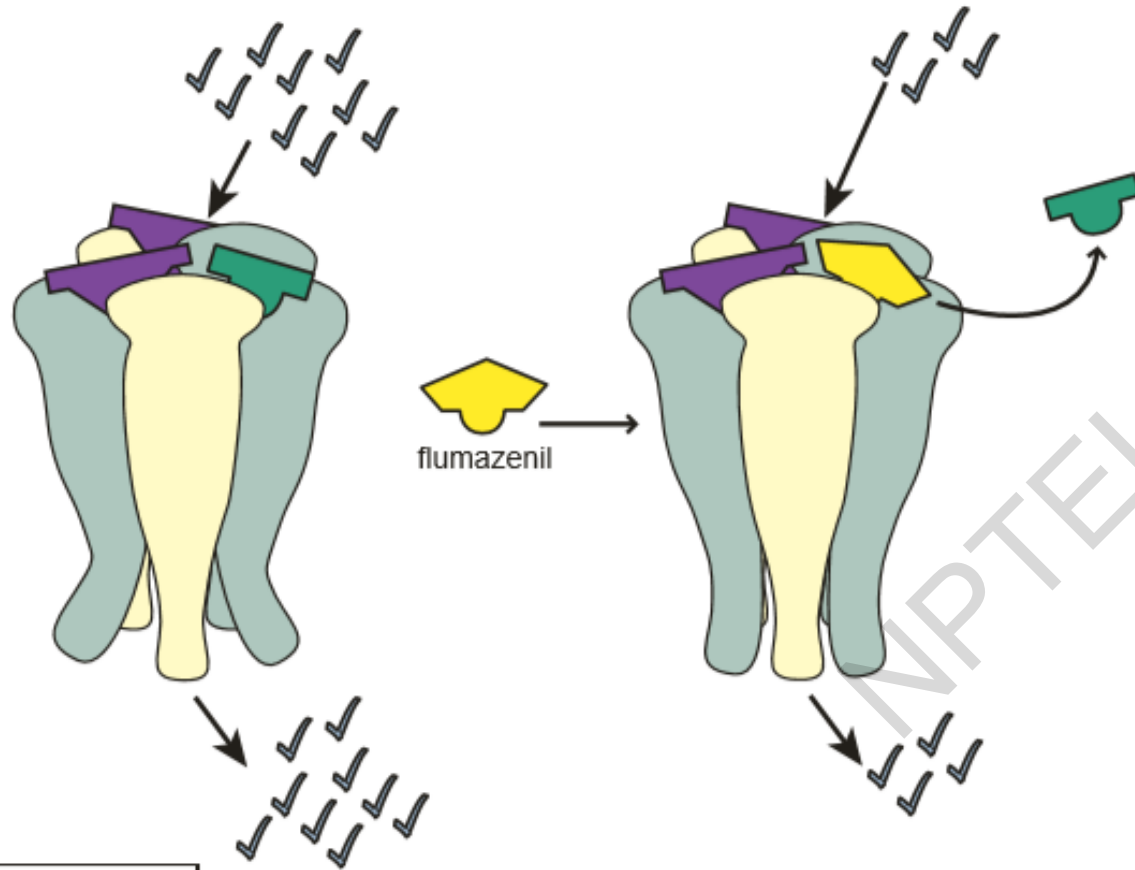


Benzodiazepine-sensitive GABA_A receptor subtypes (with γ subunits and $\alpha 1-3$ subunits) are thought to be postsynaptic in location and to mediate a type of inhibition at the postsynaptic neuron that is phasic, occurring in bursts of inhibition that are triggered by peak concentrations of synaptically released GABA



When a positive allosteric modulator such as a benzodiazepine binds to the GABA_A receptor in the presence of GABA, it causes the channel to open even more frequently than when GABA alone is present.

Positive allosteric modulation of GABA_A receptors



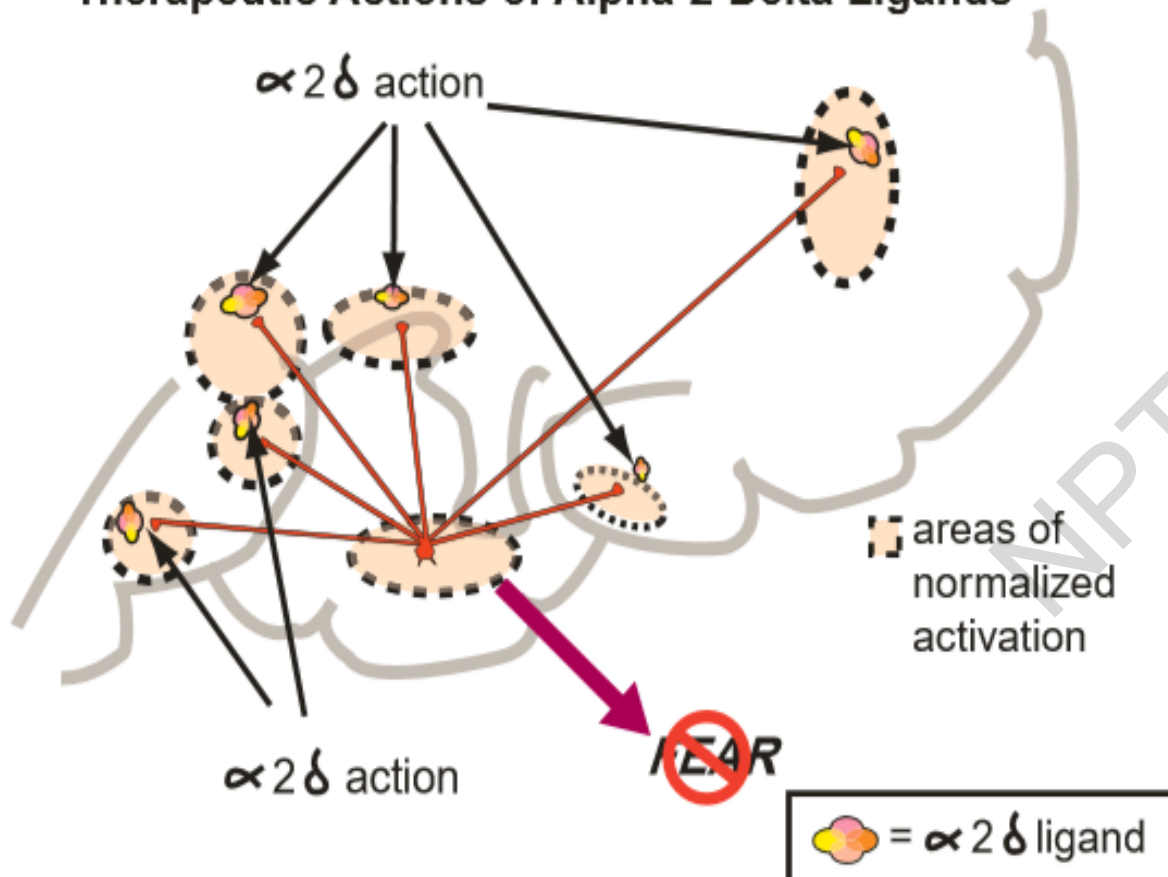
Flumazenil is a short-acting intravenously administered antagonist to benzodiazepines that can reverse overdoses or anesthesia from benzodiazepines but can also induce seizures or withdrawal in patients dependent upon benzodiazepines.

