



NPTEL ONLINE CERTIFICATION COURSES

Course Name Basics of Mental Health & Clinical Psychiatry
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Lecture 26 : SUBSTANCE -I

CONCEPTS COVERED

- Concept of Addiction .
- Criteria for Dependence according to ICD
- Concepts of Opioids dependence and its treatment.
- Concepts of Alcohol Dependence and its treatment
- Concepts of Benzodiazepine dependence and its treatment

Substance - I

- Addiction has always been intriguing and fascinating domain of Psychiatry.
- It poses questions :???
- HOW DO PEOPLE GET PLEASURE OUT OF DOING SOMETHING ??
- WHICH IN DUE COURSE OF TIME AND THROUGH VICIOUS CYCLE DEVELOPS INTO A HABIT (CONDITIONING)??
- ULTIMATELY GETS INTO ADDICTION???
- BY GOING THROUGH A PROCESS??

Drug Preoccupation → Craving → ADDICTION → DEPENDANCE → WITHDRAWAL → REPLAPSE

There can be various ways which people seek some of their special rewarding activities to get pleasure out of it like

a) Pizza b) Ice cream c) Gambling d) sports e) Shopping f) Drugs

Drugs what makes them so special??

Answer is quality of Dopamine impulses in Natural activities like sports , pizza, ice cream etc with respect to intake of drugs and behavioural addiction like internet addiction

- Naturally rewarding activities, such as achieving major accomplishments or enjoying a good meal, can cause fast and large increases in DA in the mesolimbic pathway.
- Unlike a natural high, the activation caused by drugs of abuse can eventually cause changes in reward circuitry that are associated with a vicious cycle of



Dopamine is a final common pathway of reward, many other neurotransmitters are involved as in opioid system, which mediates hedonic evaluation of natural rewards.

It mediates the rewards caused by

i) **Alcohol** consumption ii) Reward associated with **nicotine** iii) Reward associated with **cannabinoids**.

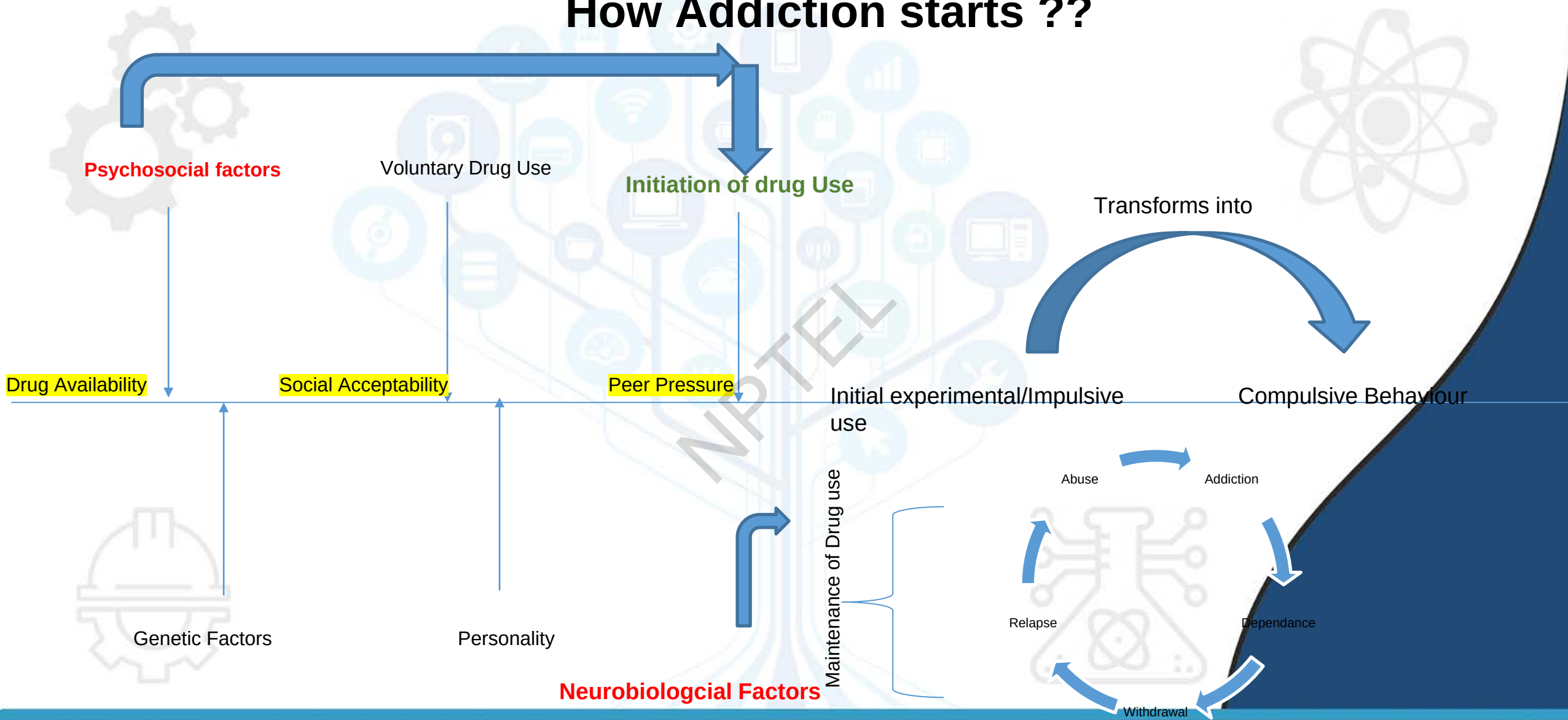
Acute drug use causes DA release in the striatum.

Pleasure will be continued/ will be inhibited

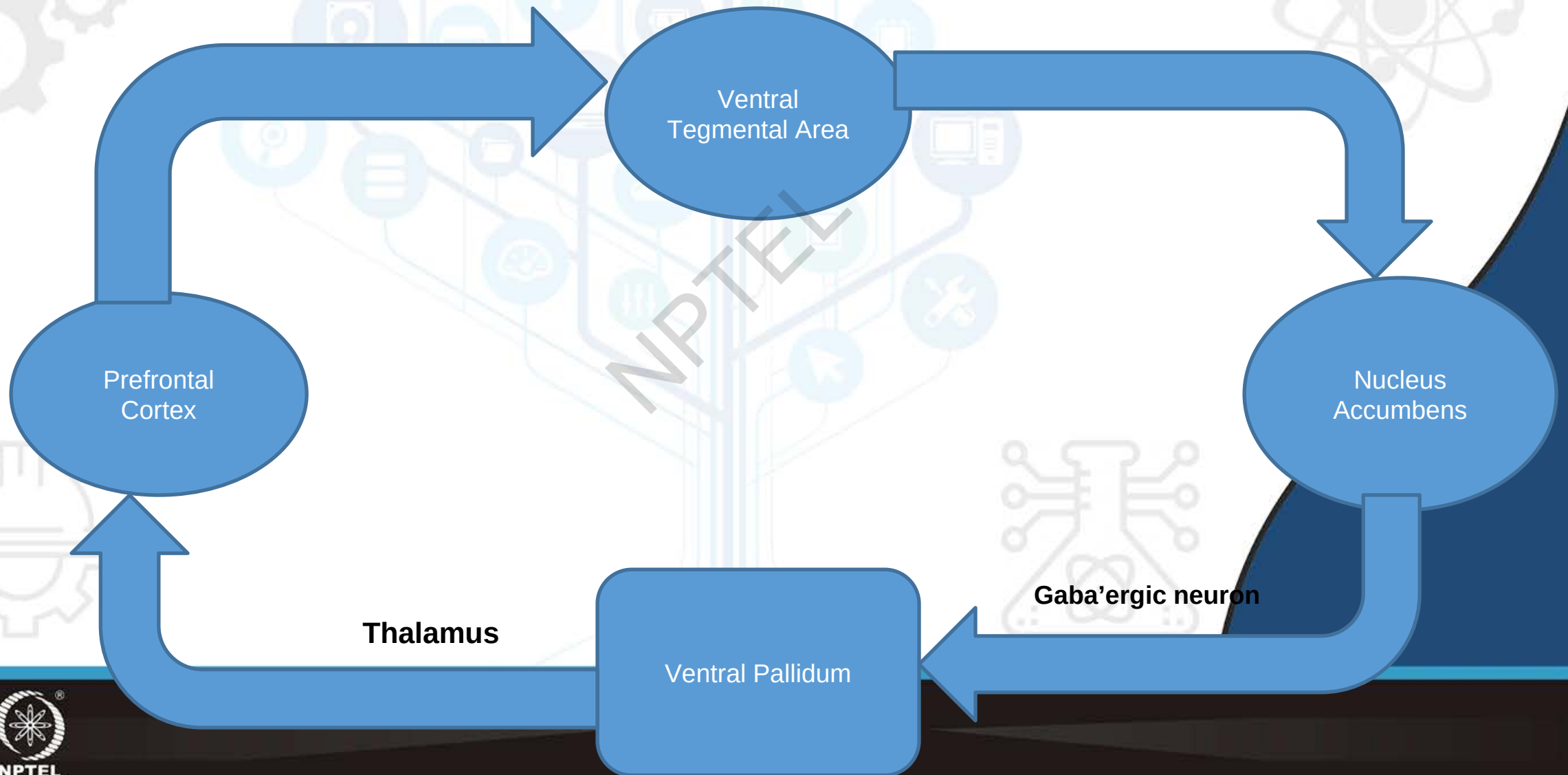
- The reactive reward system consists of the VTA, which is the site of DA cell bodies; the nucleus accumbens, where DA neurons project; and the amygdala, which connects to both the VTA and the nucleus accumbens.
- Rewarding input to the nucleus accumbens is due to bursts of DA release (i.e., phasic DA firing).
- Connections of dopaminergic neurons to the amygdala are involved in reward learning (such as the memory of pleasure associated with drug abuse),
- Connections of the amygdala back to the VTA communicate whether anything relevant to a previously experienced pleasure has been detected.
- Connections of the amygdala to the nucleus accumbens communicate that emotions have been triggered by internal or external cues and signal an impulsive, almost reflexive response to be taken.

- Stimulatory input from the “bottom up” reactive reward system is regulated by the “top down” reflective reward system, which consists of projections from the prefrontal cortex to the nucleus accumbens.
- Projections from the orbitofrontal cortex (OFC) are involved in regulating impulses, projections from the dorsolateral prefrontal cortex (DLPFC) are involved in analyzing situations and regulating whether an action takes place
- Projections from the ventromedial prefrontal cortex (VMPFC) are involved in regulating emotions.
- Insula is reciprocally connected to the amygdala, the VMPFC, and the ventral striatum and is necessary (along with the VMPFC) for emotional decision making.
- When all the inputs are integrated, the final output is either to stop the action that the reactive reward system is triggering (e.g., drug seeking) or to let it happen.

How Addiction starts ??



What makes reward to convert into goal directed behaviour



Conditioning occurs with repeated drug use

Increased Dopamine in Dorsal striatum

[Associated with Habitual Learning]



Pleasure



Amygdala remembers that rewarding experiences

[memory]

In Addicted individuals exposure to drug & drug cues causes



Increased metabolism in Ventral Anterior Cingulate Cortex



Orbitofrontal cortex (Impulsivity)



Dorsal Striatum



Craving ++



Striatum



Ventral

Dorsal

Initial Experimental/Impulsive use



Preference for Immediate Reward



Transforms into Compulsive behaviour

Loss of Control of drugs is due to

Dopamine Reactive reward Pathway & goal directed behaviour

+

Dysfunction in function with glutamatergic projections from prefrontal cortex to VTA & NA

Dependence

According to ICD

Dependence requires at least 3 out of following list satisfied in last 12 months:

1. strong desire or compulsion to take the substance
2. Difficulty in controlling the onset, termination or levels of use.
3. Experiencing withdrawal symptoms if substance not taken
4. Use of substance to relieve from withdrawal symptoms
5. Tolerance as evidenced by the need to escalate dose over time to achieve same effect
6. Salience – neglecting alternate forms of leisure or pleasure in life

*** The narrowing personal repertoire of substance use.**

Important Terms related to Substance use Disorders

- Misuse- Applies to drugs prescribed by physicians that are not used properly.
- Abuse- Use of any drug , but usually by self administration, in a manner that deviates from approved social or medical patterns(for Non medical purposes).
- Addiction- A behavioural pattern of drug abuse characterized by overwhelming involvement with the use of drug (Compulsive use), securing of its supplies and a high tendency to relapse after discontinuation.

• Behavioural Dependence

Consequences
Of
Behaviour

Physiological Dependence

Withdrawal

Psychological dependence

Habituation

Opioids

- Obtained from Papaver Somniferum.
- Opiates are natural
- Opioids are synthetic, examples include meperidine , methadone, pentazocine propoxyphene, morphine, heroin
- M: F – 3:1
- Method of administration

Smoking , Snorting , orally , Intravenous, Intramuscular , Subcutaneous.

Effects of Opioids are due to opioid receptors, namely 3

1. Mu

Analgesia, constipation, respiratory depression , sedation

2. Kappa

Analgesia, diuresis, sedation

3. Delta

Analgesia

- 3 Classes of endogenous opioids within the brain : **Endorphins , Dynorphins, Enkephalins**

Rewarding properties of various drugs like Alcohol , cannabis and opioids are mediated through endogenous opioids.

Clinical features

Feeling of warmth , dry mouth itchy face (nose),facial flushing.

Initial euphoria is followed by sedation.

For 1st timers nausea , vomiting, dysphoria

Intoxication

Unresponsiveness
Coma Hypothermia
Hypotension
Slow respiration

Coma , Pinpoint pupils , respiratory depression triad---
-- Opioid over dose

Physical effects

Respiratory depression , pupillary constriction ,
smooth muscle contraction, constipation,
changes in BP and heart rate.

Withdrawal

Yawning
Pupillary dilatation
Lacrimation, rhinorrhoea
Dysphoric mood
Muscle aches Diarrhoea
Insomnia Hypothermia

- Opioids are drugs such as heroin, opium, morphine, codeine and methadone. Opioid withdrawal can be very uncomfortable and difficult for the patient
- Short-acting opioids (e.g. heroin): Onset of opioid withdrawal symptoms **8-24 hours after last use**; duration 4-10 days.
- Long-acting opioids (e.g. methadone): Onset of opioid withdrawal symptoms **12-48 hours after last use**; duration 10-20 days.

Management Plan:

COWS (Clinical Opiate Withdrawal Scale)

Mild withdrawal; symptomatic medication only

0-10

Moderate withdrawal; symptomatic or opioid medication

10-20

Severe withdrawal; opioid medication

20-30

Mild Cases

Patients should drink at least 2-3 litres of water per day during withdrawal to replace fluids lost through perspiration and diarrhoea. Also provide vitamin B and vitamin C supplements.

Symptomatic treatment for insomnia, abdominal cramps, diarrhoea, muscle cramps and supportive care are usually sufficient for management of mild opioid withdrawal.

Moderate Cases

As for management of mild withdrawal, but with the addition of clonidine or opioid medications such as buprenorphine, methadone.

For Severe cases

Clonidine

Alpha-2 adrenergic agonist.

Relief to physical symptoms of opioid withdrawal including sweating, diarrhoea, vomiting, abdominal cramps, chills, anxiety, insomnia, and tremor.

It can also cause drowsiness, dizziness and low blood pressure.

Buprenorphine

It alleviates **withdrawal symptoms and reduces cravings.**

Its pharmacological action (partial opiate agonist), Buprenorphine should only be given after the patient begins to experience withdrawal symptoms (i.e. at least eight hours after last taking heroin).

CNS depressants- Alcohol , Barbiturates, Benzodiazepines , Inhalants
CNS Stimulants- Cocaine, Amphetamines,

Alcohol withdrawal

Simple withdrawal

Tremulousness develops	6-8 hrs
Psychotic and perceptual disturbances	8- 12 hrs
Seizures	12-24 hr
Delirium Tremens	any time within 1 st week

Black outs:

Similar to transient global amnesia.

Discrete episodes of anterograde amnesia that occur in association with alcohol intoxication.

Specific short term memory deficit , intact remote memory but unable to recall events that occurred in last 5 – 10 mins.

Neurobiological mechanism-Alcohol blocks consolidation of new memories into old

Involves Hippocampus and Temporal lobe

Delirium tremens

- Delirium is characterized by a rapid onset and fluctuating course with disturbances in the level of consciousness, cognition, psychomotor activity, and sleep-wake cycle.
- **Thomas Sutton 1813 coined** the term “**Delirium Tremens**”
- Inattention, disorientation, motor agitation, alteration of sleep wake cycle and disturbance in short term memory as common presentations of DT. Severe form of alcohol withdrawal
- DT is a short lasting condition with a usual duration of 3–4 days (but might last for even 8 days) and it classically ends with a prolonged sleep.

Characteristic Features

Prevalence of < 1 % in general population
Nearly 2 % in Alcohol Dependence.

Coarse tremors of hands

Insomnia Nausea Vomiting Psychomotor agitation Anxiety Transient Visual

Tactile or Auditory Hallucinations , along with increase in sweating, body temperature, heart rate, respiratory rate and blood pressure

Treatment

Benzodiazepines , Antipsychotics , Thiamine & Vitamins

Wernickes encephalopathy

Short term complications of alcohol

Triad – Gait Ataxia, horizontal Nystagmus and Lateral Gaze Palsy.

It includes Sluggish eye reaction to light, Anisocoria

May progress into Korsakoff's Syndrome or spontaneously clear in a few days.

Treatment

100 mg Thiamine 2-3 times daily for 1-2 weeks. Thiamine should be given before any i.v. glucose. As glucose infusion exacerbates thiamine deficiency biochemically.

Ophthalmoplegia responds within hours of treatment

Korsakoff'Syndrome

Impaired mental syndrome (recent memory) + anterograde amnesia

With or Without confabulation.

Treatment

100 mg 2- 3 times daily for 2-3 months

Rum Fits

Stereotyped

Generalised

Toni-clonic

Associated with alcohol withdrawal

Rx Benzodiazepines

Long term complications

Marchiafava Bignaminis.

- a) Generalised pyramidal tract lesion
- b) focal cerebellar tract lesions.

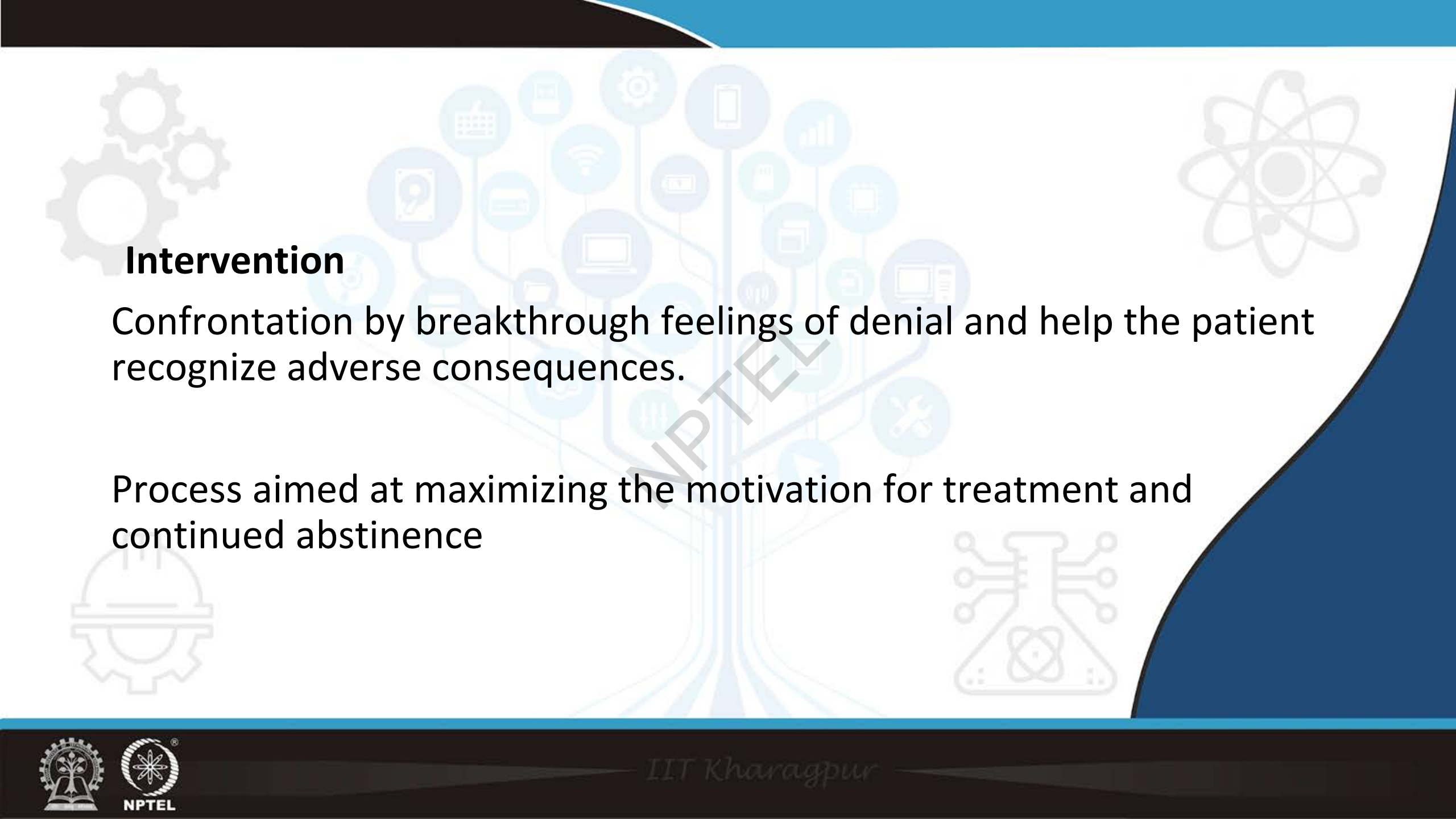
Carducci first described it in 1898 in Italian red wine drinkers.

Demyelination of Corpus Callosum and adjacent sub cortical white matter.

Dementia, dysarthria , spasticity , gait changes

Treatment

- Intervention
- Detoxification & Deaddiction
- Rehabilitation



Intervention

Confrontation by breakthrough feelings of denial and help the patient recognize adverse consequences.

Process aimed at maximizing the motivation for treatment and continued abstinence

Detoxification

- If the patient is in good health and have adequate nourishment with good social support the withdrawal is managed mildly and with ease.
- Lasts 1week to 10 days
- Include thorough physical examination
- Vitamins (thiamine), proper rest and sleep

Naltrexone

Blocks opioid receptor **resulting in reduced craving and reduced reward.**

Nausea, vomiting, abdominal pain, constipation, dizziness, headache, anxiety

Serious hepatotoxicity

Contraindicated in acute withdrawal of Opioid

Acamprosate

Acamprosate is a derivative of the amino acid taurine, and reduces excitatory glutamate neurotransmission and enhances inhibitory GABA neurotransmission.

It blocks mGlu receptors and also NMDA receptors.

When alcohol is used chronically and then withdrawn, the adaptive changes causes glutamate hyperactivity—even excitotoxicity—and GABA deficiency.

By blocking glutamate receptors, acamprosate may mitigate glutamate hyperexcitability during alcohol withdrawal and is therefore sometimes called “artificial alcohol.”