Benzodiazepines

- Given orally/iv/im
- Oral absorption is good
- Chlordiazepoxide-1st BZD used
- Lorazepam/oxazepam-short acting
- Alprazolam-anti anxiety and anti-depressant
- s/e-sedation, cognitive impairment, dependance, weight gain

Alpha-2-delta ligands as anxiolytics

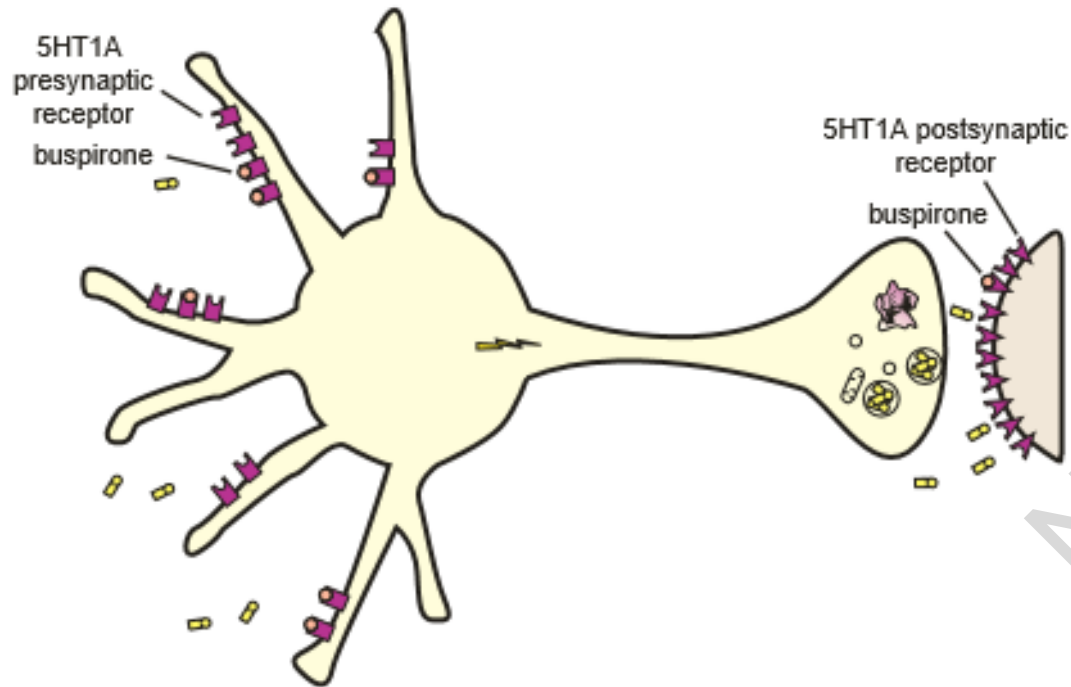
Therapeutic Actions of Alpha-2-Delta Ligands



The $\alpha 2\delta$ ligands **pregabalin** and **gabapentin** have demonstrated anxiolytic actions in social anxiety disorder and panic disorder, and are also proven to be effective for the treatment of epilepsy and certain pain conditions, including neuropathic pain and fibromyalgia.

Serotonin and anxiety

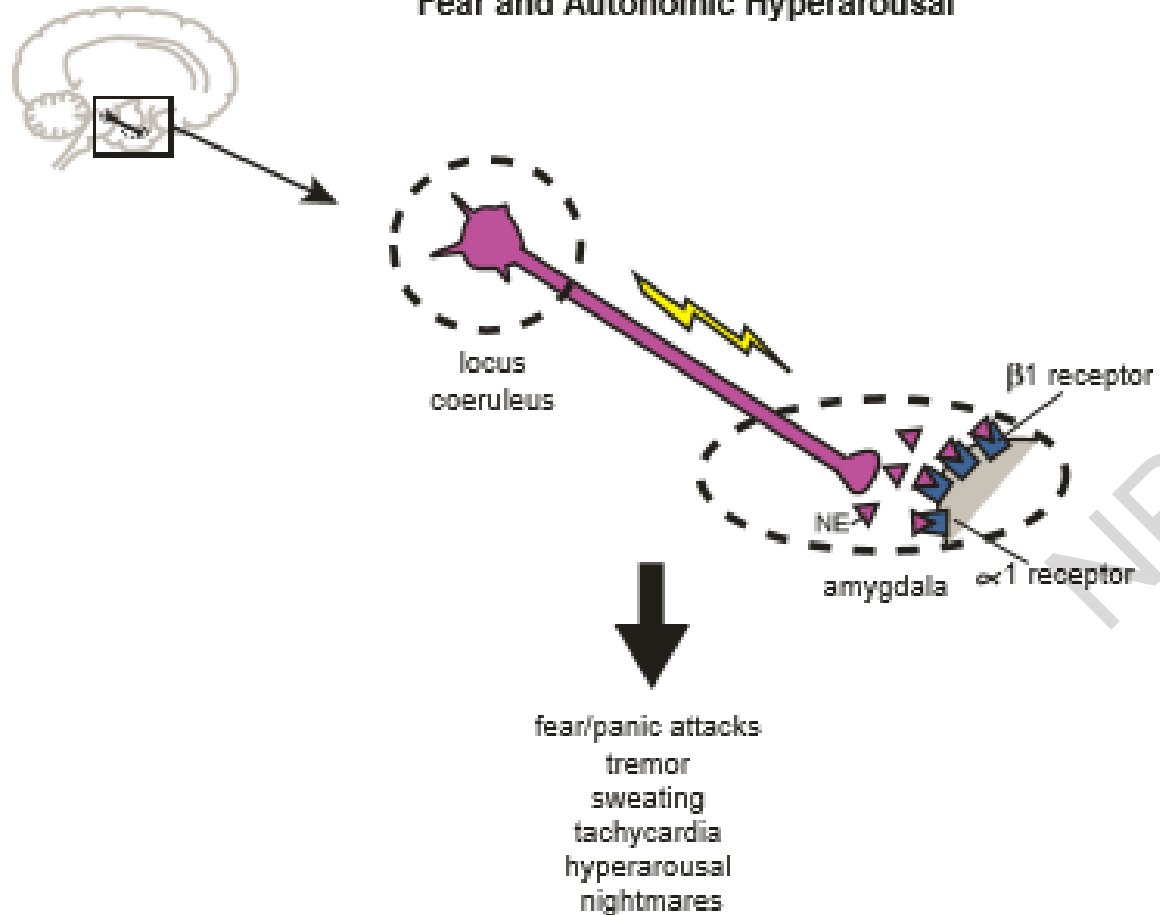
5HT1A Partial Agonist (SPA) Actions in Anxiety



- The amygdala receives input from serotonergic neurons, which can have an inhibitory effect on some of its outputs. Thus, serotonergic agents may alleviate anxiety/fear by enhancing serotonin input to the amygdala.
- A serotonin1A(5HT1A)partial agonist, **buspirone**, a generalized anxiolytic, but not as a treatment for anxiety disorder subtypes. 5HT1A partial agonists as augmenting agents to antidepressants

Noradrenergic hyperactivity in anxiety/fear.

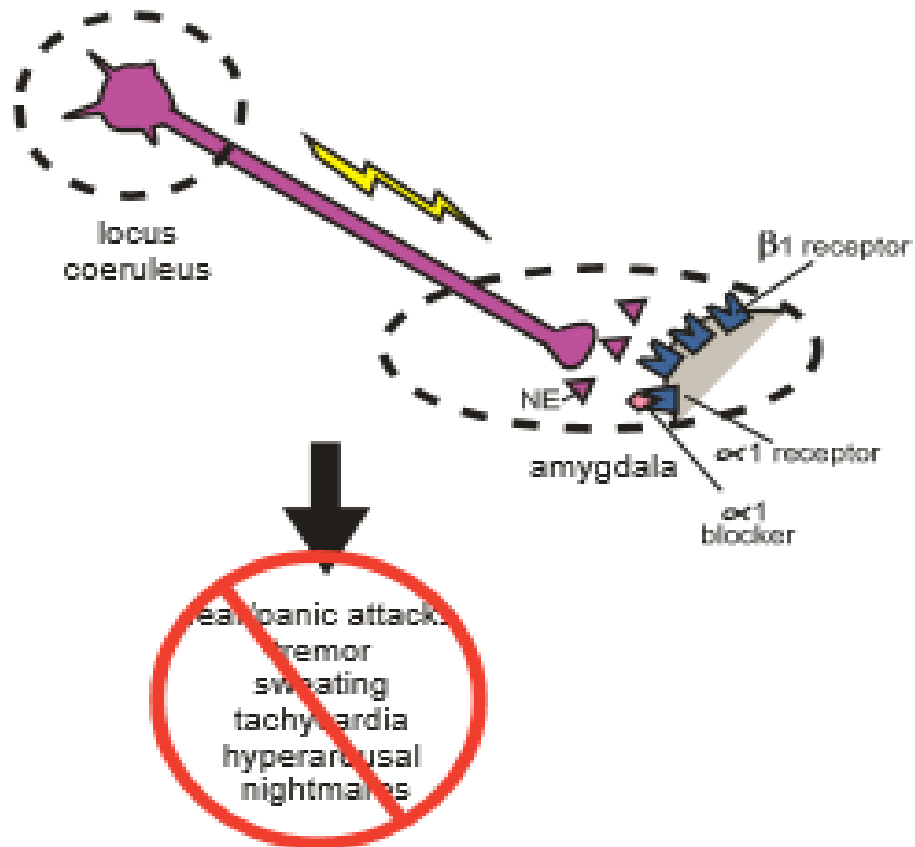
Noradrenergic Hyperactivity in Anxiety,
Fear and Autonomic Hyperarousal