Used for patients who have attained abstinence

Disulfiram

Antabuse

Approved for maintenance of Abstinence

Disulfiram is an irreversible inhibitor of aldehyde dehydrogenase, thereby blocking this second-stage metabolism

When alcohol is consumed by a patient taking disulfiram

toxic levels of acetaldehyde build up

which leads to flushing, tachycardia, nausea, vomiting, and other symptoms

This aversive experience ideally leads to negative conditioning in which patients abstain from alcohol in order to avoid the unpleasant effects.

Alcohol

↓
Acetyldehyde

↓
AsceticAcid

Mild –Moderate Withdrawal

It occurs due to habituated depressant in the brain and its sudden stoppage or abrupt reduction of dosage.

Any Depressants Benzodiazepines ,Barbiturates given in an tapering dosage (preferably Lorazepam if liver is compromised, or Chlordiazepoxide).

Dosage of the depressant to be tapered by 20 % reduction.

For Short acting drugs like lorazepam careful dose titration required as compared to long acting (diazepam/chlordiazepoxide).

Severe Withdrawal

Medical Problems needs to be ruled out

Adequate dosage for Benzodiazepines

Antipsychotics for Hallucinatory experiences (preferably Haloperidol)

Neurological Evaluation if patient does not respond with seizures.

Rehabilitation

Maintain high level of motivation for abstinence

Lifestyle modification , Relapse Prevention strategies , Cognitive behavioural therapy

Benzodiazepines

Short-acting benzodiazepines include oxazepam, alprazolam and temazepam.

Withdrawal typically **begins 1-2 days** after the last dose, and continues for 2-4 weeks or longer.

Long-acting benzodiazepines include diazepam and nitrazepam.

Withdrawal typically **begins 2-7 days** after the last dose, and continues for 2-8 weeks or longer

Symptoms include

Anxiety

Insomnia

Restlessness

Agitation and irritability

Poor concentration and memory

Muscle tension and aches.

The longer the interval between reductions, the more comfortable and safer the withdrawal.

Generally, there should be at least one week between dose reductions.

Signs & Symptoms of Benzodiazepines discontinuation

- Return of original anxiety symptoms (Recurrence)
- Worsening of original anxiety symptoms (Rebound)
- Emergence Of New symptoms (Withdrawal)

Disturbances Of Mood and Cognition

Anxiety, Apprehension , dysphoria ,pessimism , Irritability

Disturbances of Sleep

Insomnia, altered sleep-wake cycle and daytime drowsiness.

Physical Signs and symptoms

Tachycardia, elevated blood pressure , hyperreflexia , muscle tension , agitation , muscle ,joint pain , grand mal seizures.

Perceptual Disturbances

Hyperacusis , depersonalisation , blurred vision , illusions and hallucinations

Signs of Benzodiazepines Intoxication

- Incoordination
- Dysarthria
- Nystagmus
- Impaired memory
- Gait disturbances.
- In severe cases stupor coma and death occurs

Guidelines for Benzodiazepines Withdrawal

- Evaluate and treat medical and psychiatric condition
- Obtain drug history and urine and blood samples.
- To determine the dose required for stabilisation from history obtained, clinical presentation.
[Most symptoms occur on first 3 days of abstinence and seizure on 2nd or 3rd day]
- Hospitalise if there is poor social support, polysubstance dependence and /or medical problems.
- Long acting benzodiazepines preferred.
- After stabilizing reduce dosage by 30 % on second and third day
- Tapering of the dose should be done depending upon the symptoms present by counteracting them
- Psychological intervention assist patients in managing related anxiety.

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CONCLUSIONS

-In this lecture we have discussed regarding the concept of addiction , how it starts ,and evolves into a disease process ,there is loss of control over drugs

Diagnosis of Substance Dependence depending upon criteria as per International Classification of diseases

Concepts of opioids dependence and its treatment.

Concepts of Alcohol dependence and its treatment.

Concepts of Benzodiazepine Dependence and its treatment



**THANK
YOU !**



NPTEL ONLINE CERTIFICATION COURSES

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Lecture 27 : Substance II

Addiction - II

CONCEPTS COVERED

- Concepts regarding Cannabis dependence and its treatment
- Concepts regarding Hallucinogen Dependence & its treatment
- Concepts regarding Inhalant dependence and its treatment
- Concepts regarding tobacco dependence and its treatment
- Concepts of Gambling disorder and its treatment
- Concept of Behavioural Addiction (Internet addiction)

Hallucinogens

Aka Psychedelics or Psychotomimetics / intoxicants.

Have no medical use and a high abuse potential as per US-FDA

Naturally occurring hallucinogens are

Psilocybins (mushrooms), Mescaline (peyote Cactus), Ibogaine , DMT (dimethyltryptamine).

Synthetic Hallucinogen- Lysergic Acid Diethylamide (LSD).

Effects of Hallucinogens usually follows a pattern

Somatic symptoms---→Mood Symptoms-→ Perceptual changes-→Psychological changes (symptoms can overlap also) depending upon type of hallucinogen used.

Physiological /sympathomimetic effects include

1. Tremors
2. Tachycardia
3. Hypertension
4. Hyperthermia
5. Sweating
6. Blurring of vision
7. Mydriasis.

3 Conditions associated are MDD, Panic Disorders , Alcohol Dependence



[Hallucinogen use Persisting disorder-With hallucinogens use colours and textures become richer, contours sharpened , music more emotionally profound and smell and taste heightened. Two seemingly incompatible feelings will be felt at the same time .]

Treatment

1. Minimize sensory input as it reduces the psychotics experiences (hallucinatory phenomenon related with the drug).
2. Patient is kept in the environment where it is quiet as possible.
3. Antipsychotics with benzodiazepines occasionally to counter act agitation & aggression.
4. Restraints to be avoided as it can give rise to rhabdomyolysis.

Inhalant substances

- Aka Volatile substances or solvents
- Vaporize to gaseous fumes at room temperature.
- Easily available ,legal, inexpensive.

Males> females

- Mode of intake mouth/nose through transpulmonary route causing rapid absorption.
- Breathing solvent soaked cloth/ huffing vapour sprayed into plastic bag.
- Effects appear within 5 mins & lasts 30 mins to several hours.
- Tolerance develops but withdrawal symptoms are mild, detectable in blood 4 -10 hrs after use .
- Works by membrane Fluidization same as alcohol.
- 4 commercial classes: a) Glues & Adhesives b)Propellants C) Thinners d)Fuels.

Clinical features

Feeling of disinhibition , euphoria and pleasant floating sensations.

High dose can cause fearfulness, sensory illusions, auditory & visual hallucinations

Computerised Tomography of chronic inhalants show diffuse brain stem , cerebellar, cerebral atrophy with white matter leukoencephalopathy.

Functional Imaging shows both decreased/increased blood flow in cerebral areas.

Long term complications include emotional lability and impaired memory, lead encephalopathy

Neurological symptoms in the form of slurred speech , ataxia, peripheral neuropathy paraesthesia, parkinsonism, poor concentration

Treatment

No antidote available

Antipsychotics for psychotic behaviour , benzodiazepines for agitation and vitamins for adequate nourishment along with rest and sleep.

Group and Individual therapy

Cannabis/Marijuana

Cannabis

- Cannabis obtained from Cannabis Sativa.
- Principle component Delta 9-THC (Tetrahydrocannabinol)
- Commonly known as Weed/Marijuana grass , Mary Jane.
- Cannabis when smoked euphoric effects appear within 30 mins to 4 hrs.

Ganja- the flowering or fruiting tops of the female cannabis plant

Bhang- Dried leaves

Charas- resinous extract

Hashish- when cigarettes smoked with cannabis leaves

Hash oil- concentrated and most potent form

M : F – 2:1

Clinical features

Dilatation of conjunctival blood vessels (red eye) with mild tachycardia.

Increased appetite referred to as munchies

Practice of smoking ends of cannabis containing cigarettes 'roaches'

Long term cannabis use associated with cerebral atrophy , seizure susceptibility

Cognitive impairment (longer periods of heavy cannabis use)

Amotivational syndrome:

Long term heavy cannabis use leads to person unwillingness to persist in tasks , apathetic , anergic, slothful.

Intoxication

Heightens users sensitivity to external stimuli

makes colours seen brighter and richer

subjectively slows the appreciation of time.

Motor skill are impaired by cannabis use.

8-12 hrs after cannabis use motor skills are affected
like driving

Treatment

Antipsychotics for Cannabis induced psychotic disorder

Mood stabilisers and antidepressants for Cannabis induced mood disorder

Benzodiazepines to alleviate agitation

Nsaids for pain management

Withdrawal

Symptoms occurs within 1-2 weeks of cessation.

It includes

irritability

cannabis cravings

nervousness

Insomnia

Disturbed appetite

Weight loss

depressed mood

restlessness

Headache

sweating

and

tremors

Stimulants / Analeptics/Sympathomimetics (Amphetamine)

Amphetamines

- First synthesized as Racemic Amphetamine sulphate (Benzedrine) in 1887.
- Later introduced as inhaler for – treatment of Asthma and Nasal Congestion.
- Formulations like Dextroamphetamine , Methamphetamine, Amphetamine like compounds-Methylphenidate , Phenylpropanolamine , ephedrine.

All amphetamines can be taken orally ,injected or taken through “snorting”(inhaled) but some by intravenous route also

Designer Amphetamine: like MDMA (Ecstasy)- (3,4methylenedioxymethamphetamine)

Neurotransmitter involved Dopamine +Serotonin + Norepinephrine

MDMA (LSD + Amphetamines) = **Hallucinogenic + Stimulant actions** (R- LSD, R+ Amphetamine)

Lower doses stimulant like effects

Higher doses Hallucinogenic Effects

Effect last 4-8 hrs

Stimulant Intoxication

Mydriasis
Tachycardia or Bradycardia
Cardiac Arrhythmias
Dystonia
Weight Loss
Muscular weakness

Physical

Myocardial Infarction
Cerebrovascular diseases
Iv use leads to HIV, Hepatitis
For pregnant women congenital anomalies

Withdrawal

“Crash” occurs with Anxiety, Tremulousness, Dysphoric mood , Lethargy , Fatigue, nightmares with rebound REM sleep , headache , muscle cramps and hunger Withdrawal symptoms peak in 2 -4 days and resolved in 1 week.

The most serious symptom is depression

Narcotics a drug (such as opium or morphine) that in moderate doses dulls the senses, relieves pain, and induces profound sleep but in excessive doses causes stupor, coma, or convulsions.

Adverse Effects

Psychological

Restlessness
Insomnia
Irritability
Hostility
Confusion
Generalised Anxiety disorder & panic disorder
Delusion & hallucination

Tobacco

Most prevalent , deadly and costly of all substance dependence.

M > F

Psychoactive substance of tobacco is Nicotine.

Mechanism Of action

Agonist at acetylcholinergic receptors.

Effects also on Norepinephrine,epinephrine receptors.

Increase release of vasopressin and beta endorphins.

Half Life – 2 hrs

Withdrawal

1. Begins within 2 hrs of smoking the last cigarette, peak in 24 – 48 hrs and can last for weeks or months.
 2. *Symptoms include Tension, irritability, difficulty concentrating, drowsiness and paradoxical trouble sleeping.*
- Decreased motor performance and increased muscle tension.*

Clinical Features

Improved attention , learning , reaction time ,problem solving ability.

Tobacco users reports

1. Cigarette smoking lifts their mood
2. Decreases tension
3. Lessens depressive feeling.

Short term usage leads to increase in CBF, but in long term CBF decreases.

Adverse effects are more as compared to initial stimulatory benefits.

Various treatment strategies available to quit smoking

- Nicotine Replacement Therapy- Given for 2 weeks after quitting smoking
- Transdermal Nicotine Patch- slowest to act (16- 24 hrs)
- Nasal Sprays - Fastest to act (5-10 mins)
- Gum/inhaler - 20 mins
- Buprenorphine - smoking must be stopped on day 8th of treatment
- Varenicline - Reduces **rewarding properties** and **withdrawal**

Anabolic steroids

Testosterone, methyltestosterone

Should not be confused with corticosteroids.

Exploited for muscle building and masculinizing effects

Males > females

Indications

Male hypogonadism , Hereditary Angioedema

Breast Cancer, Osteoporosis in women

Clinical Features

Euphoria ,Hyperactivity, Increased anger , irritability, “Roid Rage”,

Those who stop taking abruptly become depressed, anxious and concerned with physical status of their body.

Adverse Effects

Testicular Atrophy , gynaecomastia & sterility in males

Shrinkage of breast , clitoral hypertrophy, hirsutism in females

Dermatological reactions in the form of Acne and Male pattern baldness

Gambling Disorder

Definition

- ❑ Persistent and recurrent problematic gambling behaviour leading to clinically significant impairment or distress.
- ❑ It leads to economic problems and significant disturbances in personal , social, or occupational functioning.

Diagnosis

1. Preoccupation with gambling
2. Need to gamble with increased amount of money to achieve desired excitement (tolerance)
3. Repeated and unsuccessful attempts to control-,cut back or stop gambling.
4. Gambling as a way to escape from problems.
5. Feeling restless and irritable when attempting to reduce or stop gambling (withdrawal)
6. Jeopardizing personal or vocational relationships for gambling.
7. Gambling to recoup losses.
8. A reliance for others to pay off debts

Prevalence .1 – .7 % in general population

2/1000

M > F

Adolescents-Males Late middle age – Females

Disorder runs in 4 phases

1. Winning Phases- hooks the patient mostly
2. Progressive loss phase- losses money in a sequential pattern going towards bankruptcy.
3. Desperate Phase- Gambling with large amounts of money not paying them taking loans altogether making life miserable.
4. Hopeless Phase- Losses can never be restored.

Clinical features

Overconfident , Energetic, spendthrift , Antisocial personality traits , involvements in fraud and forgery very often

Rx

No FDA drug till now

But beneficial results with

Naltrexone-Opioid Antagonists

SSRI - fluvoxamine 200-250 mg , Paroxetine 51.7 mg

Mood Stabilisers- Valproate ,lithium Carbamazepine

Psychological :

Insight oriented Therapy, Cognitive Behaviour therapy



IIT Kharagpur

Substance Time present in urine

Alcohol	Up to 12 hrs
Amphetamine	Up to 48 hours
Benzodiazepine	3 days (depending on $t_{1/2}$)
Cannabis Occasional use –	Up to 3 days. High daily use for long time – up to 4 weeks.
Cocaine	6 – 8 hrs; metabolites up to 2 - 4 days
Codeine	48 hours
Heroin	1 to 3 days
Methadone	3 days or more
Morphine	2 to 3 days
Phencyclidine (PCP)	3 to 8 days
LSD	Less than 24 hours (average users)

•

Behavioural Addiction

Diagnostic and Statistical Manual of Mental Disorders V includes a major change to the diagnostic category for addictions.

The *Substance-related Abuse and Dependency* category has been **reabeled** *Substance-Related and Addictive Disorders*

Modified to include two subdivisions:

- a) Substance-related disorders
- b) Non-substance-related disorders (*addictive disorders that do not involve ingestion of a psychoactive substance*)

Behavioral addictions **resemble** *substance addictions* in many domains.

- 1) Natural history (chronic, relapsing course with higher incidence and
- 2) Prevalence in adolescents and young adults
- 3) Phenomenology (subjective craving, intoxication [“high”], and withdrawal)
- 4) Tolerance
- 5) Comorbidity
- 6) Overlapping genetic contribution
- 7) Neurobiological mechanisms (with roles for brain glutamatergic, opioidergic, serotonergic and dopamine mesolimbic systems)
- 8) Response to treatment.

Non Substance Related disorders

- The current Diagnostic and Statistical Manual, 5th edition has designated formal diagnostic criteria for several of these disorders which includes Pathological gambling which was a part of Behavioural Addiction is incorporated now in substance related disorders
- A different domain of Behavioural Addiction consisting of:
 - i) Computer/ video game playing, and internet addiction
 - ii) Sexual addiction (non-paraphilic hypersexuality)

Internet Addiction

First described in 1998 by Young

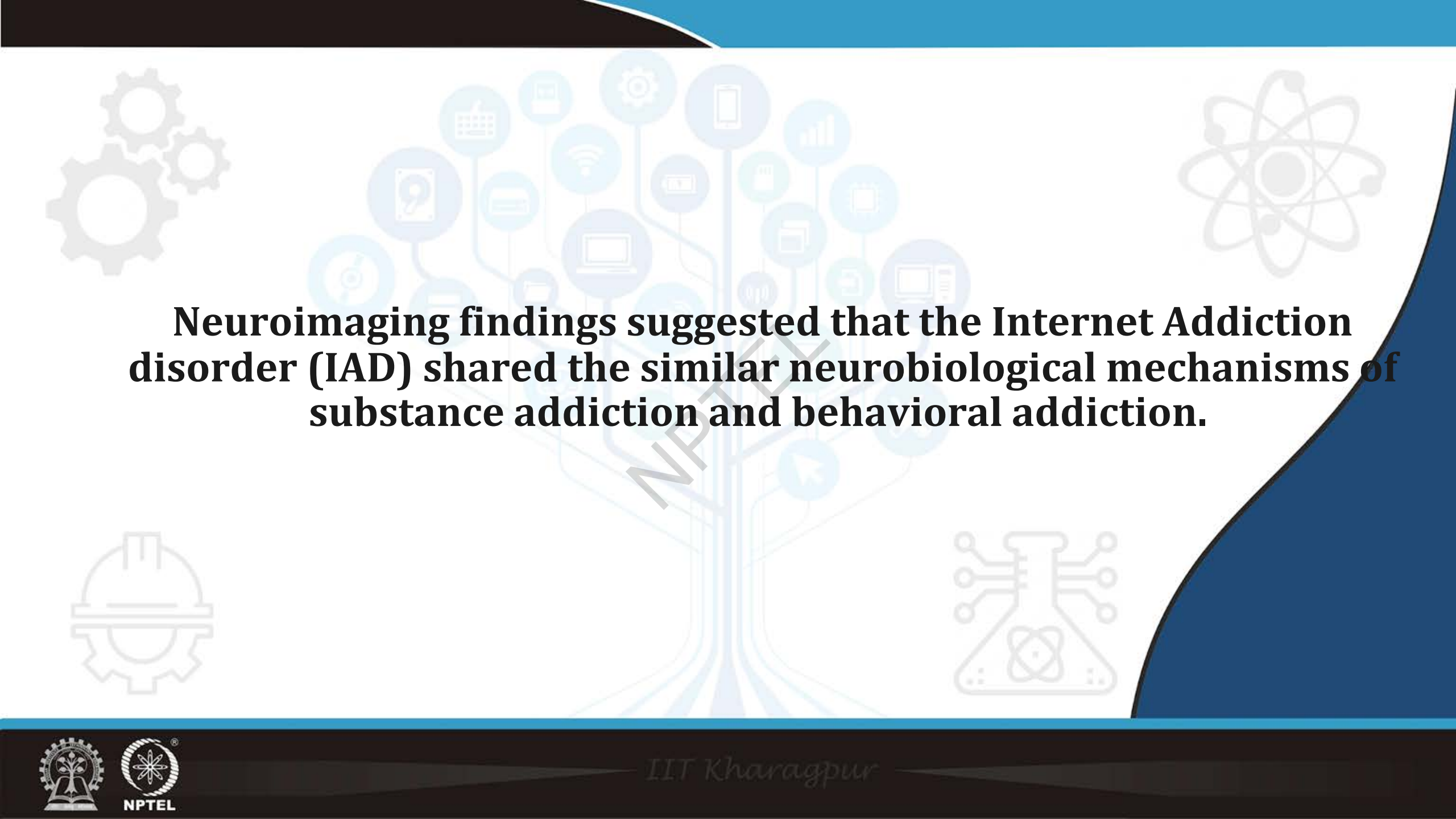
5 criteria has to be present in order to diagnose **Internet addiction**

- (1) Is preoccupied with the Internet (thinks about previous online activity or anticipate next online session)
- (2) Needs to use the Internet with increased amounts of time in order to achieve satisfaction
- (3) Has made unsuccessful efforts to control, cut back, or stop Internet use
- (4) Is restless, moody, depressed, or irritable when attempting to cut down or stop Internet use
- (5) Has stayed online longer than originally intended.

Age group : Late Adolescence

Additionally, at least one of the following must be present

- 1. Has jeopardized or risked the loss of a significant relationship, job, educational or career opportunity because of the Internet
- 2. Has lied to family members, therapist, or others to conceal the extent of involvement with the Internet
- 3. Uses the Internet as a way of escaping from problems or of relieving a dysphoric mood (e.g., feelings of helplessness, guilt, anxiety, depression)



Neuroimaging findings suggested that the Internet Addiction disorder (IAD) shared the similar neurobiological mechanisms of substance addiction and behavioral addiction.

Sexual Addiction

DSM V includes Sexual Addiction as a disorder characterized by intrusive and obsessive sexual thoughts and fantasies, associated with loss of control over sexual behavior that causes negative consequences at work, emotional, and social levels .

This is a psychopathological condition that is correlated with an alteration of the cerebral gratification system involving a coarctation of the modality in which the individual obtains satisfaction and pleasure.

One of the fundamental hallmarks of compulsive sexual behavior is continued engagement in sexual activities despite the negative consequences created by these activities.

Compulsive sexual behaviour can be divided into paraphilic and non-paraphilic subtypes.

Paraphilic behaviours refer to behaviours that are considered to be outside of the conventional range of sexual behaviors.

8 paraphilias

**i) Exhibitionism ii) voyeurism iii) Pedophilia iv) Sexual masochism v) Sexual sadism
vi) Transvestic fetishism vii) Fetishism viii) Frotteurism.**

Non-paraphilic behaviors represent engagement in commonly available sexual practices, such as

i) Attending strip clubs ii) Compulsive masturbation iii) Paying for sex through prostitution iv) Excessive use of pornography v) Repeated engagement in extramarital affairs.

Consequences of compulsive sexual behaviors

- 1) Medical Disorders like HIV, STD's , Hepatitis b & C , Syphilis** **2) Loss of time & productivity**
- 3) Legal consequences due to engagement in illegal acts.** **4) Psychological consequences** are numerous like Compulsive sexual behaviors can establish unhealthy and unrealistic expectations of what a satisfying sexual relationship should be , leads to separation and divorce and, in turn, puts any future healthy relationship in doubt.
- 5) Finally, the shame and guilt**

Treatment

Psychosocial

- 1 . Cognitive behavioural therapy
2. Psychodynamic psychotherapy

Pharmacological

No FDA drug approved as of now

SSRI's limited evidence (Fluoxetine , sertraline, fluvoxamine)

Opioid Antagonists (Naltrexone) some evidence

REFERENCES

- 1 Oxford test book of psychiatry
- 2 Comprehensive text book of psychiatry
- 3 Text book of psychiatry (tasman & leibermann)
- 4 How can we conceptualize behavioural addiction without pathologizing common behaviours?
Daniel kardefelt-winter, alexandre heeren, adriano schimment, antonius van rooij, pierre maurage, mic
5. Introduction to behavioral addictions jon E. Grant, marc N. Potenza, aviv weinstein, and david A. Gorelick
- 6 Relationships between behavioural addictions and psychiatric disorders: what is known and what is yet to be learned vladan starcevic and yasser khazaal.
- 7 Murali et al., 2012
- 8 .Stephen stahl's psychopharmacology

CONCLUSIONS

-In this lecture we have discussed regarding concepts of Benzodiazepines, Cannabis , Hallucinogens ,Tobacco ,Inhalants , Stimulants, Gambling dependance and its treatment.