Norepinephrine provides input not only to the amygdala but also to many regions to which the amygdala projects. Noradrenergic hyperactivation can lead to anxiety, panic attacks, tremors, sweating, tachycardia, hyperarousal, and nightmares.

A

Therapeutic Actions of Alpha 1 Antagonists on Nightmares and Hyperarousal



B

Noradrenergic hyperactivity may be blocked by the administration of $\alpha 1$ -adrenergic blockers, which can lead to the alleviation of anxiety and other stress-related symptoms.

Noradrenergic hyperactivity may also be blocked by the administration of a norepinephrine transporter (NET) inhibitor

Keypoints

- Anxiety disorders have core features of fear and worry.
- GABA (γ aminobutyric acid) is a key neurotransmitter in anxiety and the benzodiazepine anxiolytics act upon this neurotransmitter system.
- Serotonin, norepinephrine, $\alpha_2\delta$ ligands for voltage-gated calcium channels, and other regulators of anxiety circuits are also discussed as approaches to the treatment of anxiety disorders.

REFERENCES

1. Stahl's Essential Psychopharmacology, 4e
2. Behavioural Neuroscience, Breedlove, 6e

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**THANK
YOU !**



NPTEL ONLINE CERTIFICATION COURSES

Course Name Basics of Mental Health & Clinical Psychiatry

Faculty Name Dr Sumit Kumar

Department Name Psychiatry

TATA MAIN HOSPITAL

Lecture 40 : Forensic Psychiatry

CONCEPTS COVERED

- **Medical Malpractice**
- **Negligent Prescription Practices**
- **Confidentiality**
- **Privilege**
- **Testamentary & Contractual Capacity**
- **Informed Consent**
- **Criminal Responsibility**
- **Ethic in Psychiatry**

Forensic Psychiatry

Forensic – “Belonging to the court”.

It covers:

- i) Psychiatrists professional , ethical ,legal duties to provide competent care to patients.
- ii) Rights of self – determination to receive and refuse treatment.
- iii) Court decisions
- iv.) Legislative directives
- v) Governmental regulatory agencies vi) Licensure boards
- vii) Evaluation of those charged with crimes to determine culpability viii) Ability to stand trial.
- ix) Ethical codes and practice guidelines of professional- organization and their adherence

Medical Malpractice/ Tort

Negligence means doing something that a physician with a duty to care for the patient should not have done or failing to do something that should have been done.

To prove malpractice

Plaintiff (patient ,family) must establish that:-

- i) Doctor – patient relationship existed
- ii) Deviation from the standard of care existed
- iii) Patient was damaged
- iv) Deviation directly caused the damage.

4 D's

Duty

Deviation

Damage

Direct causation

Each of the 4 elements of a malpractice claim must be present or there can be no finding of liability

It includes :-

Negligent Prescription Practices

- i) Exceeding recommended dosages & failing to adjust the medication level to therapeutic levels.
- ii) Unreasonable mixing of drugs
- iii) Prescribing medication that is not indicated
- iv) Failing to disclose medication effects.
- v) Informed consent to be obtained each time the drug is changed or dose is increased.
- vi) Failure to treat adverse effects that should have been recognized.
- vii) Prescribing addicted drugs to vulnerable patients
- viii) Failure to refer to a specialist if needed.
- ix) Negligent withdrawal of the medication.

Confidentiality (section 23) MHA 2017

Physicians , surgeons , paediatricians , Psychiatrists & the whole of doctor fraternity are bounded by premise of medical ethics to hold secret all information given by patients.

This professional obligation is called Confidentiality.

Confidentiality applies to certain groups & not to others. Such groups include treating team , clinical supervisors and consultants.

Subapneoa/summons:

It can force a psychiatrists to break the pact and breach the confidentiality if courts compel them to testify in order for law to take its course.

Psychiatrist must however obtain an informed consent from the patient before such disclosure(in case of verbal consent formally written and documented)

Permission to be reobtained for subsequent disclosures also.

If a clinician believes that the information may be destructive the matter should be discussed and the release may be refused

Privilege

Privilege is the right to ,maintain secrecy or confidentiality in the face of a subpoena

Privileged communications are statements made by spouse , priest , doctor – patient and the law protects from forced disclosure on the witness stand.

It belongs to the patient.

Physician may claim privilege but this privilege ceases to act in case of military courts.

Testamentary & Contractual Capacity

Psychiatrists are asked to evaluate patients capacity and their competence to make a will.

3 Psychological abilities are required:

- a) Patients must know the nature and extent of their property
- b) the fact that they are making a will .
- c) The identities of their natural beneficiaries (spouse , children and relatives).

Competence is determined on the basis of a persons ability to make a sound judgement ie to weigh, to reason and to make reasonable decisions

Durable Power of attorney

Development proceeds with a permission for persons to make provisions for their own anticipated loss of decision- making capacity is called a **durable power of attorney**.

Document permits the advance selection of a substitute decision maker who can act without the necessity of court proceedings when the signatory becomes incompetent through illness.

Ethics in Psychiatry

Ethical Conflicts- Defined as tension between what one wants to do and what is ethically right to do.

Ethical dilemmas- Conflicts between ethical perspectives or values.

Ethics- deals with the relations between people in different groups and often entail balancing rights.

Professional ethics- derive from a combination of morality , social norms and the parameters of the relationship people have agreed to have.

4 ethical principles :

- i) Respect for Autonomy-
- ii) Beneficence- fiduciary relationship with patients
- iii) Nonmaleficence- Primum non nocere / “first do no harm”
- iv) Justice

Informed Consent

Informed consent is the process in which a health care provider educates a patient about the risks, benefits, and alternatives of a given procedure or intervention.

The patient must be competent to make a voluntary decision about whether to undergo the procedure or intervention.

Basic elements of a Consent Form

- i) Fair explanations of the procedures to be followed and their purposes, including identification of the procedures that are experimental.
- ii) Any discomfort or risks which can be expected.
- iii) Disclosure of any benefits expected.
- iv) disclosure of any appropriate alternatives which may be advantageous to the patient.
- v) An instruction that they are free to withdraw consent and discontinue participation in the project or activity.

Exceptions to Informed Consent:

It includes:

- (1) When the patient is incapacitated
- (2) Life-threatening emergencies with inadequate time to obtain consent.
- (3) Voluntary waived consent.

If the patient's ability to make decisions is questioned or unclear, an evaluation by a psychiatrist to determine competency may be requested.

A situation may arise in which a patient cannot make decisions independently but has not designated a decision-maker. In case there is no availability of decision – maker , a legal guardian is appointed by the court.

Children and Informed Consent:

Children (typically under 17) cannot provide informed consent. So parents must permit treatments or interventions

In this case, it not termed "**informed consent**" but "**informed permission.**"

An exception to this rule is a **legally emancipated child** who may provide informed consent for himself.

- (1) Who are under 18 and married
- (2) Serving in the military
- (3) Able to prove financial independence
- (4) Mothers of children (married or not).

Legislation regarding minors and informed consent is state-based as well. It is important to understand the state laws.

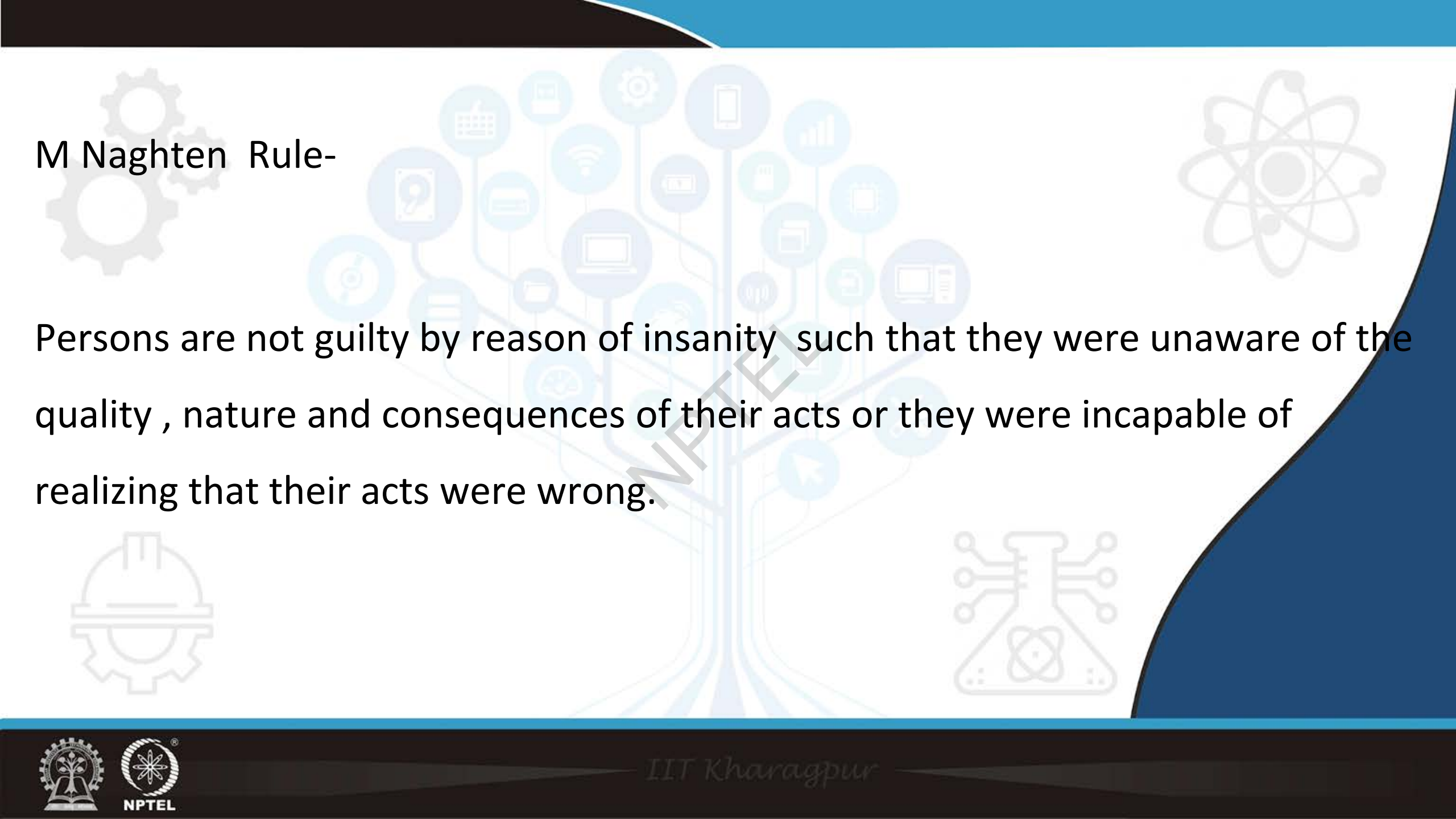
Criminal Responsibility

According to law

Committing an act that is socially harmful is not the sole criterion of whether a crime has been committed

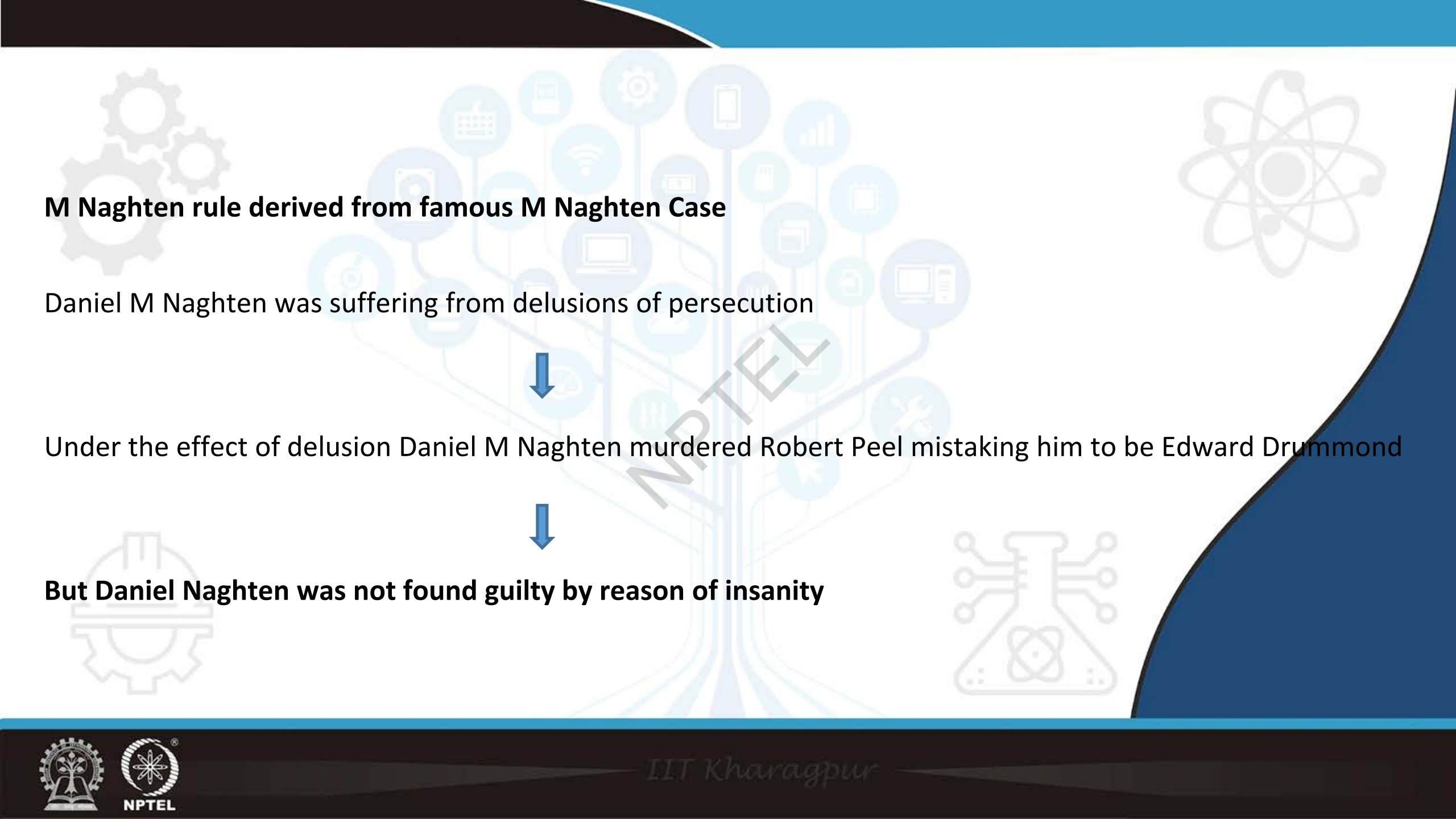
It must have 2 components:

- a) Actus Reus- Voluntary Conduct
- b) Mens Rea- Evil Intent



M Naghten Rule-

Persons are not guilty by reason of insanity such that they were unaware of the quality, nature and consequences of their acts or they were incapable of realizing that their acts were wrong.