Concepts regarding Behavioural Addiction in brief



**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 28: Physiology of sensations

CONCEPTS COVERED

1. Sensory receptors
2. General principles of sensory processing
3. Touch
4. Pain

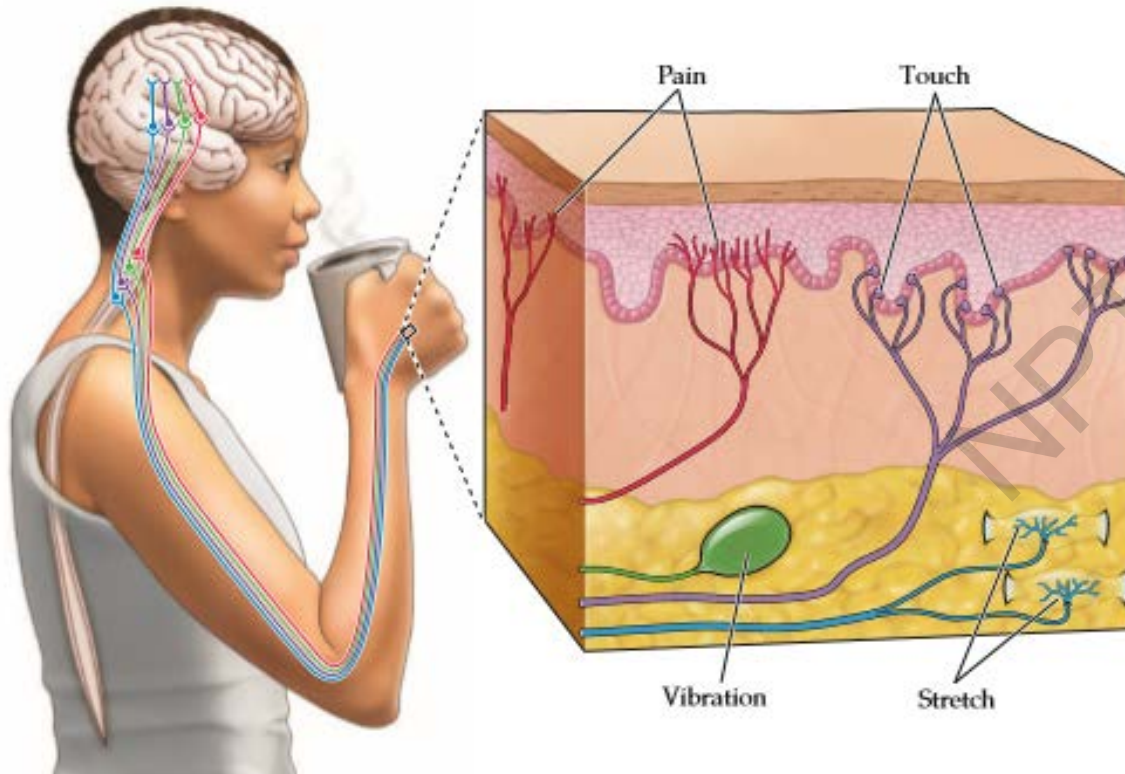
Sensory receptor organ - an organ (such as the eye or ear) specialized to receive particular stimuli.

Stimulus- A physical event that triggers a sensory response

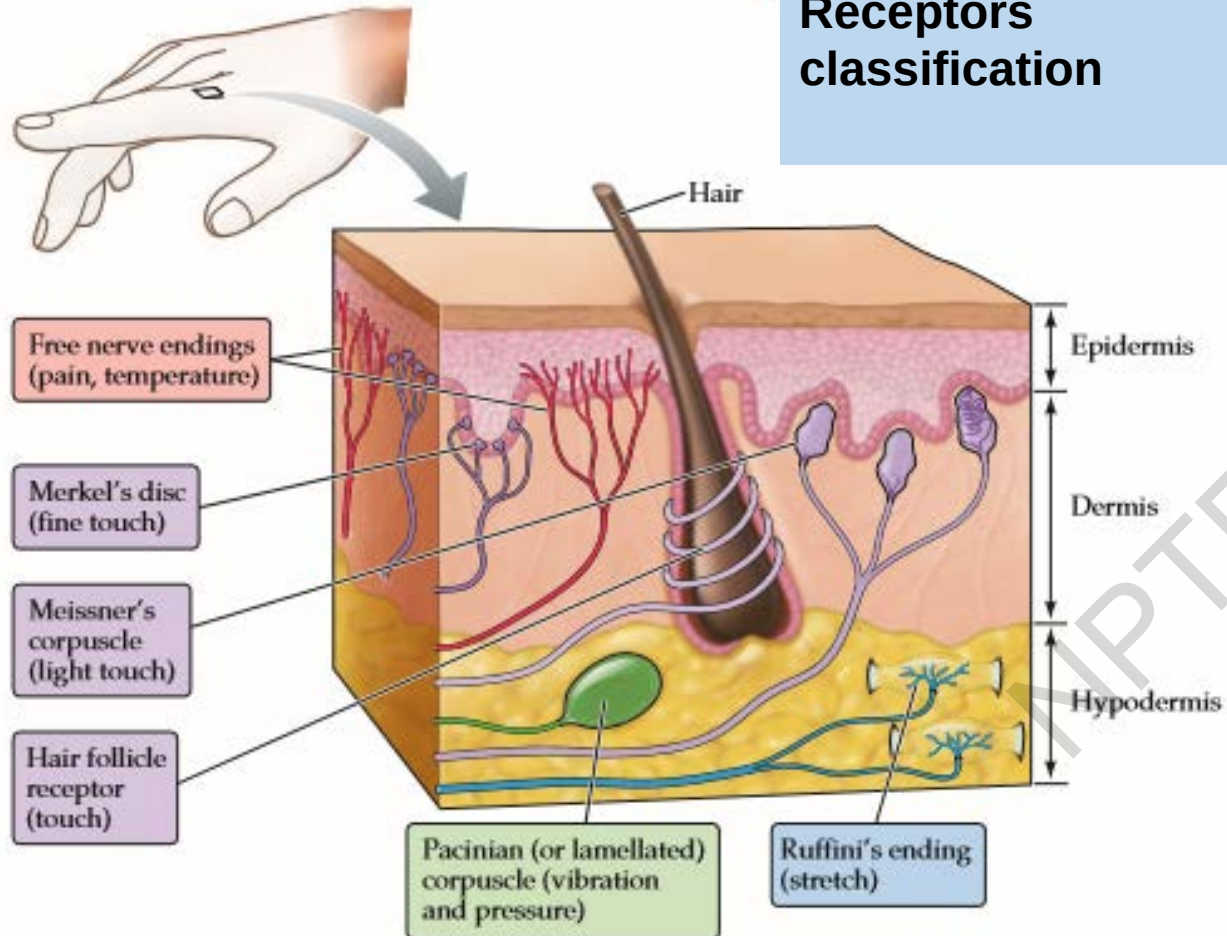
Receptor cell- A specialized cell that responds to a particular stimulus

Muller-doctrine of specific nerve energies,

LABELED LINES Each type of receptor (stretch, vibration, pain, touch) has a distinct pathway to the brain,



Receptors classification



Mechano R

Tactile

Nociceptor

Proprioceptor

Hair cells of organ of corti

Thermo R → activated by temp, **cold**
Max present – lips>fingertips

Photo R → rods & cones

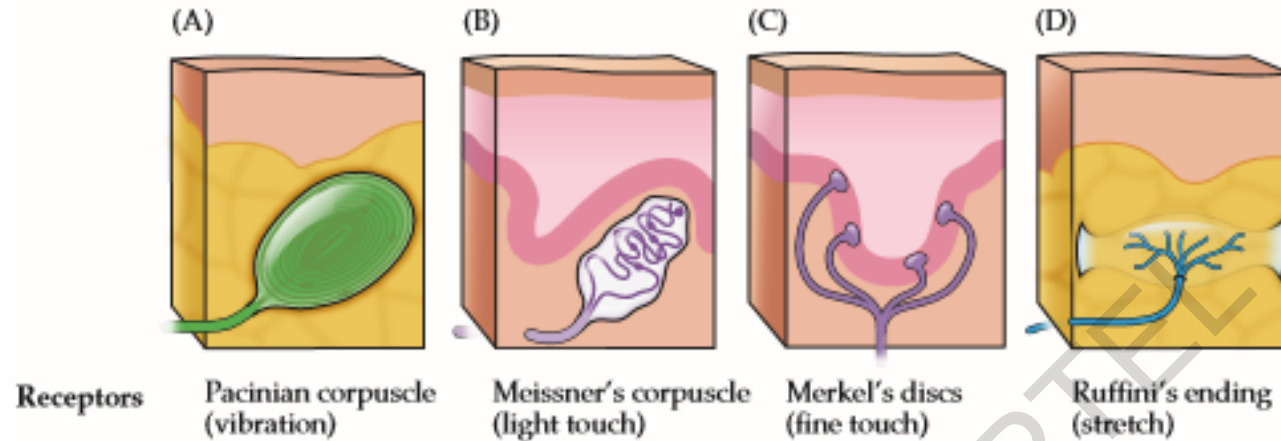
Chemo R

Polymodal

hot/warm

Tactile receptors

Max fingertips > lips



Most sensitive
Largest

numerous
movement
non-hairy skin

epidermis
2 point discrimination
point/edges/corner

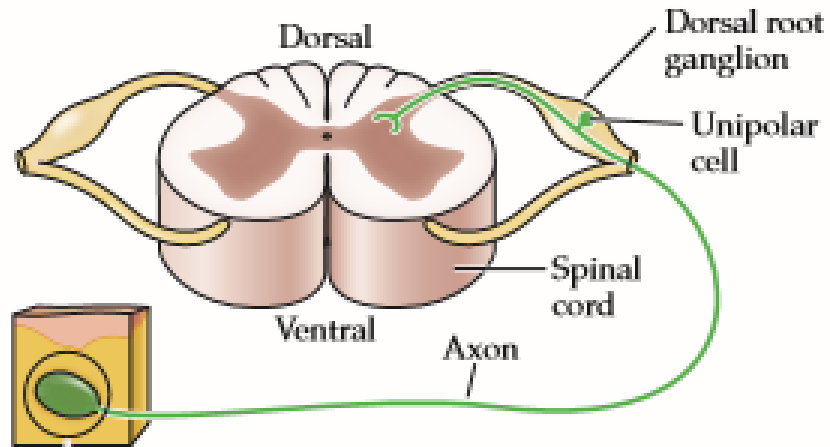
Adaptation- the progressive loss of receptor sensitivity as stimulation is maintained.

coding - rules by which action potentials in a sensory system reflect a physical stimulus.

Adaptation- decrease in AP frequency

The initial stage of sensory processing is a change in electrical potential in receptor cells

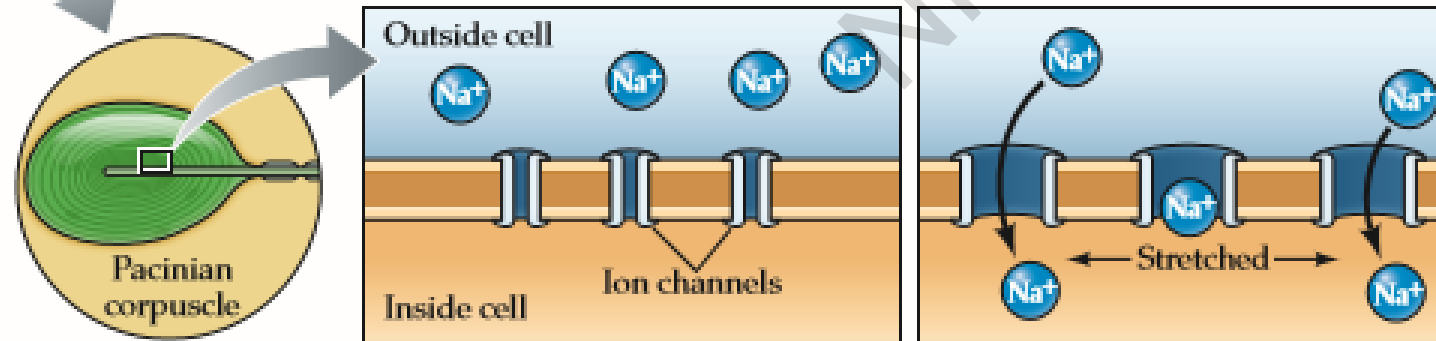
(A) Innervation of a Pacinian corpuscle



(B)

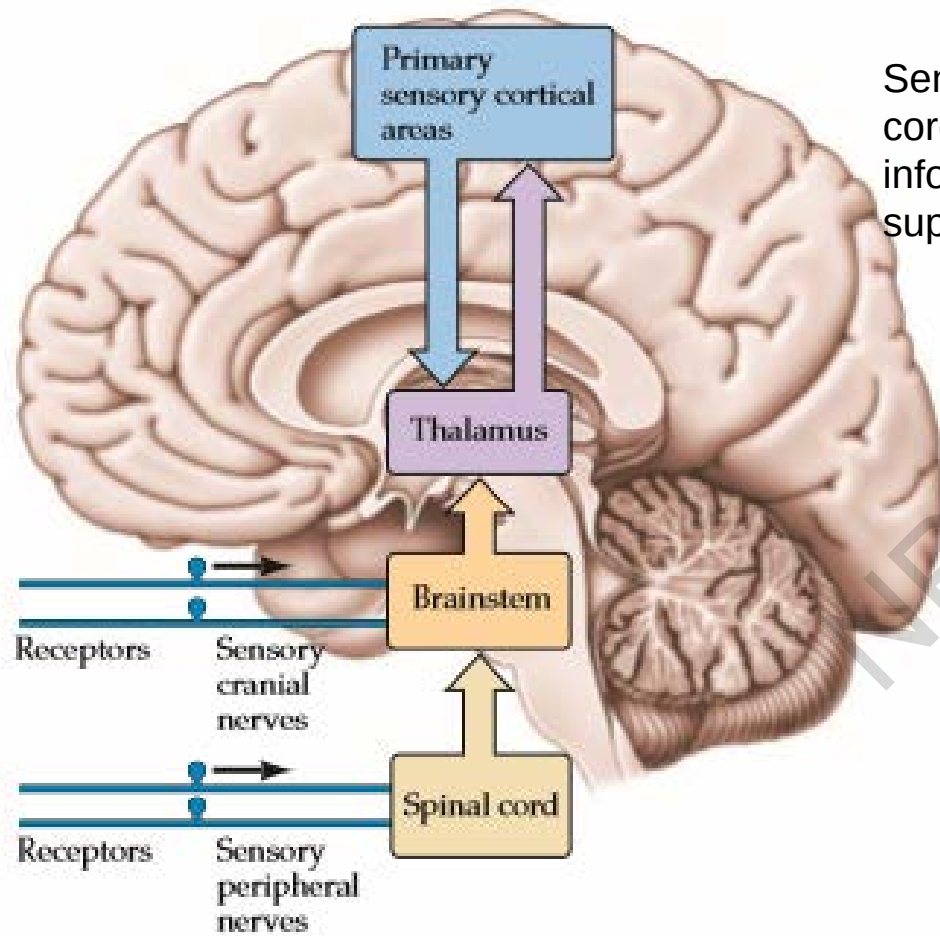
Nerve membrane at rest

Membrane stretched, excited section

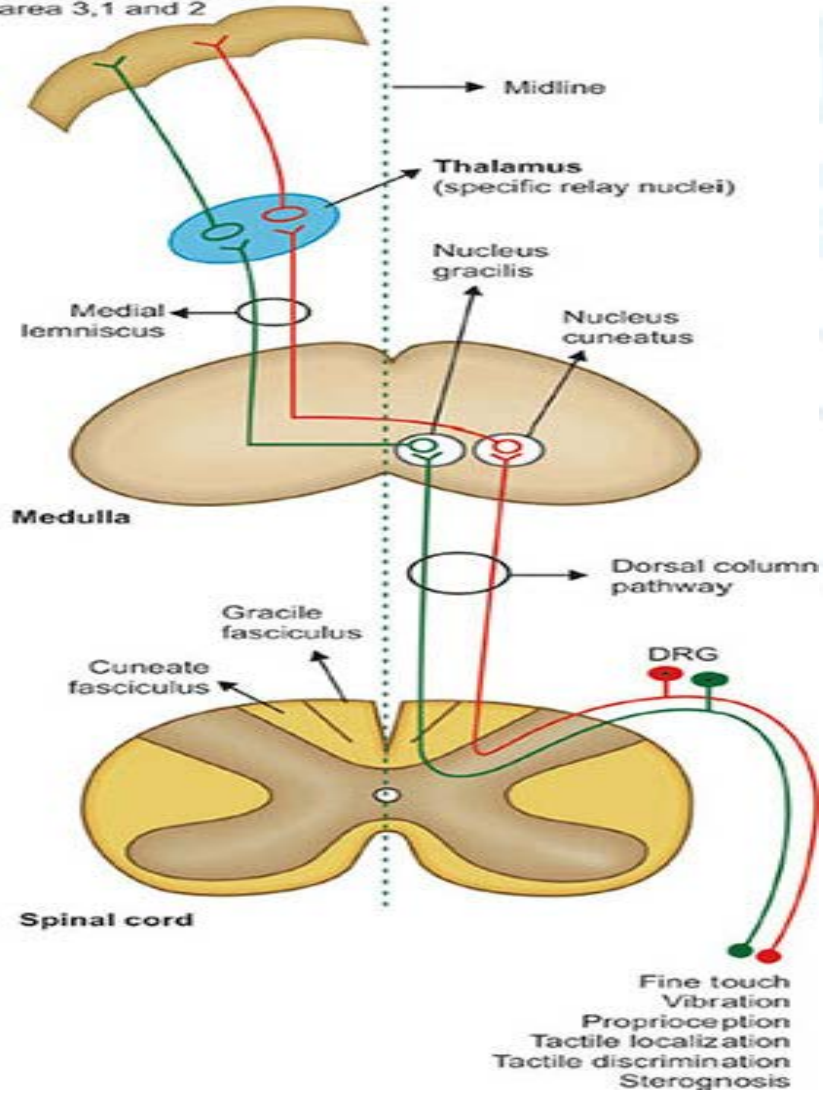


Levels of sensory processing

Sensory information enters the CNS through the brainstem or spinal cord and then reaches the thalamus. The thalamus transmits the information to the cerebral cortex; the cortex directs the thalamus to suppress some sensations.



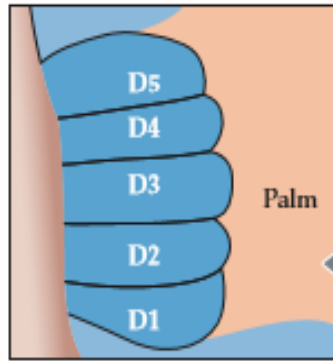
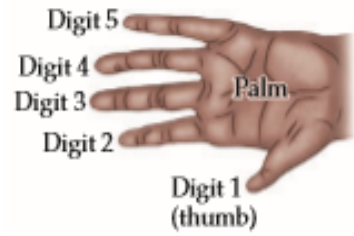
Sensory cortex
(postcentral gyrus)
area 3,1 and 2



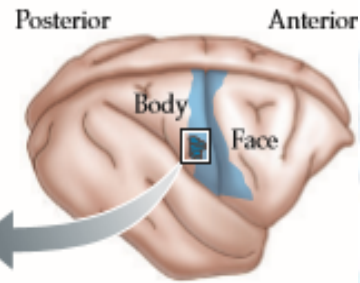
The Dorsal Column System Carries Somatosensory Information from the Skin to the Brain

THE PLASTICITY OF SOMATOSENSORY REPRESENTATIONS

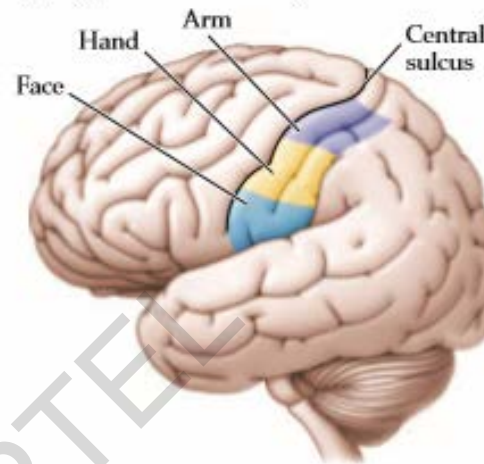
(A) Representation of the left hand in primary somatosensory cortex in right hemisphere of monkey brain



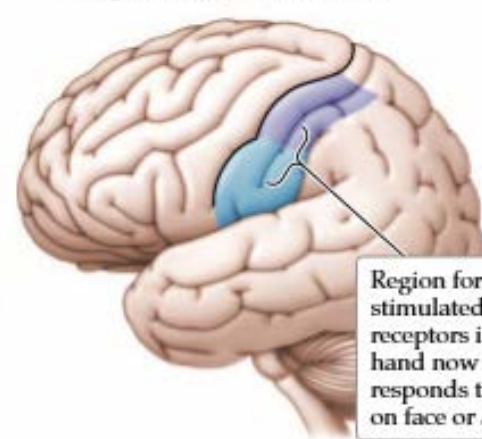
Details of cortical map
(D5 = digit 5, etc.)



(A) Typical somatosensory cortex



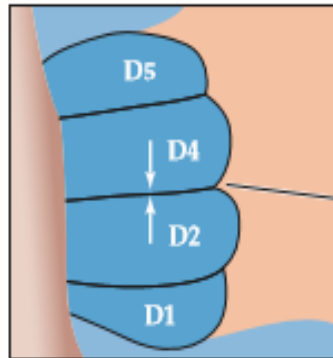
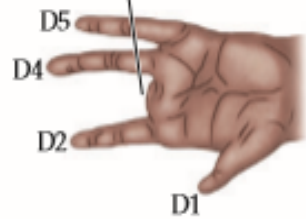
(B) Somatosensory cortex reorganized after loss of hand



Region formerly stimulated by receptors in the hand now responds to touch on face or arm.

(B) Experiment 1

D3 surgically removed.



Several weeks later, areas representing D2 and D4 have expanded, replacing the representation of D3.