M Naghten rule derived from famous M Naghten Case

Daniel M Naghten was suffering from delusions of persecution



Under the effect of delusion Daniel M Naghten murdered Robert Peel mistaking him to be Edward Drummond



But Daniel Naghten was not found guilty by reason of insanity

2 Points in favour of M Naghten

- i) To establish a defence on the ground of insanity, it must be clearly proved that at the time of committing the crime, the **accused was under a defect of reason from disease of the mind**, as not to know the nature and quality of the act he was doing **or if he was doing he did not know it was wrong**.
- ii) The **act committed were real** because he was under the defect of delusion.

Mental Health Act 2017: How it evolved ?

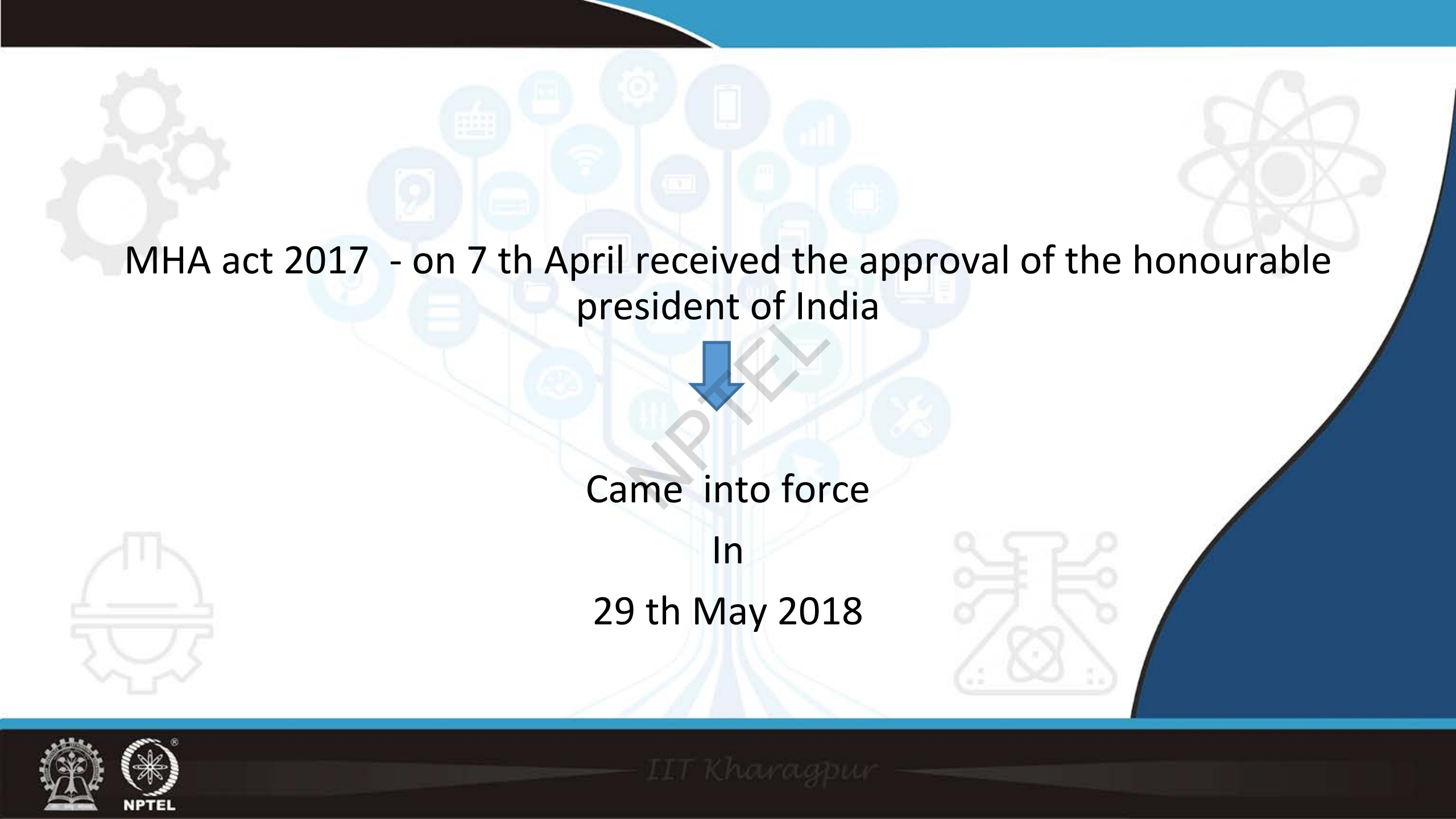
ILA act 1912- Custodial care of persons with mental illness



MHA act 1987- Treatment of persons with mental illness



MHA act 2017 – protect human rights during treatment
aligning with UNCRPD



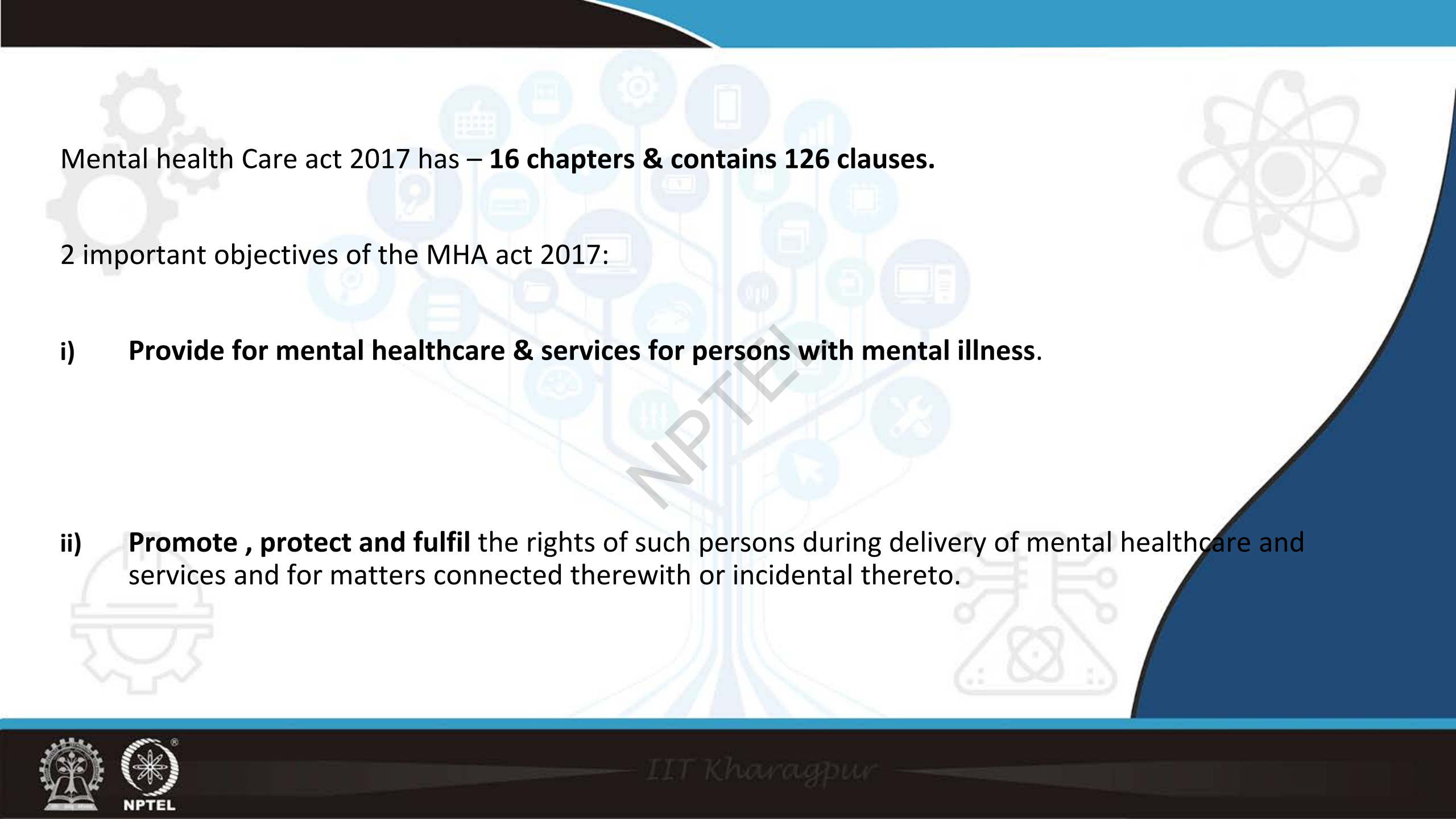
MHA act 2017 - on 7 th April received the approval of the honourable president of India