Nociceptors

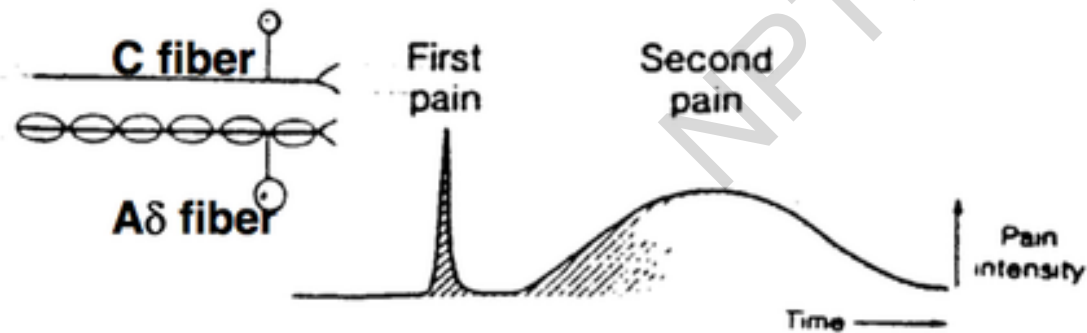
Stimuli- Mechanical, Thermal, Chemical

Slow pain

fast pain

Present in free nerve endings
Contain various ion channels
Eg TRP

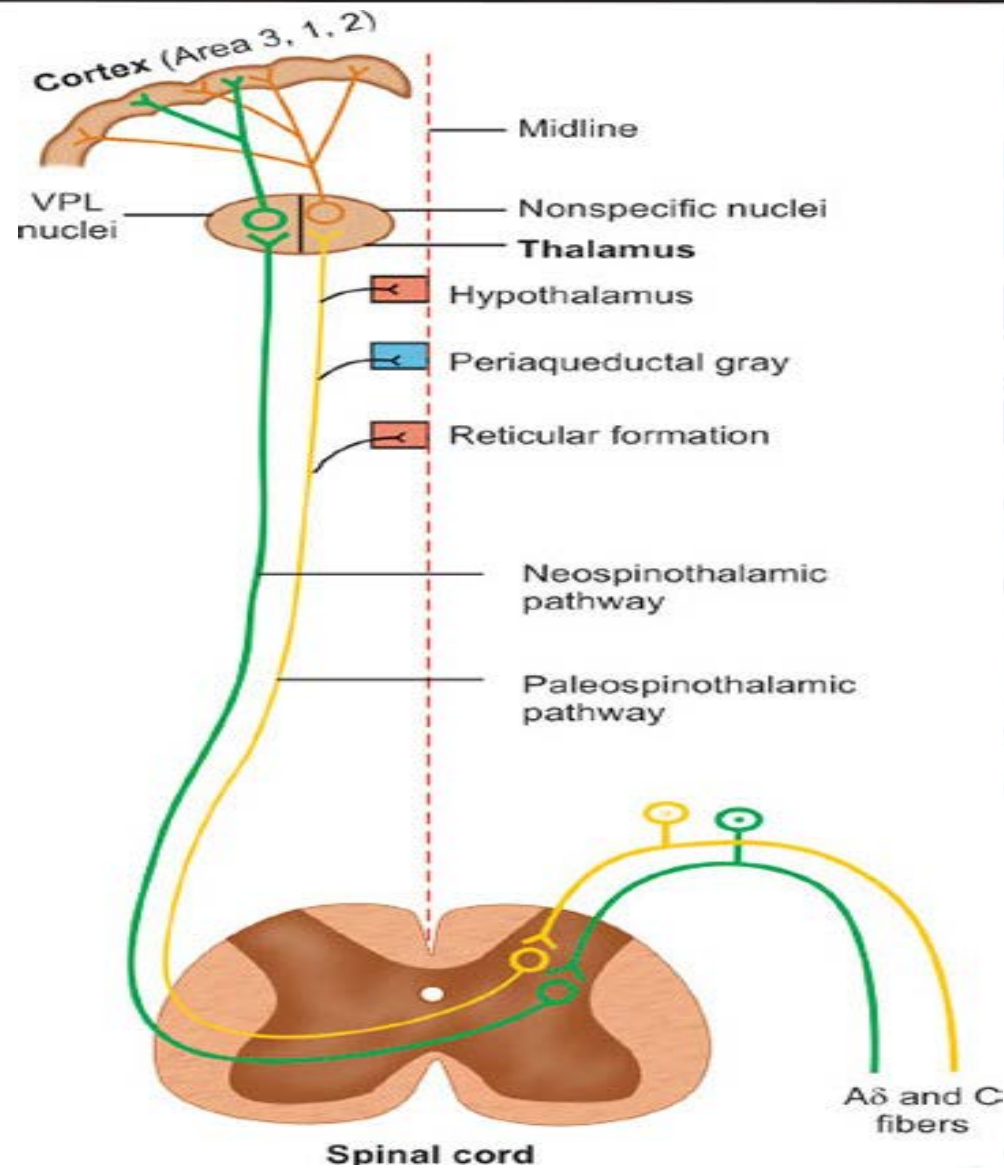
NaV1.7- A voltage gated sodium channel
used almost exclusively by nociceptors to
initiate action potentials.



Mechanical	Thermal		Chemical
TRPA1	COLD	HOT	Bradykinin
P2X/P2Y	CMR1	TRPV1	serotonin
MRGPR-D	TRPA1	TRPV2	ASIC
		TRPV3	NGF

Hyperalgesia- exaggerated pain due to decreased threshold of nociceptor

Allodynia- pain to non-nociceptive stimuli



Pain pathway- fast & slow

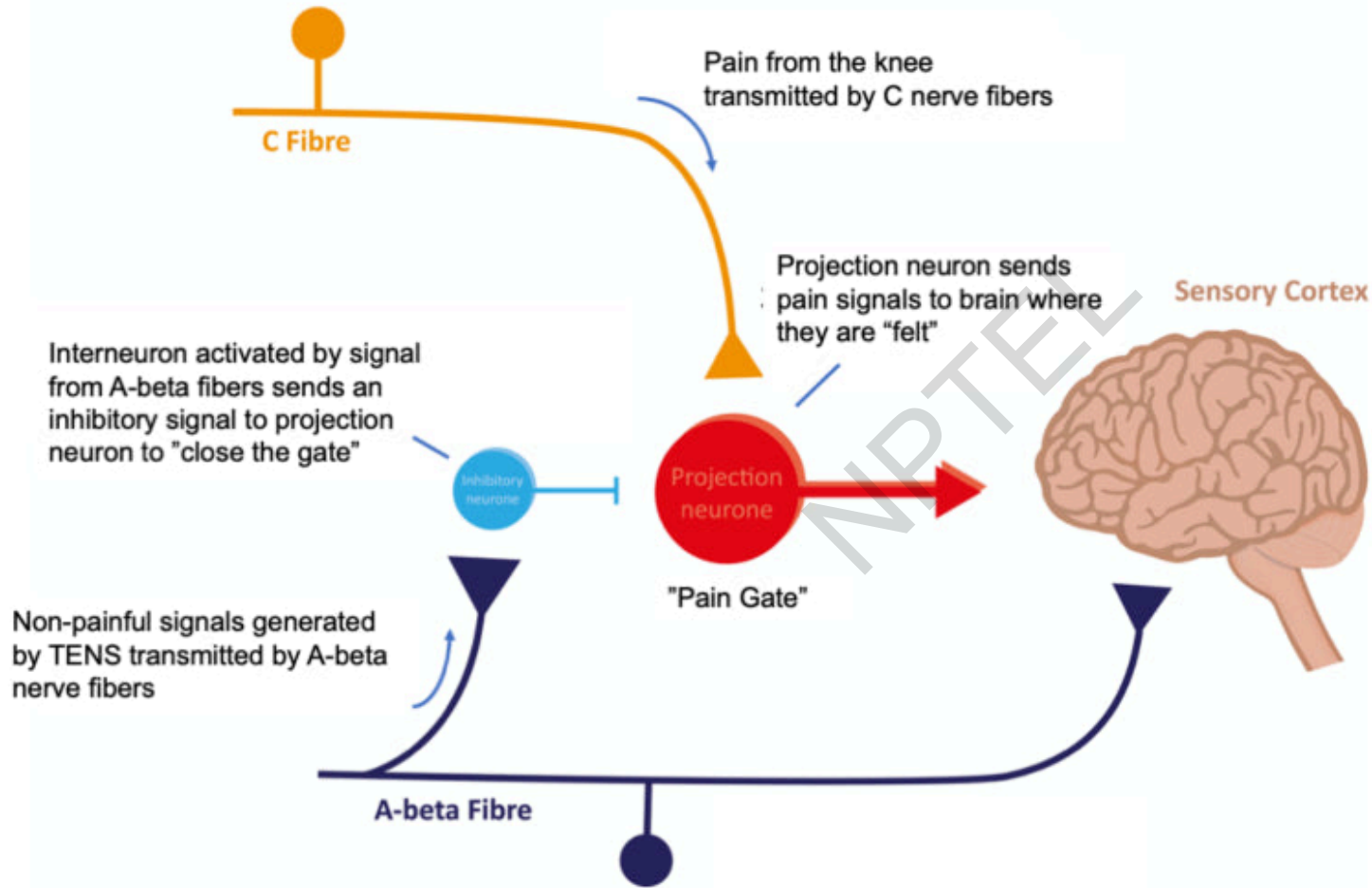
A delta → Lamina 1 → glutamate → 2nd order neuron

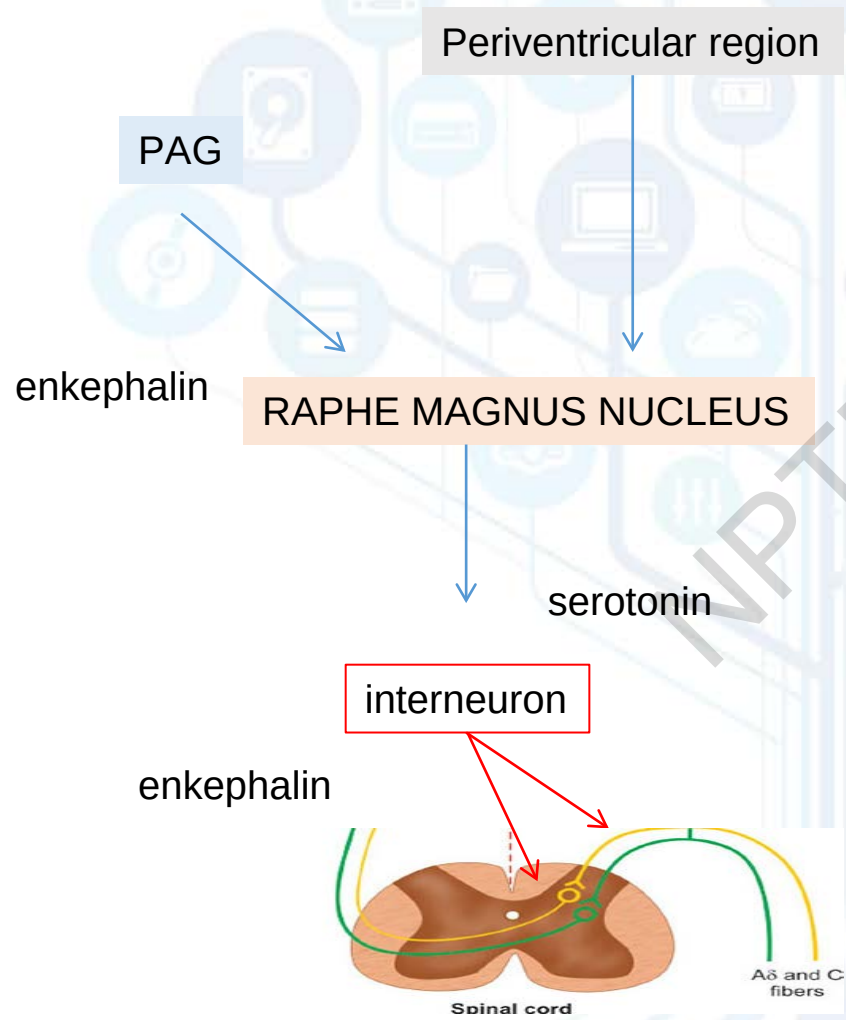
C → lamina 2,3 → lamina 5 → sub P → 2ND order neuron

Thalamus- Thalamic pain syndrome

The most dramatic example is a person's continued perception of chronic pain coming from a missing limb after loss of an arm or leg. Called phantom limb pain, this sensation is an example of neuropathic pain,

Gate Control Theory of Pain





Keypoints

- The receptive field of a neuron is the region in space where a stimulus will change the firing of that cell.
- Touch information from the body courses through a distinct spinal pathway, the dorsal column system.
- Free nerve endings act as nociceptors, detecting mechanical damage or temperature changes because they have specialized receptor proteins that detect these conditions (such as transient receptor potential vanilloid 1 [TRPV1]).
- Pain, temperature, and itch information enters the spinal cord, crosses the midline, and ascends through the anterolateral (spinothalamic) system.

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1. Guyton and Hall Textbook of Medical Physiology, John E. Hall, 13e
2. Stahl's Essential Psychopharmacology, 4e
3. Behavioural Neuroscience, Breedlove, 6e



**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 29 : Psychosomatic Illness

CONCEPTS COVERED

- Introduction to Psychosomatic illness
- Hypochondriasis
- Illness anxiety disorder
- Fibromyalgia
- Chronic fatigue syndrome
- Factitious Disorder
- Malingering
- Pain disorder
- Psychogenic Excoriation

Psychosomatic illnesses / Psychological factors affecting physical condition

Introduction to Psychosomatic illness

Psycho/somatic-

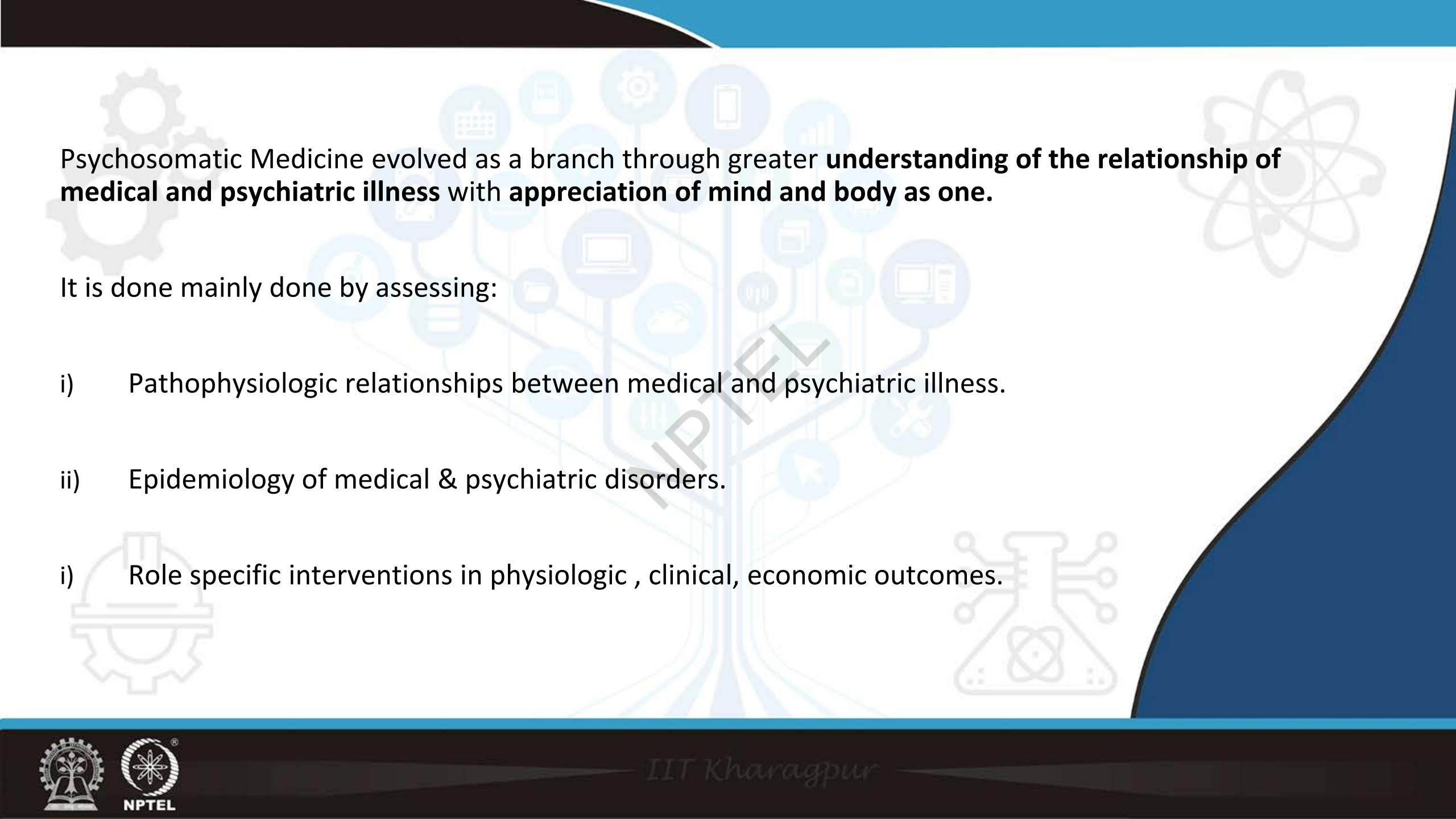
Psyche- soul

Soma – body

It literally means how mind affects body.

There can be various problems in Psychosomatic illness :

- i) Psychiatric symptoms secondary to a medical condition → Delirium, Dementia
- ii) Psychiatric Complication of a medical condition → Depression after interferon treatment.
- iii) Medical complications of psychiatric conditions → Neurolept Malignant Syndrome
- iv) Co-occurring medical & psychiatric condition → Depression in setting of cancer treatment.



Psychosomatic Medicine evolved as a branch through greater **understanding of the relationship of medical and psychiatric illness with appreciation of mind and body as one.**

It is done mainly done by assessing:

- i) Pathophysiologic relationships between medical and psychiatric illness.
- ii) Epidemiology of medical & psychiatric disorders.
- i) Role specific interventions in physiologic , clinical, economic outcomes.

Role of Psychiatric evaluation in Psychosomatic illness

Complete Psychiatric history which includes:

- i) Past history
- ii) Family history
- iii) Developmental history
- iv) Review of current & medical history
- v) Mental status examination which includes cognitive ,neurological & physical examination.
- vi) To gain the understanding of the patients experience of illness.
- vii) To develop understanding of the patients developmental and personal history as well as key dynamic conflicts.

Inference:

- i) Understanding of the patients explanation of stress, their personality traits , their coping strategies employed to deal with stress and defence mechanism if any.
- ii) **Gives an insight to shape the treatment models in line with the requirements of the patient (tailor made)*.**

Treatment for Psychosomatic illness

i) **Stress Management & Relaxation therapy**

CBT – a) To help individuals to be aware about their own Cognitive appraisals of stressful events.

b) To educate individuals about how their cognitive appraisal influence negative emotional & behavioural responses as to alter them

c) To teach various effective cognitive & behavioural stress management skills.

ii) **Cognitive restructuring**

iii) **Relaxation Exercises**

iv) **Self Observation** – diary to record as to how people respond in stressful or challenging situations

v) **Medical Management**- when comorbidly associated with anxiety/depression , panic disorder

Hypochondriasis/ Somatic symptoms Disorder

Diagnosis:

It is Characterized by 6 or more months of a general and non-delusional preoccupation with fears of having or the idea that one has a serious disease based on the persons misinterpretation of bodily symptoms.

This preoccupation causes significant distress and impairment in ones life.

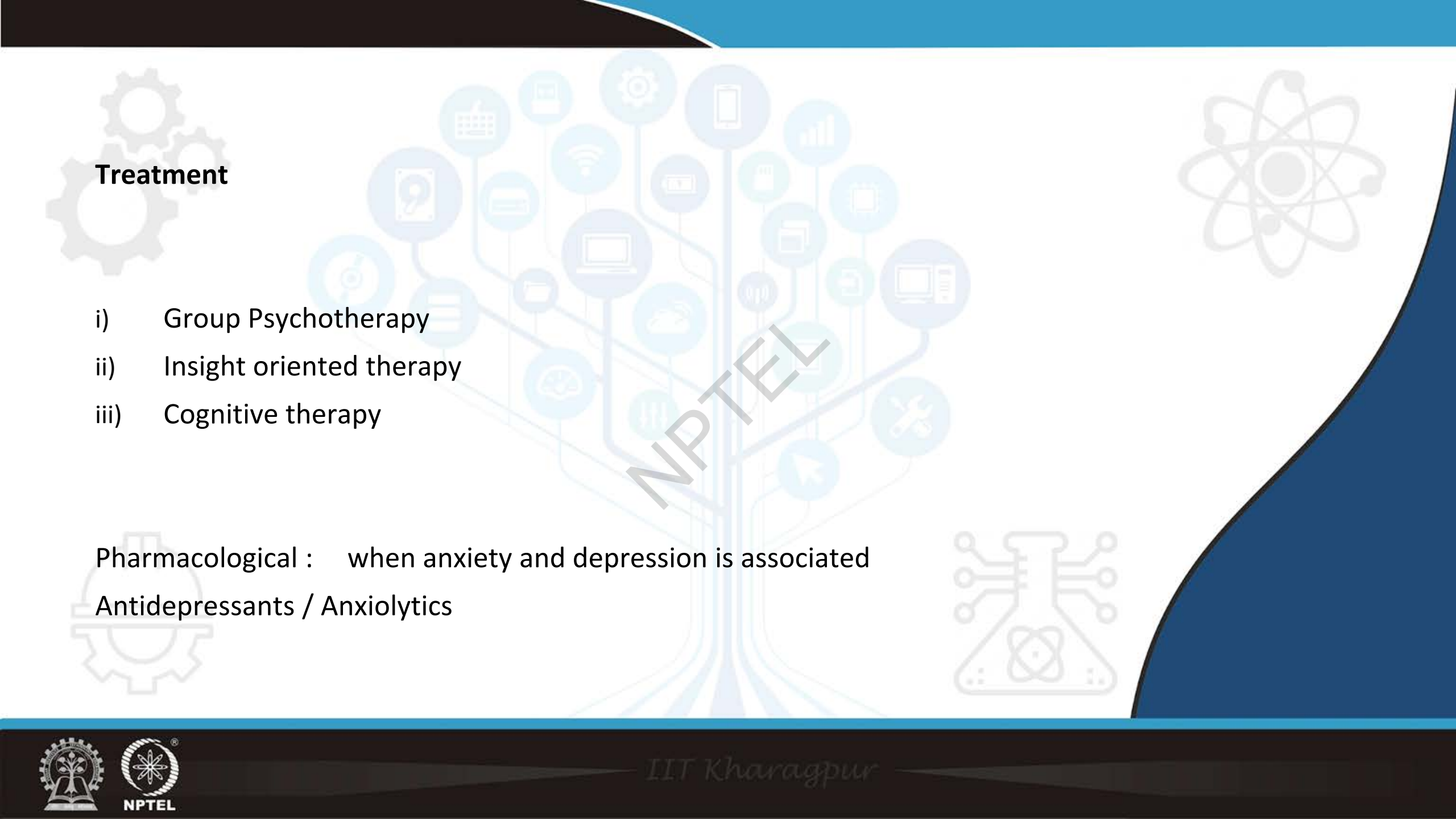
It should not be accounted for by other medical /psychiatric condition.

Prevalence- 4-6%

Males marginally more than females

Etiology

- a) Social learning Model- sick role
- b) Psychodynamic Model- aggressive , hostile wishes transferred into physical complaints
- c) Defence against guilt



Treatment

- i) Group Psychotherapy
- ii) Insight oriented therapy
- iii) Cognitive therapy

Pharmacological : when anxiety and depression is associated
Antidepressants / Anxiolytics

Illness Anxiety Disorder

New entity added in DSM – 5

Diagnosis:

Patients preoccupied with the false belief that they have or will develop a serious disease and there are a few if any physical signs and symptoms.

This belief must last 6 months and cannot be accounted by medical /psychiatric illness neither should it be delusional.