Came into force

In

29 th May 2018



Mental health Care act 2017 has – **16 chapters & contains 126 clauses.**

2 important objectives of the MHA act 2017:

- i) **Provide for mental healthcare & services for persons with mental illness.**
- ii) **Promote , protect and fulfil** the rights of such persons during delivery of mental healthcare and services and for matters connected therewith or incidental thereto.

(Chapter V)MHA act 2017

I) Mental Health Capacity- sec 4

II) Advance Directive – Sec 5 to 13

III) Nominated Representative- sec 14 to 17

iv)Rights of PWMI – sec 18 to 28

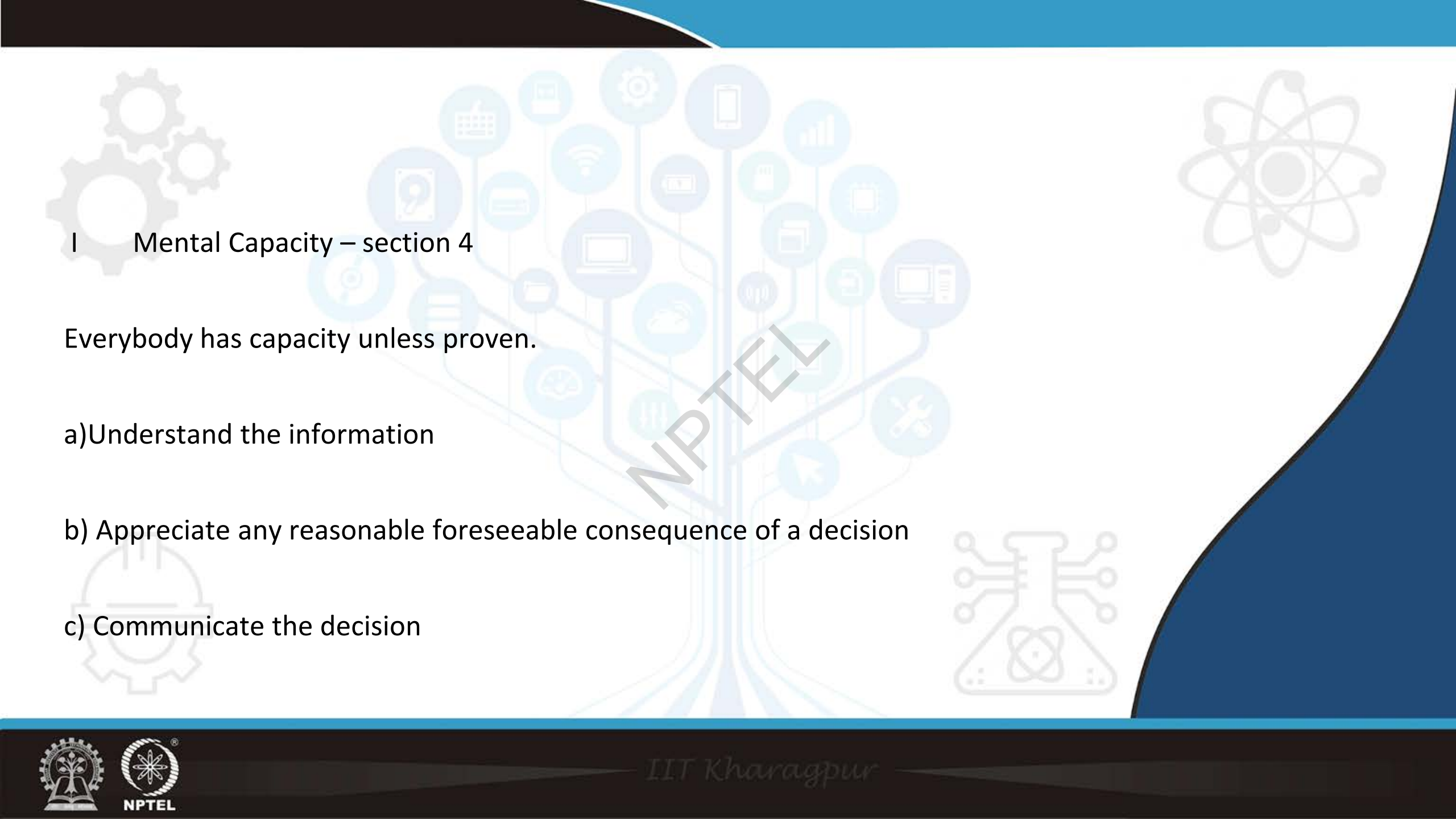
Section 121– Mental health care (Rights of person with mental illness)

Section 121 (1) & (3)- Central Mental authority and Mental health Review Board.

Section 121 (1) & (4)- State Mental Health Authority

Section 81 – Mental Capacity

Section 122- Central Mental Health authority



I Mental Capacity – section 4

Everybody has capacity unless proven.

- a) Understand the information
- b) Appreciate any reasonable foreseeable consequence of a decision
- c) Communicate the decision

Advance Directives – section 5 to 13

Every Adult has the right to :

- i) Wishes to be cared
- ii) Wishes not be cared
- iii) Can appoint Nominated Representative (NR)

Shall be invoked only when he / she ceases to have capacity.

Advance directive is for mental health care , admission or treatment and not any other purpose

Nominated Representative – section 14-17

Every adult has the right to appoint a Nominated representative

NR must be an adult who must follow the rules in accordance with MHA 2017

If there is no NR

i) Relatives - ii) Caregivers iii) Suitable persons appointed by MHRB

iv) Board shall appoint, Director, department of Social Welfare to consider

NGO as appointed by Mental Health Review Board (MHRB)

Rights of Persons With Mental Illness: section 18 to 28

Access to mental health care

- i) All type of services
- ii) Affordable cost, quality , quantity
- iii) Compensatory
- iv) Free treatment for BPL/destitute
- v) Long term care also included

Right to Equality and Non discrimination- section 21

Right to information – section 22

- a) Reason for Admission b) Treatment procedure c) Language

Right to access medical records – section 25

- i) Access to basic medical records not the complete set of records.
- ii) Shall inform the person with mental illness of his right to apply to the concerned board.

Right to community living -section 19

Right to legal aid – section 27

Right to person contact and communication – section 26

Right to make complaints about deficiencies in provision of services- section 28

Implementing Authorities

Central Mental Health Authority- section 33 to 44

State Mental Health Authority – section 45 to 56

Mental Health Review Board – section 73 to 84

Admission of Psychiatric Patients under Mental Health Act 2017

✓ Independent Admission (voluntary) -section 85,86

Adult

Any person who is not minor and who considers himself to have a mental illness and desires to be admitted to any mental health establishment for treatment may request the medical officer or mental health professional to be admitted as an independent patient.