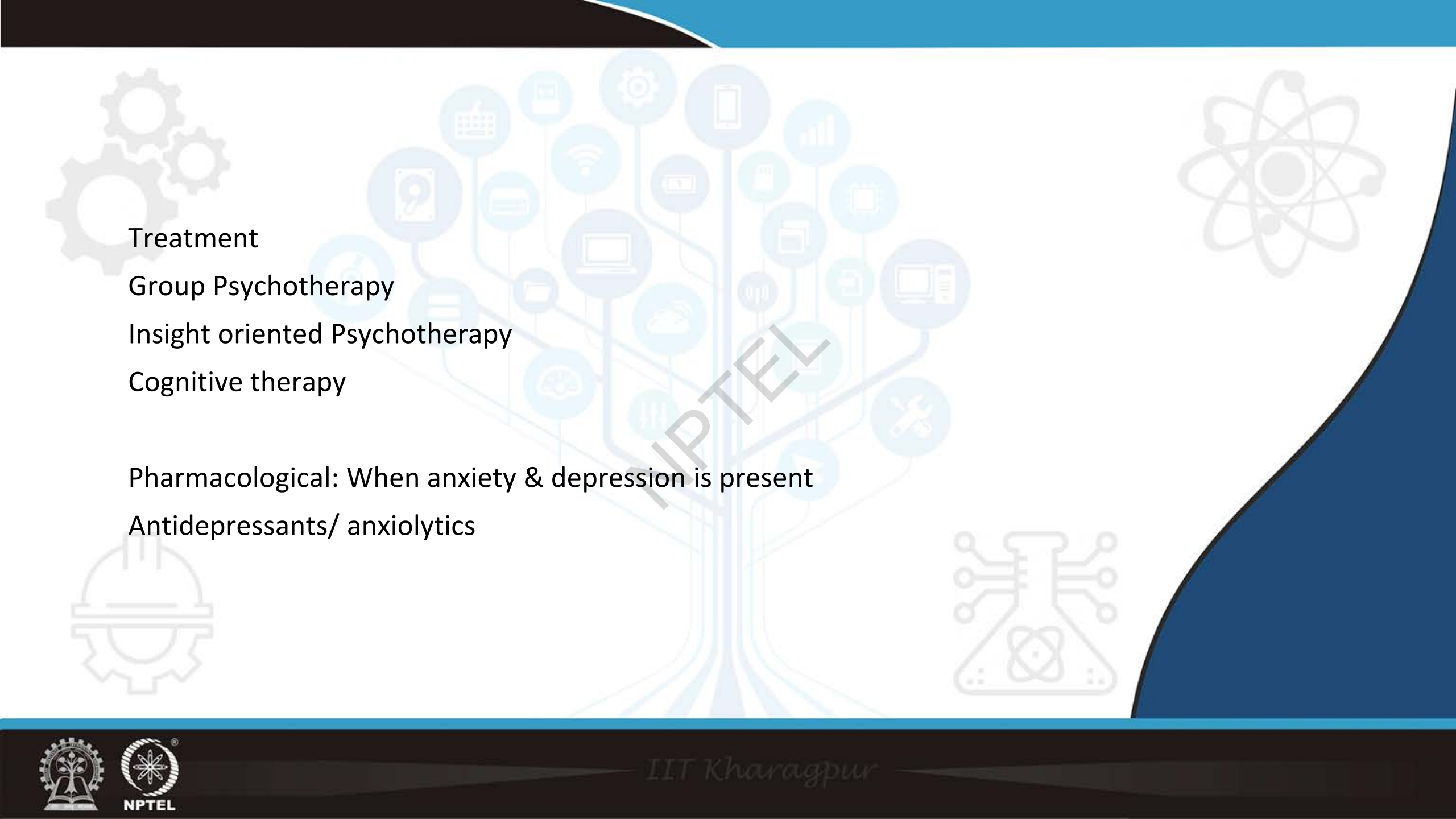
This should cause significant distress and impairment in one's life.

Prevalence - 4- 6%

older age > Young age

Etiology

- i) Social learning Model
- ii) Psychodynamic Model
- iii) Defence against guilt



Treatment

Group Psychotherapy

Insight oriented Psychotherapy

Cognitive therapy

Pharmacological: When anxiety & depression is present

Antidepressants/ anxiolytics

Fibromyalgia

Diagnosis:

Patients must have widespread pain for atleast 3 months at predefined tender points upon palpation.

The symptoms are always broader than pain lone and include

- i) Complaints of fatigue
- ii) Muscular weakness
- iii) Sleep disturbances
- iv) Impairment of cognitive domains such as concentration.

Diagnosis should be done by excluding rheumatic disorders/ hypothyroidism

Females > Males

Etiology

Precipitated by stress that causes localized arterial spasms that interfere with perfusion of oxygen in the affected areas.

Most commonly associated with Chronic fatigue syndrome, depression, anxiety disorders and post traumatic stress disorders.

Characterized by pain, stiffness of the soft tissues such as muscles- , ligaments , tendons,
Cervical and thoracic areas are affected most often.

Treatment

FDA approved – pregabalin

Antidepressants

NSAIDS for pain

Non pharmacological

Insight oriented psychotherapy

Chronic fatigue Syndrome

Criteria as per Centre for disease control & prevention:

A Severe unexplained fatigue for over 6 months that is:

- i) Of a new or definite onset
- ii) Not due to continuing exertion
- iii) Not resolve by rest
- iv) Functionally impairing

B The presence of 4 or more of the following new symptoms.

- i) Impaired memory or concentration
- ii) Sore throat
- iii) Muscle pain
- iv) Pain in several joints
- v) New pattern headaches
- vi) Unrefreshing sleep
- vii) Post exertional malaise lasting more than 24 hours.

Etiology : Probably multi factorial and is best understood by dividing it into

1. Childhood inactivity or illness)
2. Proposedly viral origin (Infectious mononucleosis, Q fever, Lyme disease or serious life events)
3. Strong belief in physical cause, activity avoidance, poor self control, low self perception of cognitive ability).

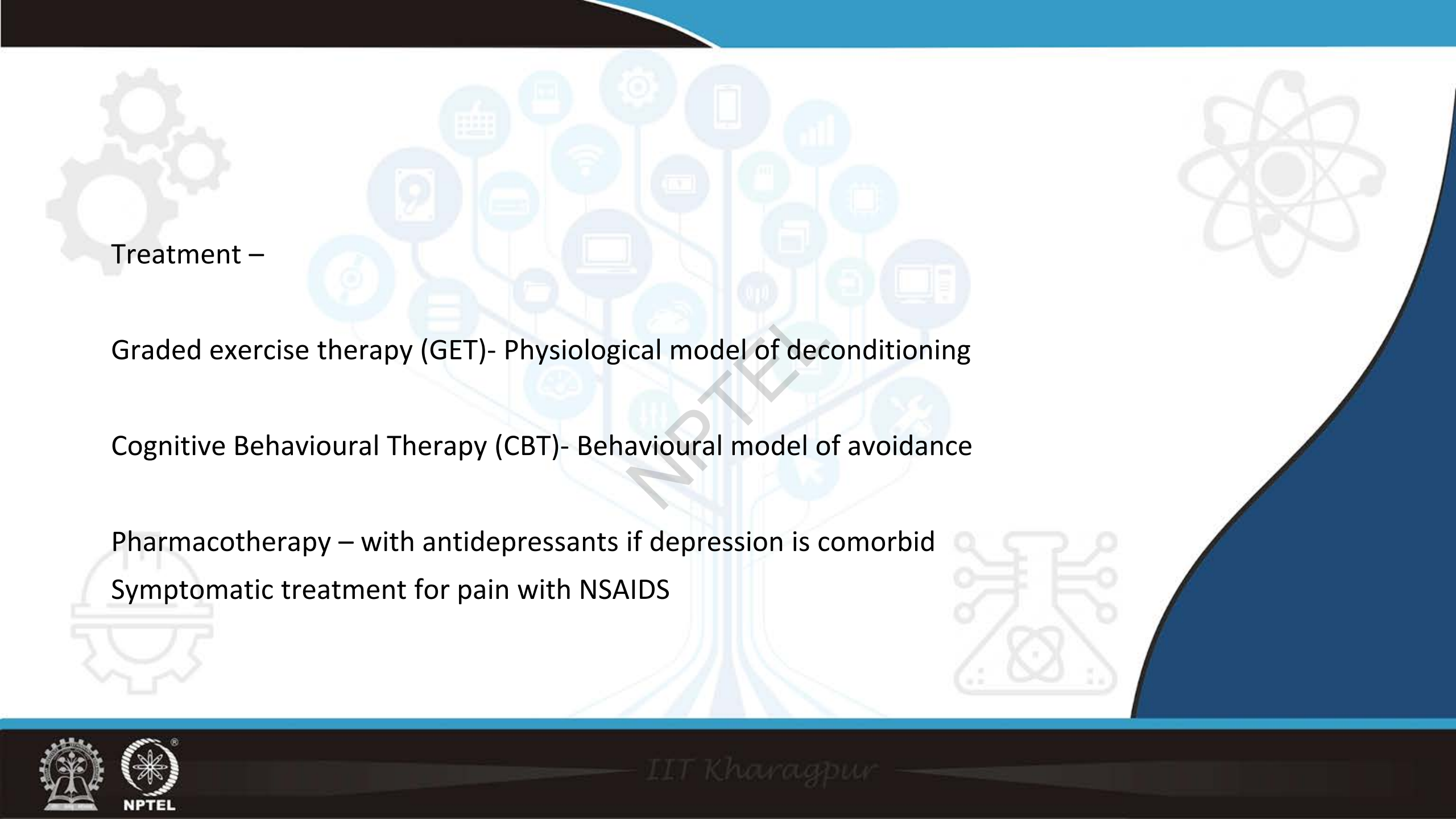
Cytokines alpha interferons and IL -6 are under investigation as possible etiological factors.

1/3rd have hypocortisolism.

F:M- 2:1

CFS patients have an abnormality in HPA axis and serotonin pathway suggesting altered physiological response to stress.

Upto 80 % of patients with CFS meet the criteria for depression, but none of them presents with guilt, suicidal ideation or anhedonia



Treatment –

Graded exercise therapy (GET)- Physiological model of deconditioning

Cognitive Behavioural Therapy (CBT)- Behavioural model of avoidance

Pharmacotherapy – with antidepressants if depression is comorbid

Symptomatic treatment for pain with NSAIDS

Factitious Disorder

Patients simulate , induce or aggravate illness to receive medical attention, regardless of whether or not they are ill. They inflict painful ,deforming or even life – threatening injury on themselves , their children or other dependants. The primary motivation is not avoidance of duties, financial gain or anything concrete but simply to receive medical care and to partake in the medical system.

3 Types

- a) Factitious Disorder with predominantly psychological signs and symptoms
- b) Factitious Disorder with predominantly physical signs and symptoms
- c) Combined psychological + physical signs & symptoms

Males: Females – 2:1 (Factitious disorder with physical symptoms)

Females: Males- 3: 1 (Factitious disorder with psychological disorder)

Etiology

1 . Psychosocial Factors-

- a) Patients had suffered childhood abuse or deprivation , not able to form close relationships with parents.
- b) Masochistic personality traits
- c) Patients whose relative or family members die, patients hope to reunite with the diseased in a magical way by simulating the same behaviour.

2. Biological Factors

Treatment

It is laid on the lines of **managing the illness rather than the cure for illness.**

3 Goals:

- i) To reduce the risk of mortality & morbidity
- ii) To address the emotional needs or psychiatric diagnosis underlying factitious illness behaviour.
- iii) To be mindful of legal and ethical issues.

Ealy recognition of the illness plays important role in successful management.

Factitious disorder with physical signs & symptoms

Aka Washboard abdomen/Hospital addiction/polysurgical addiction.

1. Repeated hospital admissions feigning medical/surgical disorders so that they gain admission and stay in hospital.
2. Clinical presentations are so dramatic which results in giving excellent histories capable of deceiving even experienced clinicians.

Factitious disorder with psychological signs & symptoms

1. Psychiatric symptoms are feigned like hallucinations , depression , dissociative and abnormal behaviour
- 2 .Deception acts as an ego supporting device
- 3 .Pseudologia fantastica-Listeners interest pleases the patient and thus reinforces the symptom

Factitious Disorder by proxy

Person intentionally produces physical signs & symptoms in another person.

2 purposes identified by the patient

1. Indirectly assume the sick role
 2. Relieved of the caretaking role by having the child hospitalized
- Patient deceives through
 - a) False medical history
 - b) Contamination of laboratory samples
 - c) Alteration of records or induction of injury
 - d) Illness in the child

Malingering

It is the deliberate falsification of physical or psychological symptoms in an attempt to achieve a secondary gain .

It can be present during:

- i) Avoidance of criminal responsibility, trial and punishment

Criminals may feign insanity at the time of perpetration of the crime.

- ii) Financial gain

Seek financial gain in the form of undeserved disability insurance ,compensation, tort damages for purported psychological injury.

- iii) Avoidance of military services or hazardous duties

May malingering to avoid hazardous duties

iv) Avoidance of work , social responsibility
Maligner to avoid or escape from litigation-related consequences.

v) Admission to hospital

vi) Transfer from prison to hospital

Prevalence- 1% among mental health patients

In litigious context it increases to 10-20%

vii) Drug seekers

viii) Child custody

Differentiate Malingering vs Dissociative Disorder

Sl no	Malingering	Dissociative Disorder
1.	Suspicious, aloof , uncooperative	Friendly , cooperative, clinging
2.	Often avoid diagnostic evaluations & refuse recommended treatment	Welcome evaluation & treatment
3.	Provide extremely detailed history and descriptions of events precipitating their illness	Report historical gaps , inaccuracies

Pain Disorder

Aka Somatoform Pain Disorder / Psychogenic Pain Disorder

It is characterized by presence of and focus on pain in one or more site and is sufficiently severe to come to clinical attention. Psychological factors are necessary in the genesis, severity, or maintenance of the pain which causes significant distress, impairment or both.

Prevalence- 12%

Etiology

Associated with depressive disorder, Dysthymia

i) Psychodynamic factors

Patients experiencing body aches and pains without identifiable and adequate physical causes symbolically expressing an intrapsychic conflict through the body.

ii) Behavioural factors

Pain behaviours are reinforced when rewarded and are inhibited when ignored or punished.

iii) Interpersonal Factors

Conceptualized as a means for manipulating and gaining advantage in interpersonal relationships.

iv) Biological Factors

Cerebral cortex inhibits the firing of afferent pain fibres. Serotonin is main neurotransmitter in the descending Inhibitory pathways.

Clinical features

1. Disorder must have a psychological factor judged to be significantly involved in the pain symptoms and their ramifications.
2. Patient is completely preoccupied with their pain and cite it as the source of all misery.
3. Some patients complicate their problems by substance related disorders because they attempt to reduce the pain through the use of alcohol and other substances.
4. Defence mechanism – Displacement & Repression

Treatment

1. It is not possible to reduce the pain but address rehabilitation.
2. Clinicians should discuss the discuss of psychological factors early in treatment and should advocate patients that such factors are important in the cause and consequences of both physical and psychogenic pain.

Pharmacological

SSRI's & SNRI's

NAIDS for symptomatic pain

Non Pharmacological

Psychodynamic psychotherapy

Cognitive therapy

Group Psychotherapy

Psychogenic Excoriation:

Psychogenic Pruritus are lesions caused by scratching or picking in response to an itch or skin sensation or because of an urge to remove an irregularity on the skin sensation or because of an urge to remove an irregularity on the skin from pre-existing dermatoses such as acne.

Behaviour in psychogenic excoriation resembles obsessive compulsive disorder which manifests as **repetitive , ritualistic tension reducing and patient resist excoriating.**

Systemic Lupus Erythematosus

2 pathologic mechanisms have been proposed contributing to Neuropsychiatric SLE:

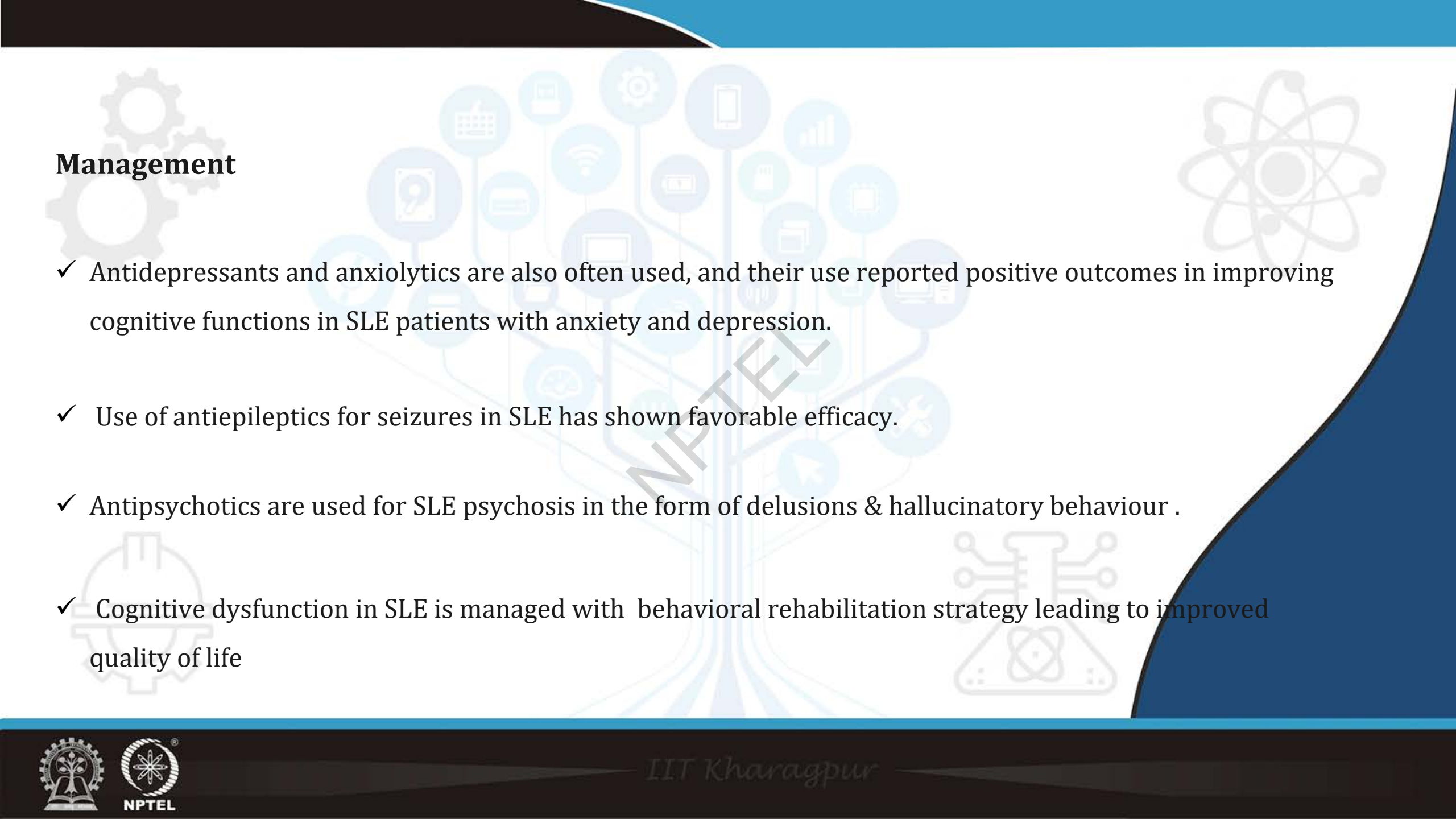
(1) Autoimmune or inflammatory pathway

Neuropsychiatric manifestations due to inflammatory mediators or autoantibodies with either intrathecal immune complex formation or disrupted blood-brain barrier (BBB).

(2) Ischemic or thrombotic pathway

It leads to cerebral microangiopathy, vascular occlusion, and haemorrhage.

Accelerated atherosclerosis, immune complex deposition, and immune-mediated vascular injury interplay plays a major role



Management

- ✓ Antidepressants and anxiolytics are also often used, and their use reported positive outcomes in improving cognitive functions in SLE patients with anxiety and depression.
- ✓ Use of antiepileptics for seizures in SLE has shown favorable efficacy.
- ✓ Antipsychotics are used for SLE psychosis in the form of delusions & hallucinatory behaviour .
- ✓ Cognitive dysfunction in SLE is managed with behavioral rehabilitation strategy leading to improved quality of life

CONCLUSIONS

- In this lecture we have discussed regarding the concepts of psychosomatic illness ,role of psychiatrist in evaluation of the various psychosomatic illness
- Concepts related to hypochondriasis , illness anxiety disorder , chronic fatigue syndrome , fibromyalgia , pain disorder , factitious disorder and malingering.

REFERENCES

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- 3 Text book Of psychiatry (Tasman & Leiber mann)
- 4 Stephen Stahl's Psychopharmacology
- 5 .Neuropsychiatric Systemic Lupus Erythematosus: A 2021 Update on Diagnosis, Management, and Current Challenges. Alexander Muacevic and John R Adler



**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 30 : Emergency Psychiatry

CONCEPTS COVERED

- Concepts of Suicide and its management
- Aggression/violence
- Catatonia & its management
- Drug induced Parkinsonism
- Serotonin Syndrome
- Neurolept Malignant Syndrome
- Lithium toxicity
- Withdrawal/intoxication related with substances

Emergency

- It is a set of circumstances in which a catastrophic outcome is thought to be imminent and the resources available to understand and deal with the situation are unavailable at the time and place of the occurrence.*

Evolution of medical emergencies

Nature of medical emergency system has been previously running on a **horizontal level** wherein the duty doctor (level I) refers the patient to emergency medicine specialists (level II) on site and then eventually to specialist (level III & ultimately (Level IV) on a hierarchical fashion.

However in due course of time it was replaced by **vertical system** that defines a facility for emergency care capability for specific critically ill patient groups namely, trauma, burn, cardiac patients and behavioural etc.

Goals of Emergency psychiatric care

- A. Triage: collect first line information, prioritize as per need and communicate/escalate appropriately to arrange further care
- B. Assessment: appropriate assessment to seek further diagnostic information pertinent to manage the presenting problem. A rapid understanding of etiology is a key skill required in this context.
- C. Diagnosis: accurate diagnostic workup is necessary for formulation and management
- D. Short term management: this involves options such as medication use, hospitalisation, seclusion/restraint and crisis social interventions and psychoeducation.
- E. Discharge planning: longer term interventions should be planned including preventative strategies aimed at averting crisis and rational follow-up strategies.

Types of Psychiatric Emergencies

General Syndromes

1. Suicide
2. Agitation/Aggression
3. Catatonia
4. Disinhibition
5. Withdrawal/Intoxication

Iatrogenic

1. Drug induced parkinsonism
2. Neurolept malignant syndrome
3. Serotonin syndrome
4. Lithium toxicity
5. Priapism

Suicide

NPTEL



Definition

WHO-states that

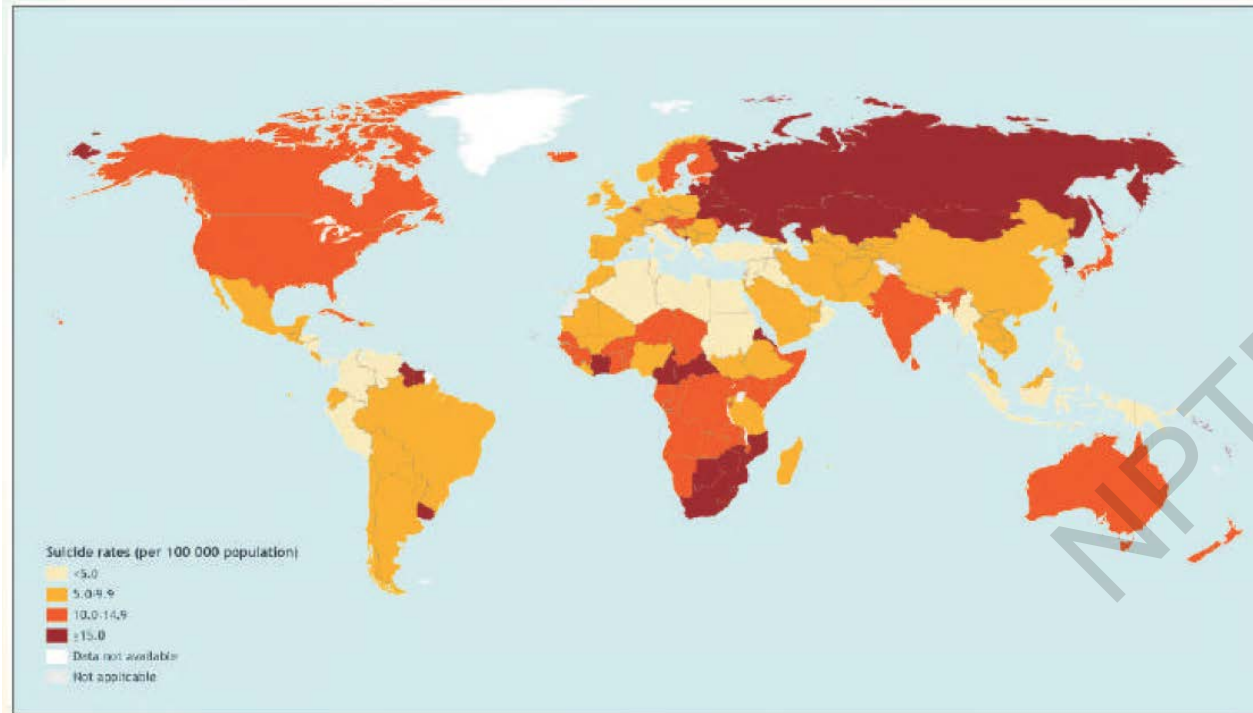
“Suicide is an act, deliberately initiated and performed by a person in the full knowledge or expectation of its outcome”

Deliberate self harm- Wilful self inflicting of painful, destructive, or injurious acts without intent to die.

Suicidal Intent – subjective expectation and desire for a self- destructive act to end in death.

Lethality of Suicidal behaviour-Objective danger to life associated with a suicide method or action

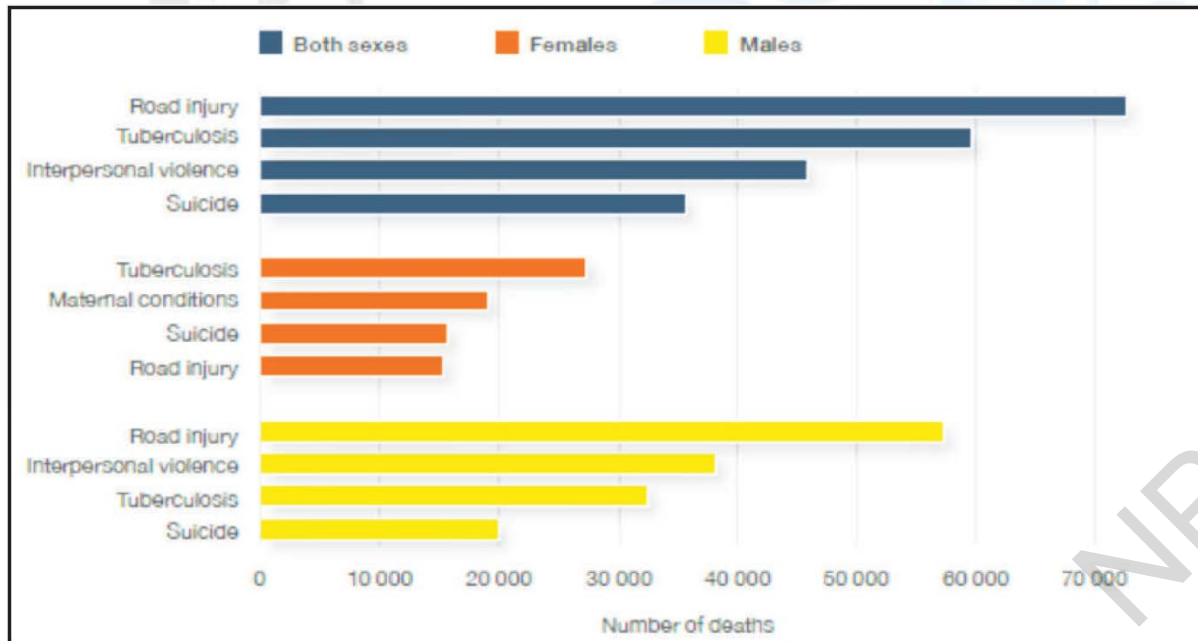
Suicide global scenario



✓ 703000 people died by suicide in the year 2019

✓ Suicide Rate 9 per 100,000 for 2019

Source: WHO 2019. Suicide worldwide in 2019: Global Health Estimates:
<https://www.who.int/publications-detail-redirect/9789240026643>



In india

a) No 1 cause of death among 15-29 yr age group

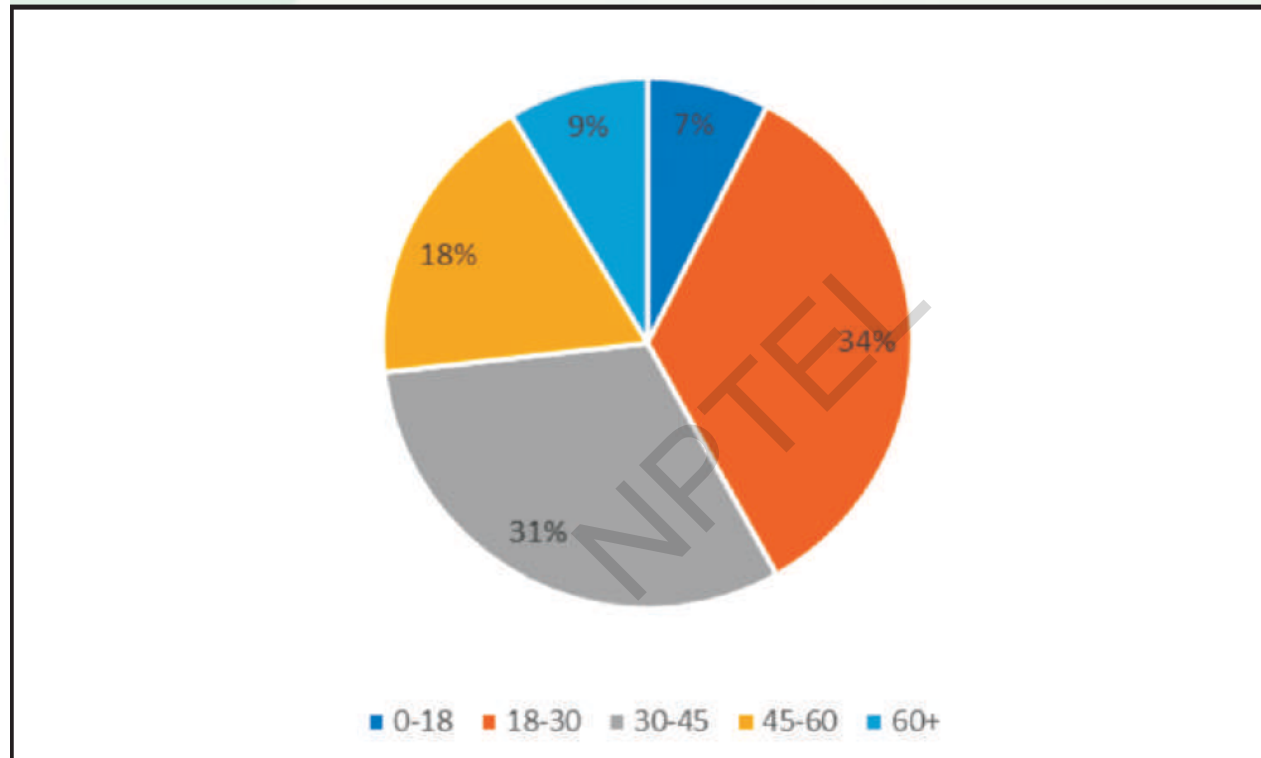
b) 1 in 4 men dying across the globe is from india

1 in 3 women dying across the globe is from india

C) Suicide rate increased from
10.2 to 11.3 per 100,000 population in last 3
years

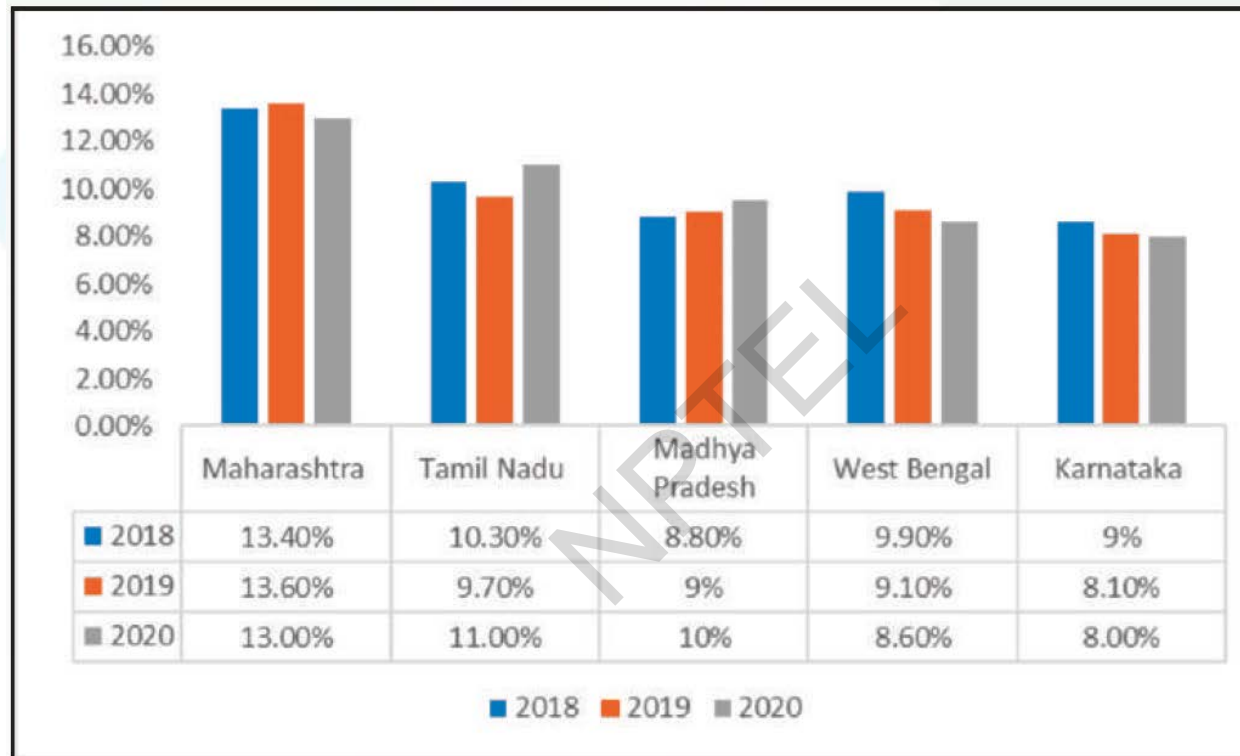
Source: WHO 2019. Suicide worldwide in 2019: Global Health Estimates:
<https://www.who.int/publications-detail-redirect/9789240026643>

Age distribution : 2020



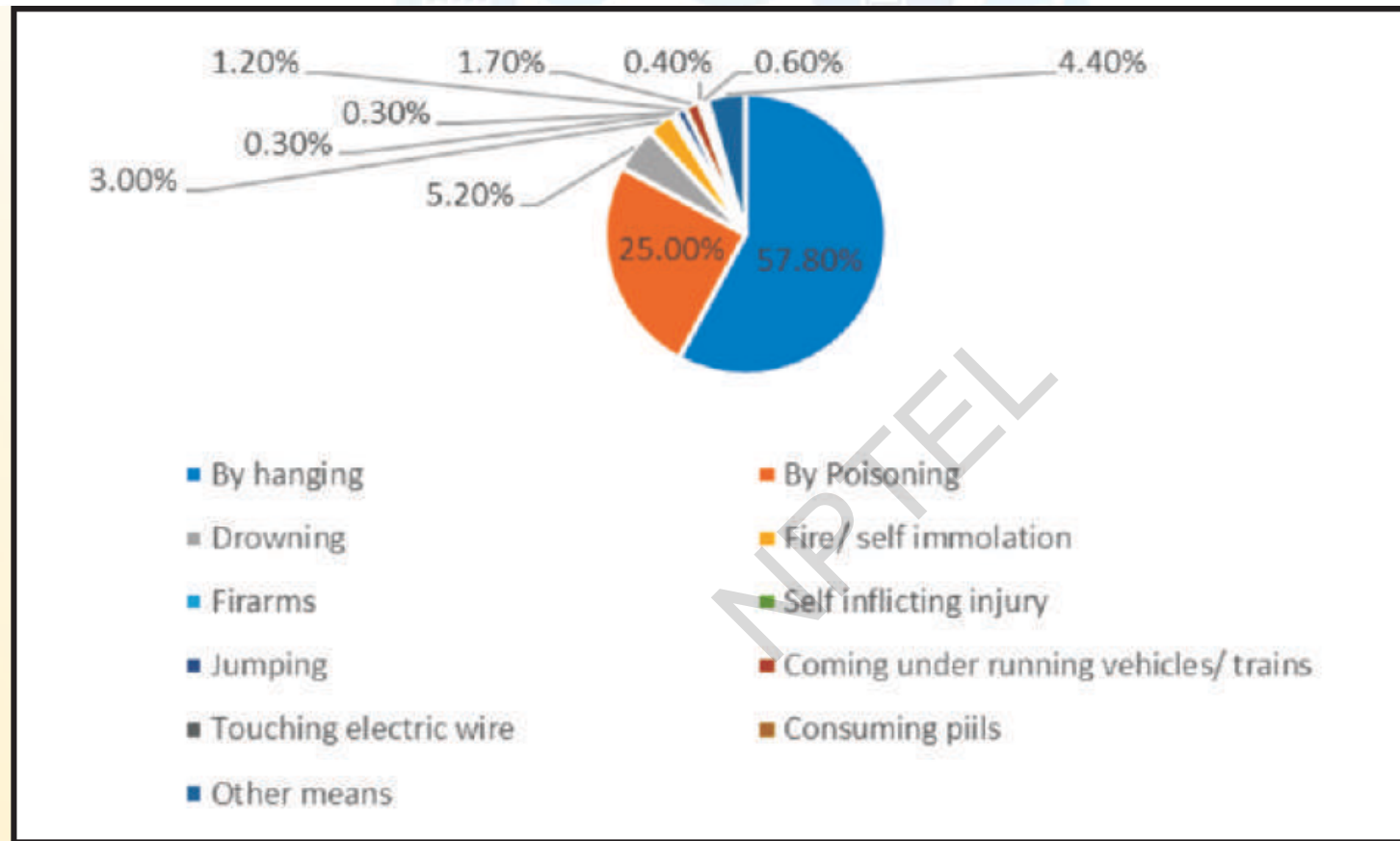
Source: National Crime Research Bureau (NCRB). Accidental deaths and suicide in India
New Delhi, India: Ministry of Home Affairs, Government of India:
https://ncrb.gov.in/sites/default/files/ADSI_2020_FULL_REPORT.pdf

Statewise distribution : Suicide



Source: National Crime Research Bureau (NCRB). Accidental deaths and suicide in India.
New Delhi, India: Ministry of Home Affairs, Government of India:
https://ncrb.gov.in/sites/default/files/ADSI_2020_FULL_REPORT.pdf

Methods of Suicide in India



Source: National Crime Research Bureau (NCRB). Accidental deaths and suicide in India. New Delhi, India: Ministry of Home Affairs, Government of India:
https://ncrb.gov.in/sites/default/files/ADSI_2020_FULL_REPORT.pdf

Risk Factors for Suicide

Bio-Psychosocial Factors

Psychiatric illnesses (particularly depression, bipolar disorder, schizophrenia, anxiety disorders and certain personality disorders)

Alcohol and other substance use disorders

Aggressive and impulsive tendencies

History of trauma or abuse Family history of suicide or previous attempted suicide

Major Physical illness

Environmental Factors

Job or financial loss

Relationship or social loss

Domestic and other violence

Child abuse

Easy access to lethal means

Interpersonal difficulties/ conflicts

Local clusters of suicide may have a contagious effect

Sociocultural Factors

Lack of social support and sense of isolation

Stigma associated with help-seeking behavior for mental disorders

Barriers in accessing health care, especially mental health and substance abuse treatment

Cultural and religious beliefs (for instance, the belief that suicide is a noble resolution of a personal dilemma)

Exposure to suicide, either through sensationalization by the media, or the influence of others who have died by suicide

Risk Factors associated with suicide

1. Gender differences
2. Age
3. Marital status
4. Occupation
5. Physical health
6. Mental health
7. Psychiatric illness
8. Alcohol dependence
9. Personality Disorders
10. Previous Suicidal behaviour - **Risk of 2nd attempt is highest within 3 months of 1st attempt**

Etiology

I. Sociobiological

a) Egoistic b) Altruistic c) Anomic

II. Psychological

III. Biological

IV. Genetic

Assessment of suicide

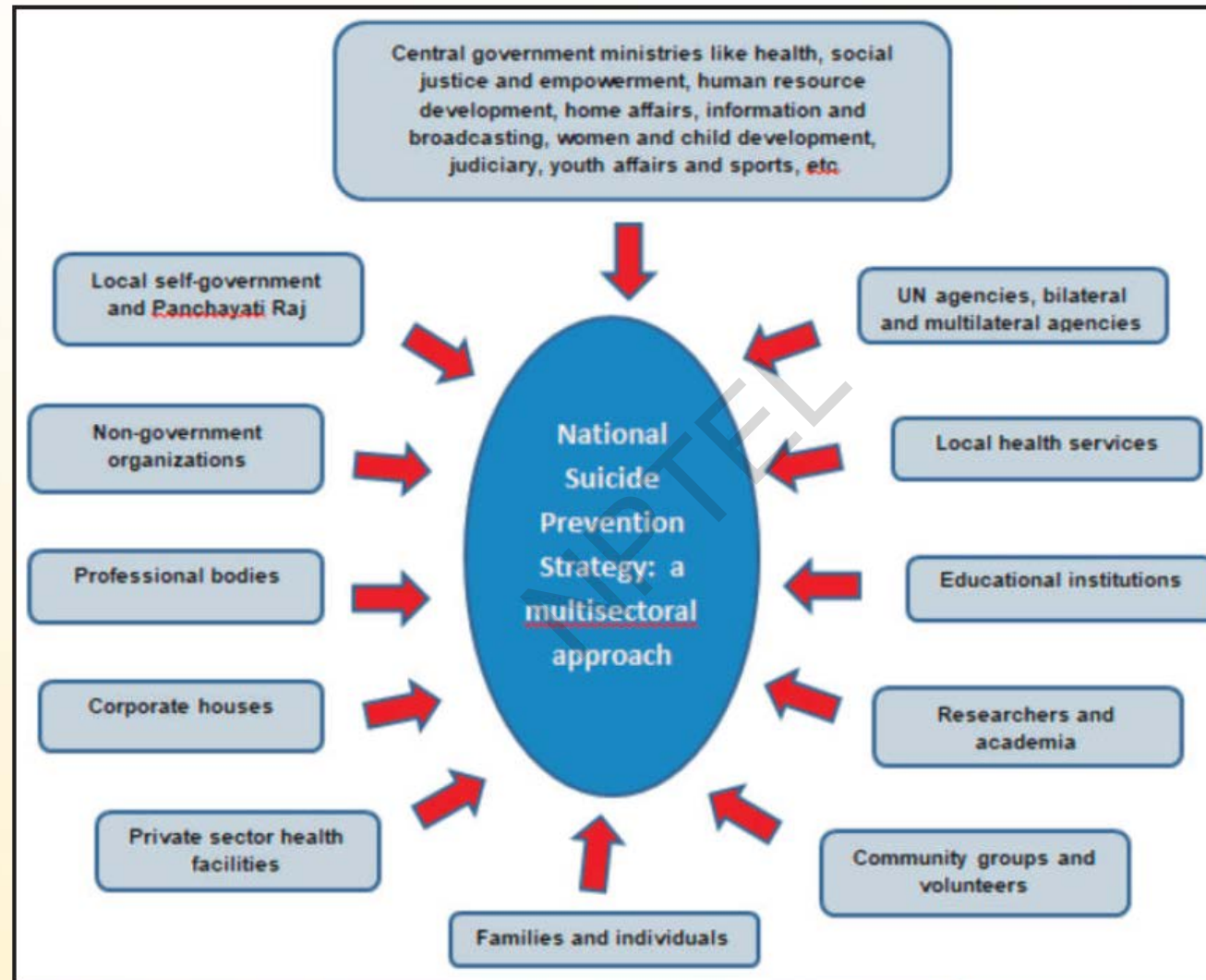
Scales commonly used for assessment of suicide

- i) **Becks Suicide Intent Scale – Developed by Aaron T Beck of University of Pennsylvania.**
- ii) **Columbia Suicide Severity rating scale- developed by John Mann of New York State Psychiatric Institute**

Goals to reduce suicide

- ✓ Promote awareness that suicide is a public health problem that is preventable.
- ✓ Develop & implement strategies to reduce stigma associated with a consumer of mental health , substance abuse & suicide prevention services.
- ✓ Promote efforts to reduce access to lethal & methods of self harm.
- ✓ Develop & promote effective clinical & professional practices.
- ✓ Improve reporting & portrayals of suicidal behaviour , mental illness & substance abuse in the entertainment & news media
- ✓ Promote & support research on suicide & suicide prevention.
- ✓ Improve & expand surveillance systems