After assessment by the competent mental health professional only can he/she be admitted to the mental health establishment.

85- is the right of the patient

86- he/she is taking treatment from the Mental Health Establishment (MHE)

✓ Independent Admission (voluntary)- section 87

Minor

This is done **with request from Nominated Representative** who can be parents/caregiver or guardian which is done in best interest for the minor

It is only after the assessment by the competent mental health professional that admission can be planned

Involuntary Admission (Supported) – duration -30 days

- a) Section 89- when the patient has threatened or attempted to harm others or self , behaved violently , inability to care for himself/herself etc ,he/she can be **admitted under 89 with request from Nominated Representative (NR)**

(If emergency intervention was done to control the agitation or for behavioural issues –section 94 is implicated during admission)

If duration of stay increases more than 30 days- section 89-> 90

b) Court Admission – Section 102-

When magistrate gives an order that the particular patient is conveyed for assessment or treatment.

Depending upon the assessment patient can be either followed up in the opd or admitted for 72 hrs during which it can be assessed under 85-86 if there is capacity or under section 89 with –nominated representative.

It is the obligation of the duty medical officer to write an assessment report and send a copy to magistrate after completion of the assessment.

c) Admission through Police (Investigating) officer (IO)- Section 100

They are duty bound for the destitute or patients that has threatened or attempted to harm others or self , behaved violently , inability to care for himself/herself and request the duty medical officer at the mental health establishment in charge for assessment and or treatment depending upon the condition.

- i) **Request from the (IO) is done under 100 (3)**
- ii) **Assessment by the Menta Health Professional/Psychiatrist is done under 100(5)**
- iii) **Discharge /follow up is done under 100 (6) when on assessment it is found that the patient can either treated on out patient basis (opd) or there is no psychiatric intervention required , officer in charge can send the patient to shelter home or patients residence .**

Prohibitions & Restrictions

a) Psychosurgery only after consent & board clearance.

b) Restraints

i) If it is the only way to prevent imminent and immediate harm to self or other.

c) Unmodified ECT- Totally prohibited

d) Sterilization – Totally prohibited

e) Chaining /seclusion/solitary confinement-Prohibited

f) ECT is prohibited in minors (except with prior clearance from the board)



Section 115 De-criminalise suicide

Section 115 of the MHA act 2017 provides de- criminalization of suicide attempts (section 309 IPC).

A person who attempts suicide shall be suffering from stress at the time and will not be punished under the Indian penal code.

National Mental health Programme

The adoption of National Mental Health Programme (NMHP) by the Government of India in August 1982, was in many ways a landmark event in the history of psychiatry in this country.

Early contributions in the field of Mental health

Efforts at developing mental health care as part of the general health care, started with the **Bhore committee-**

a) Public Health b) Medical Relief c) Professional Education d) Medical research e) Industrial health

Under the leadership of Dr. Sushila Nayar, in the 1960's as part of the **Mudaliar committee**

a) Mental health units b) School mental health program c) Training of health personnel (public mental health education)

The **Srivastava committee** which was a precursor to the village level community health worker program included

Mental health at the most peripheral level of health care in the country

Under the leadership of Director , Mental Health Division ,World Health Organization (WHO) (Dr. Norman Sartorius) the idea of developing National Programmes of Mental Health started.



It was in 1974 at Addis Ababa which marked an important expression of the W.H.O about its priority for mental health care in developing countries.



This was followed by the launch of the seven country (Brazil, Colombia, Egypt, India, Philippines, Senegal, Sudan), project “Strategies for Extending Mental Health Care (1975–1981),” to implement the 1974 recommendations at the level of general health care.



A formal resolution, urging all member countries to develop NMHP was 1st time adopted in 1979 meeting of the W.H.O. Mental Health Advisory Group in Manila, Philippines.

India was the first major country to adopt it at the national level.

How it started??

A small working group was constituted in 1980 for preparation of the draft among those were :

Dr. Bisht, Director General of Health Services

Dr. Wig (New Delhi)

Dr. Sethi (Lucknow)

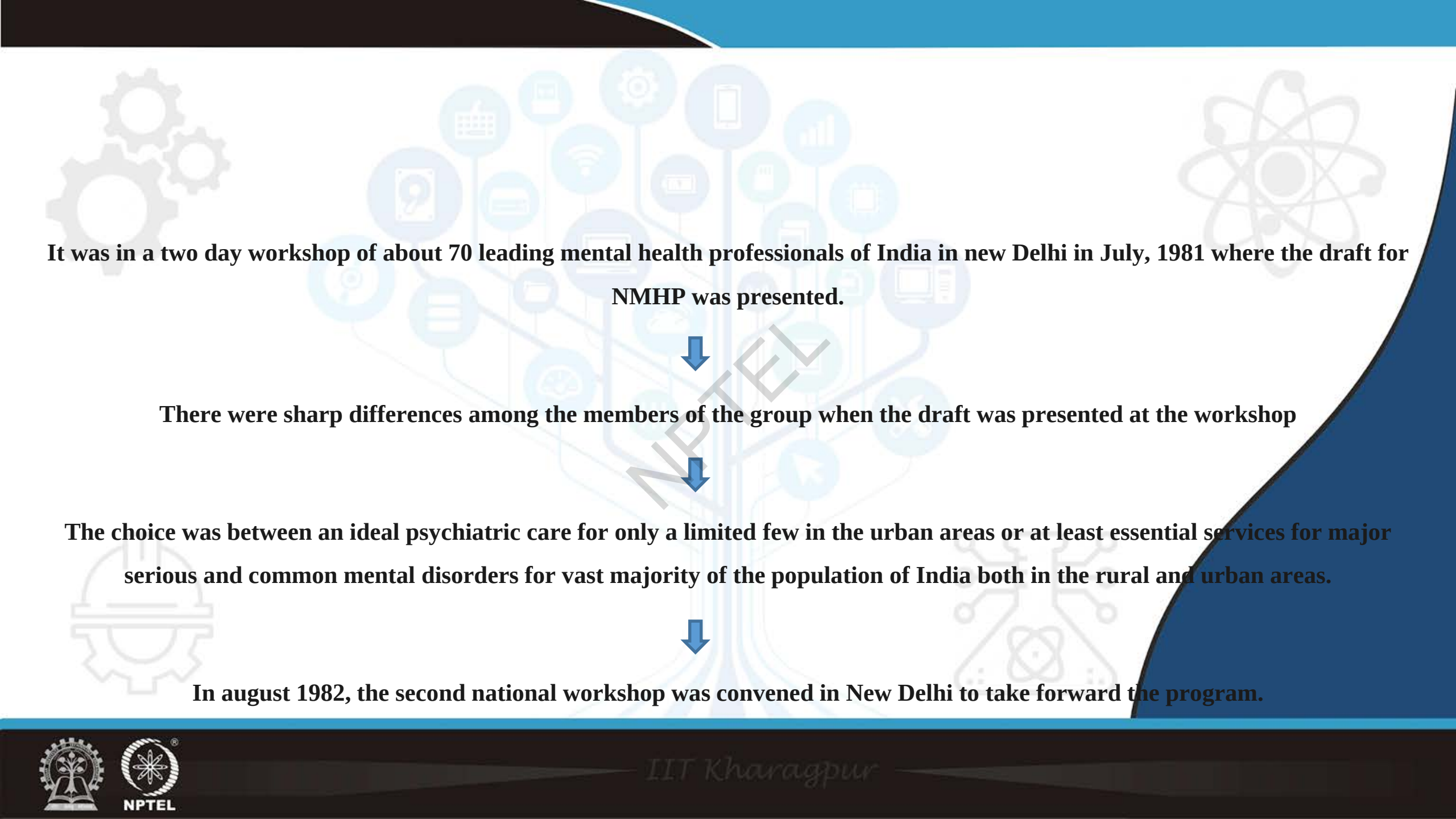
Dr. Venkoba Rao (Madurai)

Dr. Sharma (Ranchi)

Dr. Kapur (Bengaluru)

Dr. Helmut sell (W.H.O., SEARO)





It was in a two day workshop of about 70 leading mental health professionals of India in new Delhi in July, 1981 where the draft for NMHP was presented.