National Suicide prevention Strategy



Aggression / violence



NPTEL

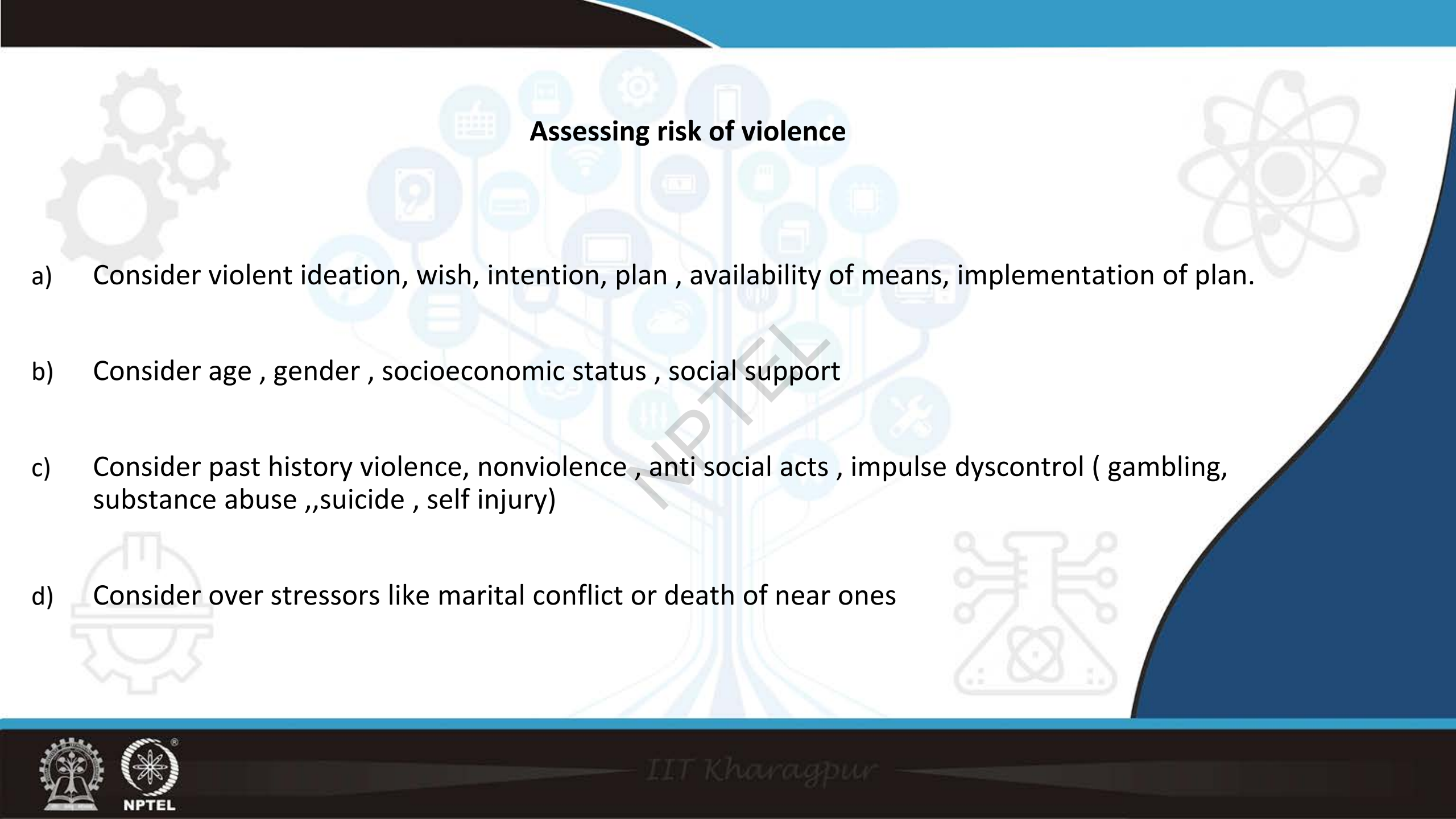


Aggression/Violence

Assessing & predicting violent behaviour

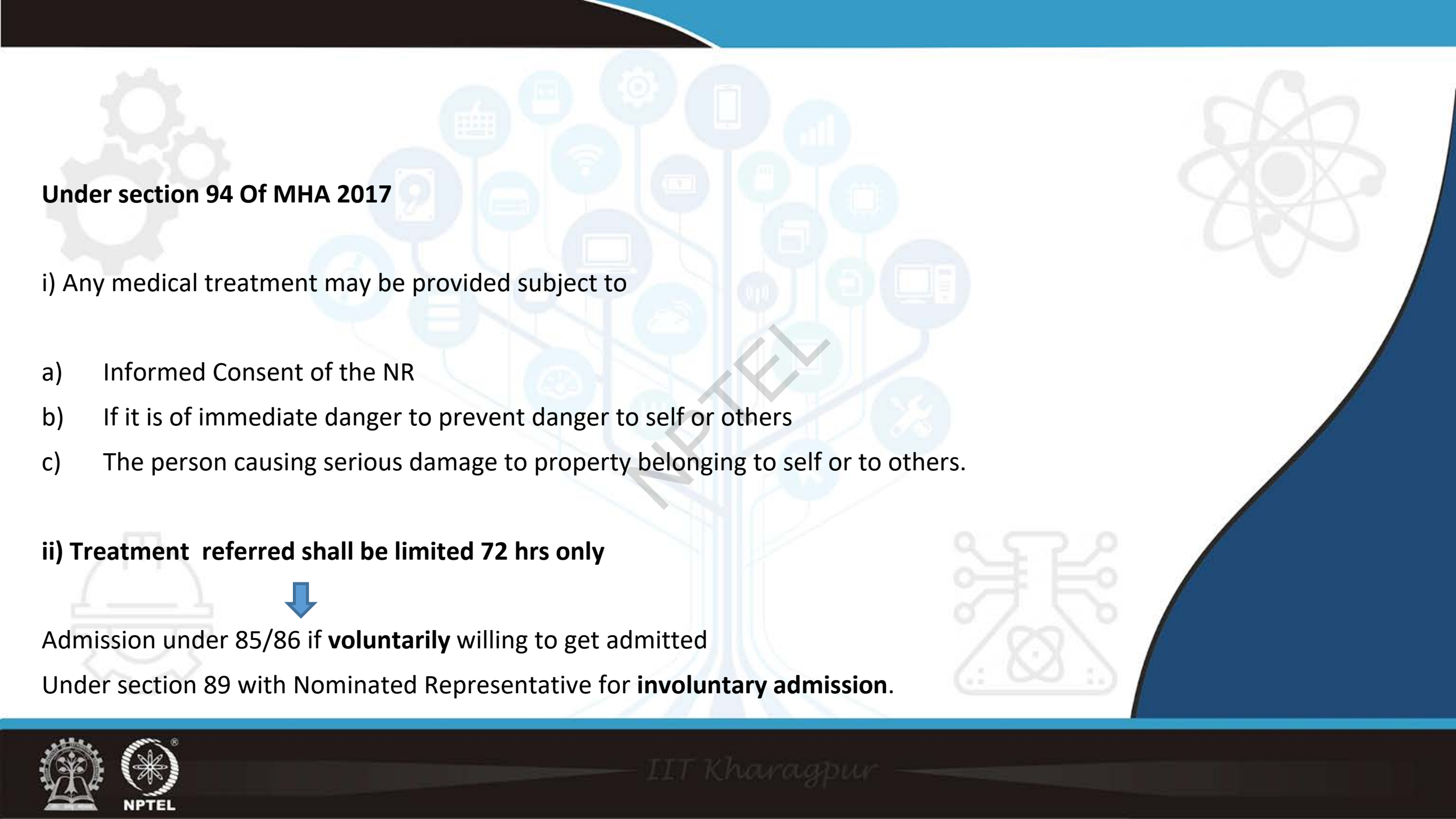
I Signs of recent violence

- a) Very recent acts of violence, including property violence
- b) Verbal or physical threats
- c) Carrying weapons or other objects that may be used as weapons eg (forks , ash trays)
- d) Progressive psychomotor agitation
- e) Alcohol or drug intoxication / Paranoid features in psychotic patients
- f) Command auditory hallucinations / Organic mental disorders global or frontal lobe findings
- g) Catatonic excitement
- h) Patients with mania
- i) Patients with agitated depression / Personality disorders prone to rage , violence



Assessing risk of violence

- a) Consider violent ideation, wish, intention, plan , availability of means, implementation of plan.
- b) Consider age , gender , socioeconomic status , social support
- c) Consider past history violence, nonviolence , anti social acts , impulse dyscontrol (gambling, substance abuse ,,suicide , self injury)
- d) Consider over stressors like marital conflict or death of near ones



Under section 94 Of MHA 2017

- i) Any medical treatment may be provided subject to
 - a) Informed Consent of the NR
 - b) If it is of immediate danger to prevent danger to self or others
 - c) The person causing serious damage to property belonging to self or to others.

ii) Treatment referred shall be limited 72 hrs only



Admission under 85/86 if **voluntarily** willing to get admitted

Under section 89 with Nominated Representative for **involuntary admission**.

Catatonia

As defined by DSM-V

Presence of 3 or more of the following

- a) Catalepsy
- b) Waxy flexibility
- c) Stupor
- d) Agitation
- e) Mutism
- f) Negativism
- g) Posturing
- h) Mannerism
- i) Stereotypies
- j) Grimacing
- k) Echolalia
- l) Echopraxia

Stupor- No Psychomotor activity and no reactivity to the activity

Automatic obedience- Subject does whatever is asked of him /her despite not to do.

Ambitendence- Patient makes alternating co-operation and opposition

Aversion- Turns away from the examiner when addressed

Echolalia- Subject repeats the words & phrases

Echopraxia- Subjects imitates the actions/ movements of the examiner

Repetition of response after the withdrawal of the stimulus.

Mitmachen-Body can be put into any posture despite instructions to resist

Mitgehen-Extreme form of mitmachen when slightest pressure results in movement in any direction

Waxy flexibility- Smooth resistant muscle tone is felt to initiate movement.

Geganzhalten- A form of negativism in which resistance to passive movement increases with force exerted.

Catalepsy-If a subject's body posture is left in an awkward posture and left, the posture is held for a period before slowly relaxing despite asking the patient to relax



Waxy Flexibility

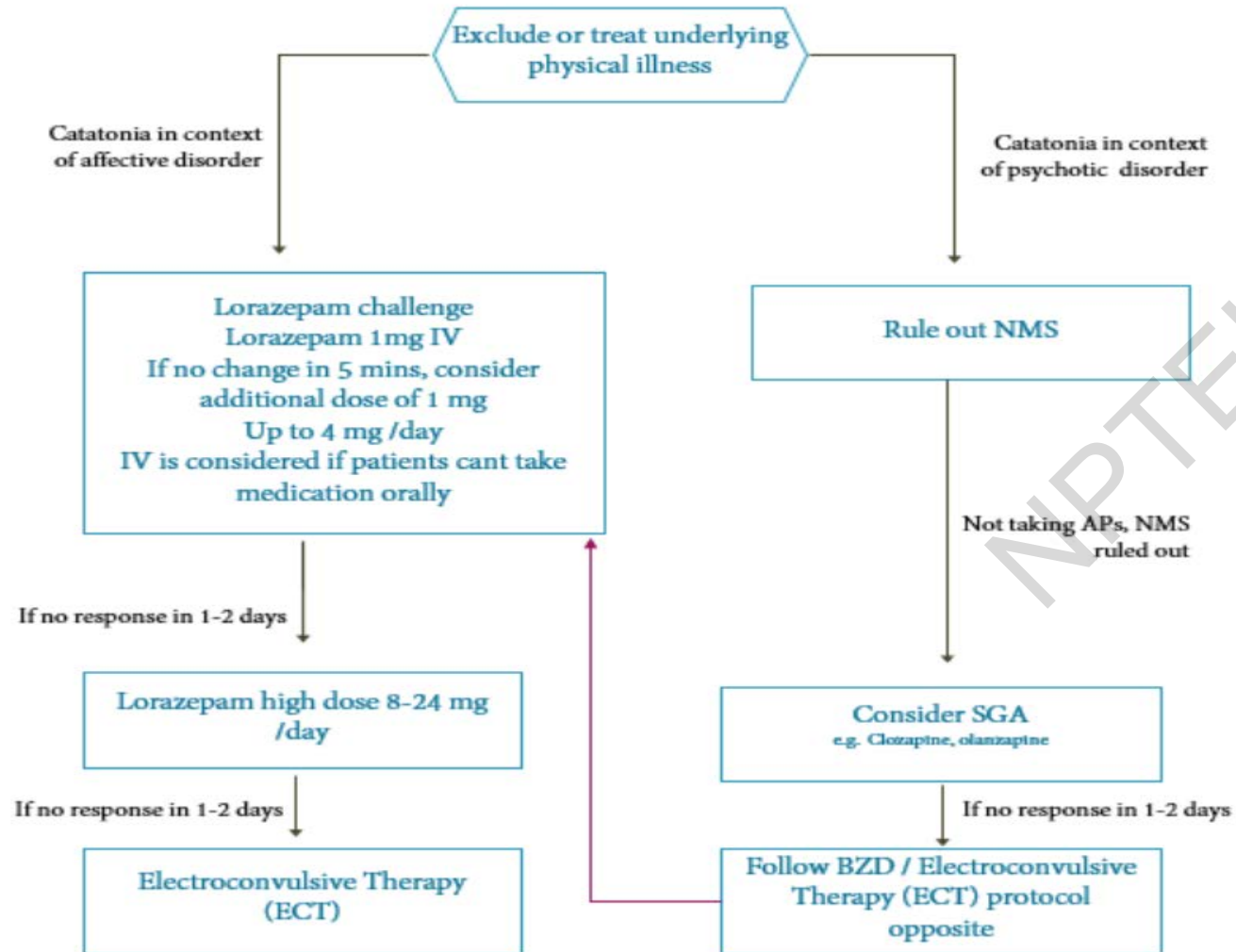


Psychological Pillow – Catatonia

Classification : Taylor & Fink 2003

- 1 Non malignant (kahlbaum)- Positive response to BZD
- 2 Delirious- Requires high dose BZD, respond to ECT and worsens with antipsychotics
- 3 Malignant Features similar to NMS

Treatment Algorithm for Catatonia

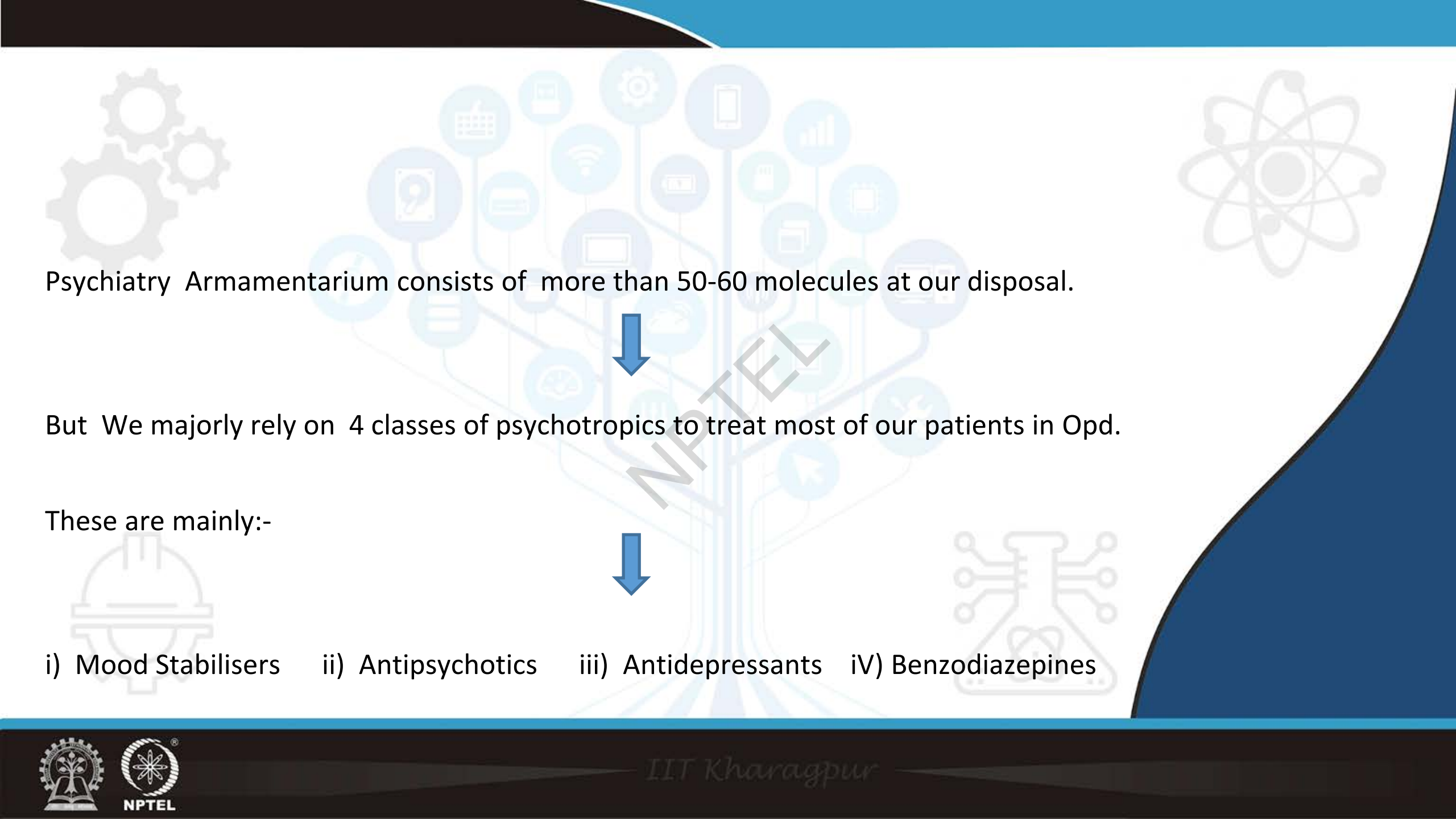


Iatrogenic Emergencies



NPTEL





Psychiatry Armamentarium consists of more than 50-60 molecules at our disposal.



But We majorly rely on 4 classes of psychotropics to treat most of our patients in Opd.

These are mainly:-



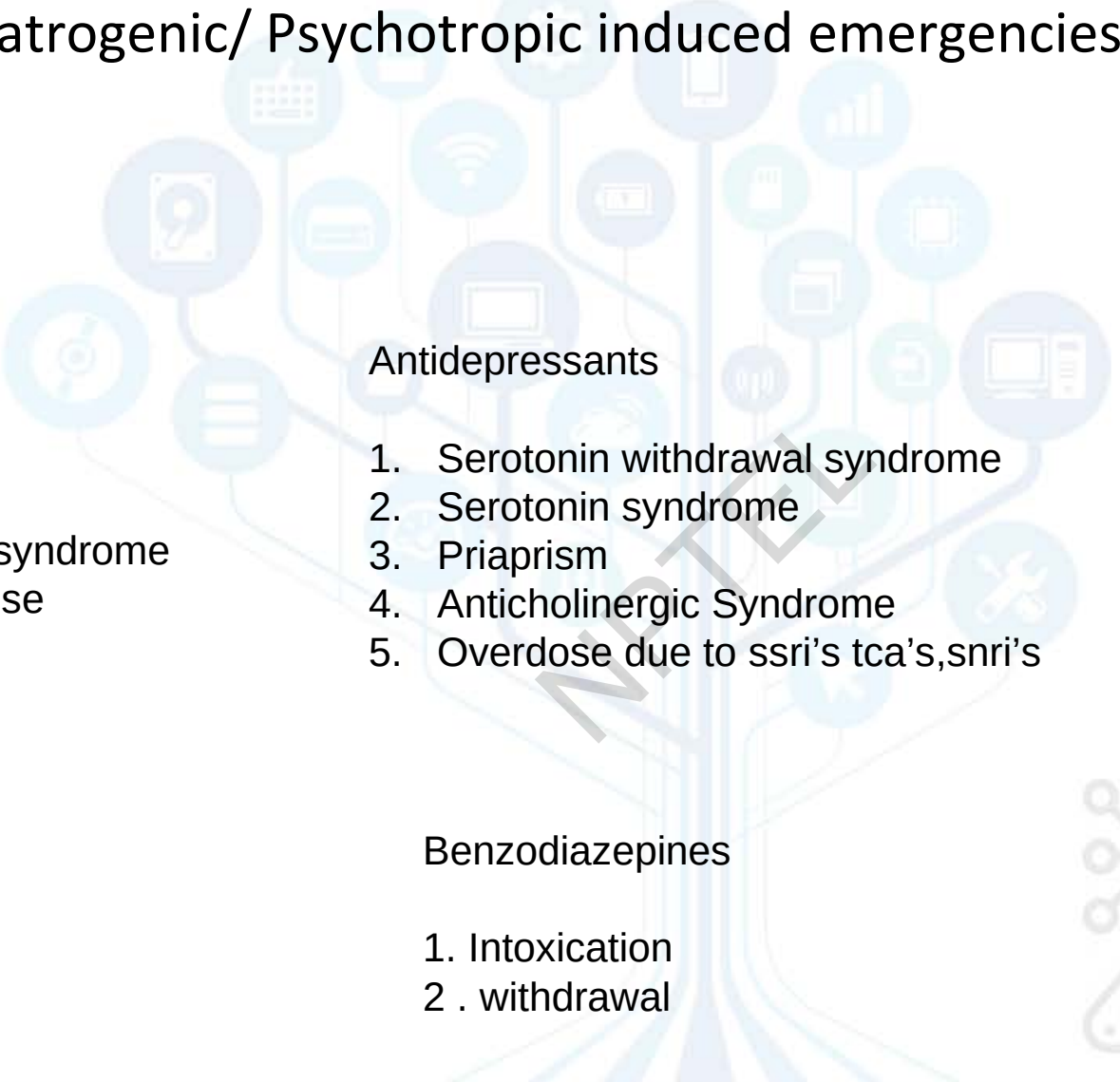
i) Mood Stabilisers ii) Antipsychotics iii) Antidepressants iv) Benzodiazepines

Iatrogenic/ Psychotropic induced emergencies



Antipsychotics

1. Acute dystonia
2. Akathasia
3. Neurolept malignant syndrome
4. Antipsychotic Overdose



Antidepressants

1. Serotonin withdrawal syndrome
2. Serotonin syndrome
3. Priapism
4. Anticholinergic Syndrome
5. Overdose due to ssri's tca's,snri's



Mood stabilisers

1. Lithium toxicity
2. Valproate toxicity



Benzodiazepines

1. Intoxication
- 2 . withdrawal

Antipsychotics Related emergencies



Dystonia is defined by involuntary maintained contraction of agonist and antagonist muscles yielding abnormal posturing, twisting and repetitive movements, or tremulous and can be initiated or worsened by attempted movement.

It assumes a pattern of

- i) overextension or over-flexion of the hand
- ii) Inversion of the foot
- iii) Lateral flexion or retroflexion of the head,
- iv) Torsion of the spine with arching and twisting of the back
- v) Forceful closure of the eyes, or a fixed grimace.

It may come to an end when the body is in action and during sleep.

Presentation at Emergency



- Oro-mandibular dystonia can manifest as night-time bruxism (jaw spasms producing forceful jaw closure) and may follow dental procedures, oro-mandibular-facial trauma, or temporomandibular joint dysfunction.
- Spasmodic dysphonia can commence as a strained voice. Despite the name dystonia, the tone of affected extremities at rest is often normal at the onset.
- In cervical dystonia, head tremor sometimes decreases with the head held in a specific position, and displacement from that position, such as turning the head to one side, may worsen it.

Management

Medications that are most potent in the treatment of dystonia include

Central Anticholinergics (trihexyphenidyl ,procyclidine , biperiden,)

Peripheral Anticholinergics (Promethazine)

GABA agonists (baclofen and benzodiazepines)

Dopaminergic agents such as bromocriptine etc.

The mechanism of action of these drugs is due to modifications in dopaminergic and cholinergic neurotransmission and reduced GABA-mediated inhibition in the dystonic central nervous system (CNS).

Akathisia

Akathisia is defined as an inability to remain still.

It is a neuropsychiatric syndrome that is associated with psychomotor restlessness.

The individual with akathisia will generally experience an intense sensation of unease or an inner restlessness that usually involves the lower extremities.

Akathisia may appear soon after the antipsychotic has been started or may appear when the dosage is increased.

Treatment

Beta-blockers such as propranolol and benzodiazepines have been used for the treatment of akathisia.

Managed by reducing the dose of the offending agent or switching to an alternative antipsychotic agent.

Drug induced Parkinsonism

Tremors - Drugs notorious to cause are selective serotonin (and norepinephrine) reuptake inhibitors (SSRIs/SNRIs) amitriptyline, lithium, valproate, β -adrenoceptor agonists, dopamine receptor antagonists etc and are managed by reducing the dose of offending agent and with the help of B blockers.

Dystonia- Develops within hours to days of initiation. Presents as muscle spasm, stiffness, oculogyric crisis)

Akathisia - (Develops within days to months of use of haloperidol - characterized by restlessness.)