There were sharp differences among the members of the group when the draft was presented at the workshop

The choice was between an ideal psychiatric care for only a limited few in the urban areas or at least essential services for major serious and common mental disorders for vast majority of the population of India both in the rural and urban areas.

In august 1982, the second national workshop was convened in New Delhi to take forward the program.

Widely representative group of experts ever assembled in the country to consider the mental health issues and approve the draft of the NMHP

- a) Representative of Directorate of Health Service
- b) Indian Council of Medical Research
- c) Indian Medical Council, W.H.O ,SEARO
- d) National Institute of Mental Health and Neuro Sciences Bengaluru
- e) Indian Medical Association

Leading Clinical Psychologists, Sociologists and representative from Ministries of Health, Social Welfare, Labor, Planning Commission.

Agreed by leading mental health specialists 1-year earlier in July, 1981.

Significance of the NMHP with its 3 broad objectives

- **To ensure availability and accessibility of minimum mental health care for all in the foreseeable future, particularly to the most vulnerable and underprivileged of the population;**
- **To encourage application of mental health knowledge in general health care and in social development; and**
- **To promote community participation in mental health services development and to stimulate effort toward self-help in the community.**

Inception of District Mental Health Programme (DMHP)

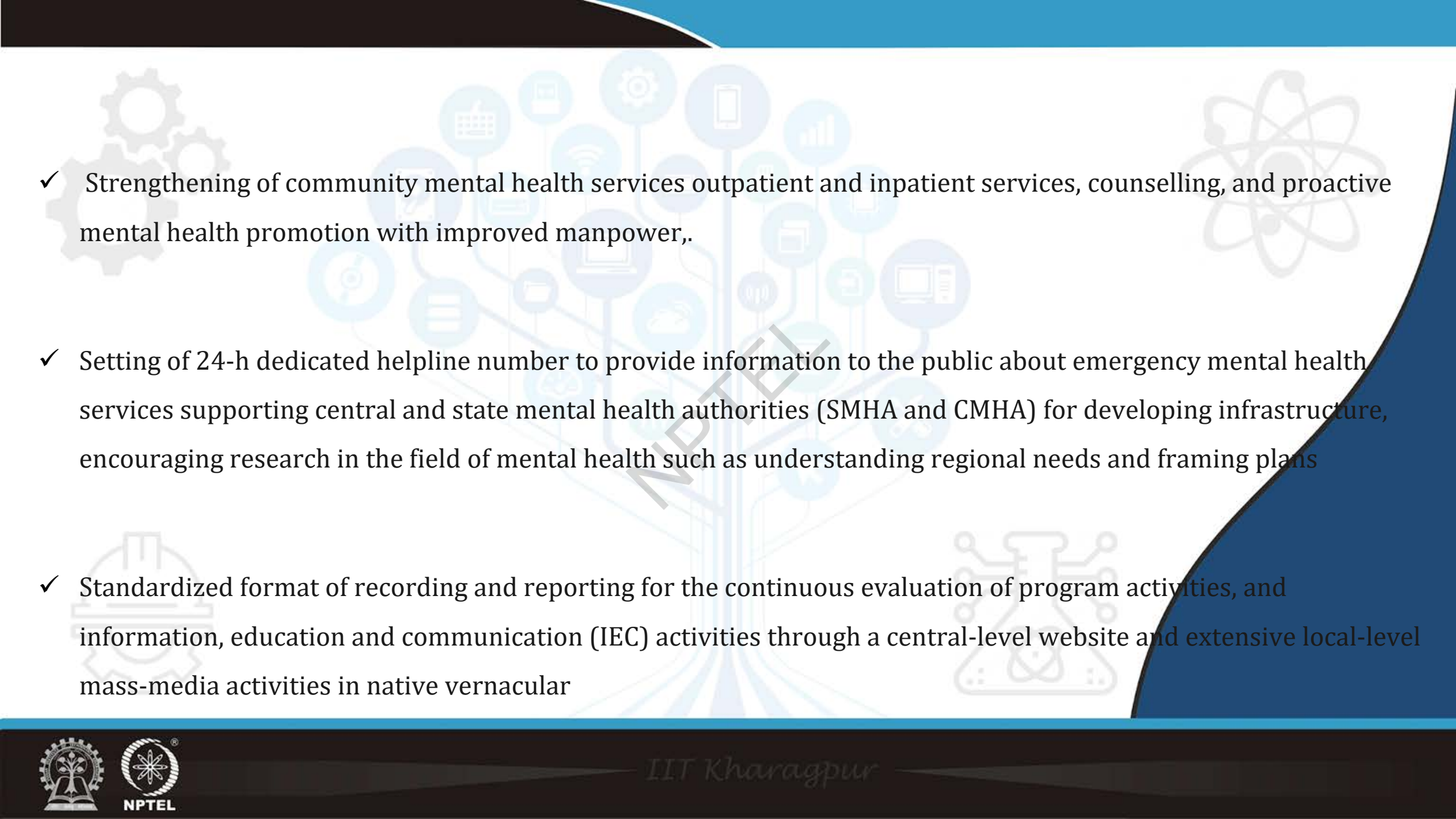
To overcome the limitations of NMHP and to scale it up

It was perceived that the **district should be the administrative and implementation unit of the program**

The National Institute of Mental Health and Neurosciences (NIMHANS) undertook a pilot project (1985–1990) at the Bellary District of Karnataka to assess the feasibility of DMHP which proved beneficial.

Efforts have been made to achieve these objectives by extending services to the community by strengthening outreach services

- ✓ Satellite clinics
- ✓ School counselling
- ✓ Workplace stress management
- ✓ Suicide prevention organizing awareness camps in the community through local bodies
- ✓ Improving community participation (by linkage with self-help and caregiver groups)
- ✓ Public-Private Partnership (PPP) with designated financial assistance for establishing daycare and long-term residential care facilities.

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- ✓ Strengthening of community mental health services outpatient and inpatient services, counselling, and proactive mental health promotion with improved manpower,.
 - ✓ Setting of 24-h dedicated helpline number to provide information to the public about emergency mental health services supporting central and state mental health authorities (SMHA and CMHA) for developing infrastructure, encouraging research in the field of mental health such as understanding regional needs and framing plans
 - ✓ Standardized format of recording and reporting for the continuous evaluation of program activities, and information, education and communication (IEC) activities through a central-level website and extensive local-level mass-media activities in native vernacular

Objectives Of District Mental Health programme (DMHP)

- ✓ To develop and implement a decentralized training program in mental health for all categories of health personnel in a way that would be the least disruptive to on-going general healthcare activities
- ✓ To provide a range of essential drugs such as antipsychotics, antidepressants, anticonvulsants, and minor tranquilizers for the management of FWM
- ✓ To develop a system of simple recording and reporting of care by mental health personnel
- ✓ To monitor the effect of service of the mental health program in terms of treatment utilization and outcomes
- ✓ To reduce the stigma by bringing about a change of attitude through public health education
- ✓ Treatment and rehabilitation of patients within the community by adequate provision of medicines and strengthening the family support system.

CONCLUSIONS

- In this lecture have discussed regarding the concepts of Medical malpractice , negligent prescription practices, confidentiality, privilege , criminal responsibility , informed consent , Mental health act 2017 and its various implications.
- National Mental health programme & district Mental health programme in brief

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**THANK
YOU !**