(Tardive Dyskinesia - (Develops after years, presents as a chore, especially orofacial region)

managed by tetrabenazine VMAT2 receptor blocker

Neurolept Malignant Syndrome

- ❑ It affects 0.01 %- 3.2 % of patients taking neuroleptic medication.
- ❑ M:F - 2:1
- ❑ Young population due to involvement of antipsychotics in early age group.

Pathophysiology

1. Sudden reduction in central dopaminergic activity due to a D2 receptor blockade
- 2 . Abrupt withdrawal of D2 receptor stimulation.
3. Develops NMS within hours or days after exposure to a causative drug, with most exhibiting symptoms within 2 weeks and nearly all within 30 days
4. Mental status changes, Fever, Muscle rigidity, and Autonomic Instability.



Medications

Antipsychotics

Haloperidol

Fluphenazine

Chlorpromazine

Risperidone

Clozapine

Amisulpiride

Drugs causing NMS

Antiemetics

Domperidone

Metoclopramide

Prochlorperazine



A.	Development of severe muscle rigidity and elevated temperature associated with the use of neuroleptic medication.
B.	Two (or more) of the following: (1) Diaphoresis (2) Dysphagia (3) Tremor (4) Incontinence (5) Changes in level of consciousness ranging from confusion to coma (6) Mutism (7) Tachycardia (8) Elevated or labile blood pressure (9) Leucocytosis (10) Laboratory evidence of muscle injury (eg, elevated CPK)
C.	The symptoms in criteria A and B are not due to another substance or a neurological or other general medical condition.
D.	The symptoms in criteria A and B are not better accounted for by a mental disorder



Treatment

- NMS is a Neurologic Emergency delay in diagnosis and treatment can lead to significant morbidity or death.
- Cessation of the suspected offending neuroleptic pharmacologic agent.
- Treatment of hyperthermia(Fever) with cooling blankets or ice packs to the axillae and groin may be needed.
- Aggressive hydration is often required, to prevent disseminated intravascular coagulation (DIC) secondary to rhabdomyolysis, deep venous thrombosis especially if highly elevated CPK levels threaten to damage the kidneys.
- All dopamine agonists given to reverse the hypodopaminergic state.

- Bromocriptine Mesylate, a dopamine agonist,
PO /NG tube starting dose 2.5mg twice /thrice daily Max-45mg.
- Amantadine starting dose -100mg twice/thrice daily Max- 600mg
- Dantrolene a muscle relaxant that works by inhibiting calcium release from the sarcoplasmic reticulum.
starting dose PO 50mg -200mg.
- Benzodiazepines, which can be helpful in controlling agitation but may also ameliorate symptoms and hasten recovery in milder cases.

Antipsychotic Overdose



NPTEL



Antipsychotic Overdose

- 'Antipsychotics,' Aka “Neuroleptics “are a broad category of medications initially developed to treat psychosis arising from a large spectrum of psychiatric and organic causes .
- Other indications such as headaches, Tourette syndrome, and post-herpetic neuralgia.
- Neuroleptic medications can be broadly divided into two categories.

Based largely on their pharmacologic properties, and, therefore, their side effect profiles:

First-generation antipsychotics (FGAs), also known as

Second-generation antipsychotics (SGAs), termed

‘Typical’ antipsychotics

‘Atypical antipsychotics

Side effect Profile

Sedation -	Due to Histaminergic receptors
Weight gain -	Due to Histaminergic receptors, 5HT _{2C} (Atypical)
Erectile dysfunction in male -	Decrease in serotonin ,dopamine
Oligomenorrhea or amenorrhea in female -	(decreases dopamine thereby increasing prolactin)
Orthostatic hypotension -	alpha 1 blocking action
Constipation -	due to cholinergic receptors
ECG changes -	(QT prolongation, torsades de pointes-PSVT)

Sedation: Chlorpromazine> Quetiapine> Olanzapine> Risperidone>Haloperidol

Hypotension: Clozapine>Risperidone>Quetiapine> Aripiprazole

Q T Prolongation: Antipsychotics

Cariprazine , lurasidone ---→ aripiprazole, clozapine , olanzapine--→ amisulpride, haloperidol, quetiapine--→pimozide

Treatment for toxicity



NPTEL



No specific antidote and Supportive treatment is the mainstay of Antipsychotic toxicity.

Consider gastric lavage or induction of emesis as soon as possible and followed by the administration of activated charcoal.

Maintenance of Airway, Breathing, and circulation are critical factors for survival.

A patent airway is ensured by using an oropharyngeal airway or endotracheal tube.

Comatose patients with a difficult airway or upper airway obstruction require a tracheostomy.

Supplemental oxygen is administered via nasal prongs or facemask.

Patients with refractory hypoxia often require intubation and mechanical ventilation.

Hypotension and circulatory collapse need aggressive treatment with intravenous fluids, concentrated albumin, and vasopressor agents such as norepinephrine or phenylephrine.



Extrapyramidal reactions such as acute dystonia are treated with Central anticholinergics.



ECG and vital signs require regular monitoring, especially for signs of torsades de Pointes or QT prolongation, or dysrhythmias.



Cardiac monitoring should be in place until the ECG becomes normal.

Antidepressants Related Emergencies



SSRI discontinuation Syndrome

- Abrupt discontinuation of SSRI with shorter half life Paroxetine , fluvoxamine

Dizziness
Weakness
Nausea
Headache
Rebound depression
Anxiety
Insomnia
Poor Concentration
Paraesthesias
Migraine like symptoms

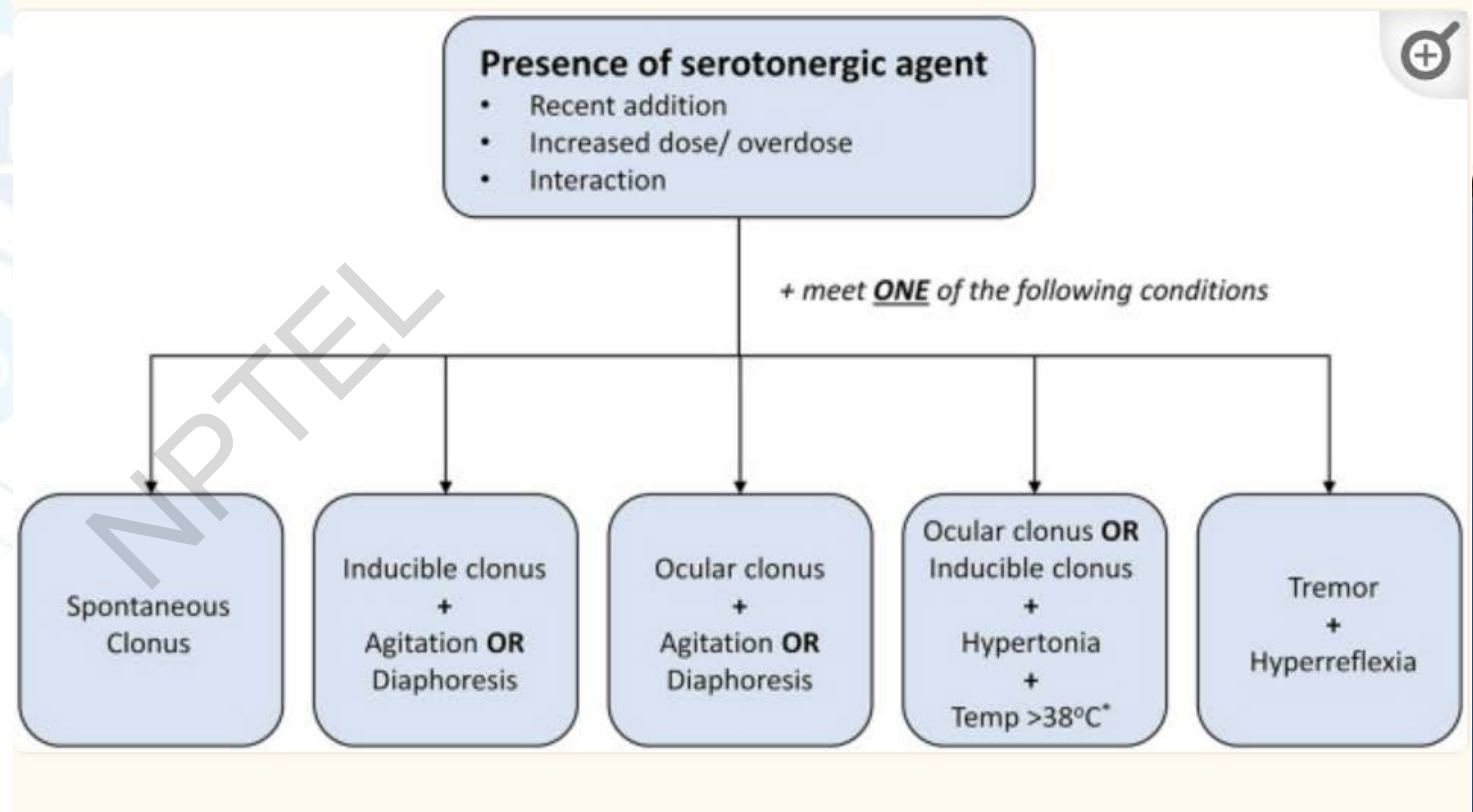
Treatment

Restart the offending agent
Fluoxetine with max half life of 1 week is substituted with the offending agent

**Persons who experience adverse effects
in first week of taking SSRI
generally experiences discontinuation syndrome**

Serotonin Syndrome

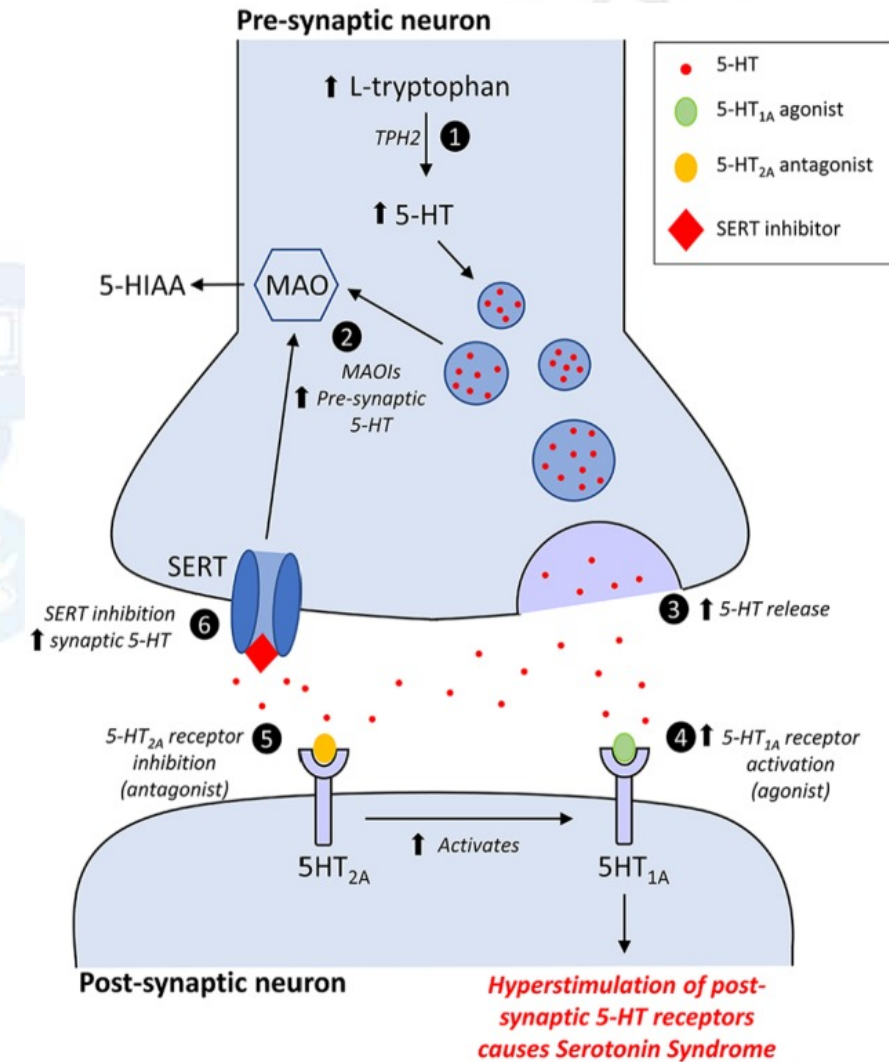
- Triad of Altered Mental Status, Autonomic Hyperactivity, and Neuromuscular Abnormalities .
- Incidence - 0.23%.
- Symptoms in Serotonin Syndrome usually begin within 24 hours of ingestion of the causative agent and that most patients seek medical advice within 6 hours.
- Hunter's Criteria Gold standard for diagnosing Serotonin Syndrome.



Pathophysiology

Mechanisms of serotonin syndrome:

- (1) Increased levels of L-tryptophan will lead to increased levels of endogenous 5-HT; a step catalyzed by the enzyme tryptophan hydroxylase 2 (TPH2)
- (2) Increased presynaptic concentrations of 5-HT due to inhibition of serotonin metabolism by MAOIs.
- (3) Increased 5-HT release by drugs including amphetamines and their derivatives, cocaine, MDMA, and levodopa
- (4) Direct or indirect activation of postsynaptic 5-HT_{1A} receptors.
- (5) Increased synaptic levels of 5-HT due to inhibition of the SERT by reuptake inhibitors such as SSRIs, and TCAs.



Clinical Features: Serotonin Syndrome

Mild- Normothermic, with Mild Autonomic Symptoms .

Moderate - Hyperthermia ($>40^{\circ}\text{C}$), Eye movement abnormalities, Agitation, pressured speech, and Hypervigilance.

Severe - Temperature greater than 41.1°C , haemodynamic/autonomic instability, hyperactive bowel sounds, delirium, and muscle rigidity.

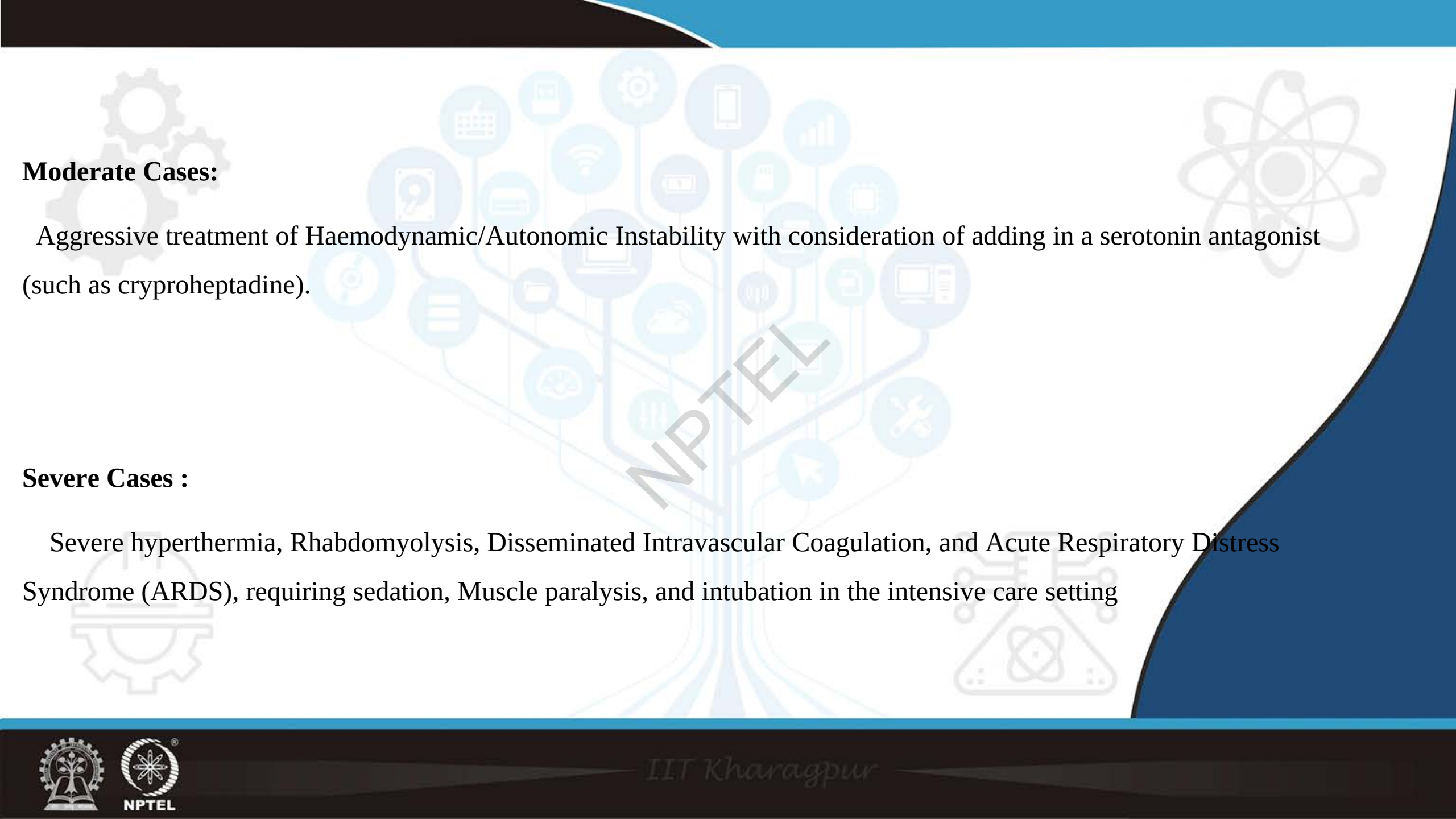
Complications in severe cases include seizures, renal failure, metabolic acidosis, rhabdomyolysis, disseminated intravascular coagulation, acute respiratory distress syndrome, respiratory failure, and even death.

Treatment

- First step is to identify and stop the offending serotonergic medication.
- Supportive care to stabilize vital signs (maintenance of oxygen saturations, intravenous fluids, and continuous temperature cardiac monitoring).
- Sedation with benzodiazepines (such as diazepam or midazolam/lorazepam).
- In severe cases, consideration of serotonin antagonists alongside muscle paralysis.

In Mild Cases:

In mild cases, discontinuation of the offending medication, sedation with benzodiazepines and supportive care is often sufficient.



Moderate Cases:

Aggressive treatment of Haemodynamic/Autonomic Instability with consideration of adding in a serotonin antagonist (such as cyproheptadine).

Severe Cases :

Severe hyperthermia, Rhabdomyolysis, Disseminated Intravascular Coagulation, and Acute Respiratory Distress Syndrome (ARDS), requiring sedation, Muscle paralysis, and intubation in the intensive care setting

Priapism

Priapism is defined as prolonged and persistent erection of the penis without sexual stimulations. (Duration >4 hrs)

2 Types

- i) **Non ischemic type** (arterial, high flow) that can be treated conservatively .
- ii) **Ischemic type** (veno occlusive, low flow) a urological emergency that requires immediate intervention

Nonischemic priapism is associated with penile or perineal trauma, cocaine, metastatic malignancy

Ischemic priapism can be caused by drugs, haematological disorders, metabolic disorders, alcohol.

Drug-induced priapism is associated with antidepressants, antipsychotics, antihypertensive medications, and accounts for approximately 15% to 41% of all cases, of which antidepressants-induced priapism is most common .

Treatment

- 1.Oral pseudoephedrine
- 2.Penile local anesthetic block
- 3.Aspiration of the corpora

Intracorporal injections of diluted phenylephrine (100 mcg - 500 mcg/ml) every 5 minutes x 3

- 4.Surgical cavernosal - spongiosum shunt
- 5.Corporal dilation with or without immediate penile prosthesis placement

Anticholinergic Syndrome

Medications with anticholinergic properties include antidepressants, antiparkinson drugs, antipsychotics,

Common physical exam findings

Flushing, anhidrosis, dry mucous membranes, mydriasis, altered mental status, fever, urinary retention, Decreased bowel movements.

Central nervous system effects may include delirium, hallucinations, agitation, restlessness, confusion, and picking at clothing and bedding.

Seizures and jerking movements are possible

Treatment

1. Physostigmine is usually only given in the case of both peripheral and central signs and symptoms of anticholinergic poisoning.
2. Maintain Hydration.
3. ECG monitoring (TCA's prone to QT prolongation)

Antidepressant Overdose



NPTEL



Tricyclic Antidepressants

- These are pharmacological agents used since 1950's for the treatment of depression.
- Other indications includes Migraine prophylaxis, neuralgic pain, treatment of obsessive compulsive disorders.
- Ever since with the advent of selective serotonin reuptake inhibitors ,the reliance on TCA's have reduced but we cannot run away with the fact that TCA's still used for various indications in the treatment of psychiatric illness
- Despite of the reduced usage the data suggest that TCA's overdose and toxicity cases are flooded in the hospitals preferentially in the ICU , emergency rooms
- This demands us to be aware of the protocol for effective management of the patients

Side effects of Amitryptaline (TCA)

Mechanism of action :

By inhibiting presynaptic reuptake of **norepinephrine and serotonin** in the central nervous system (CNS).

TCAs block - peripheral alpha-adrenergic, histaminic, muscarinic, and central serotonin receptors

1. **Seizures results from high doses**
2. **Blockade of alpha-adrenergic receptors can cause hypotension.**
3. **Blockade of muscarinic receptors can cause anticholinergic toxicity, such as tachycardia, fever, dry mouth and skin, decreased bowel sounds, and altered mental status.**
4. **Blockade of histamine receptors can also cause altered mental status, weight gain , sedation.**
5. **QT prolongation seen in cases of TCA overdose** occurs due to potassium channel blockade that may potentially cause torsades de pointes (paroxysmal ventricular tachycardia)

Management

Proper management of airway, breathing, and circulation is critical in cases of TCA poisoning.

Charcoal decontamination may be effective up to 2 hours' post-ingestion, especially if the bowel sounds are diminished.



In cases of Seizures, benzodiazepines are used, but in cases of refractory seizures, prompt administration of anticonvulsants, such as phenobarbital or propofol, or even general anesthesia, should be considered.



Sodium bicarbonate should be given to hemodynamically unstable patients, patients with seizures, and patients with QRS prolongation of more than 100 msec.



The aim of this therapy is to narrow the QRS and to keep the serum pH between 7.5 and 7.55.

Treatment

- Alpha-adrenergic agents, such as norepinephrine, should be used for cases not responding to hypotension.



- Since TCA are highly protein bound with an extensive volume of distribution, enhanced elimination with dialysis and hemoperfusion is not effective.

SSRI (sertraline)& SNRI's (venlafaxine)

Sertraline

➤ Mechanism of action :

Blockade of Reuptake of serotonin from the synaptic cleft.

- The Hunt serotonin toxicity criteria decision rule describes 5 constellations of symptoms that define serotonin toxicity
 1. Spontaneous clonus
 2. Inducible clonus and agitation or diaphoresis
 3. Ocular clonus with agitation or diaphoresis
 4. Tremor and hyperreflexia
 5. Ocular clonus, hypertonicity, and temperature greater than 38 C

Venlafaxine

- Mechanism of action :

Venlafaxine works by increasing serotonin levels, norepinephrine, and dopamine in the brain by blocking transport proteins and stopping their reuptake at the presynaptic terminal.

Venlafaxine is essentially a selective serotonin reuptake inhibitor at 75 mg, and with higher doses such as 225 mg/day.

- **Symptoms of an overdose of venlafaxine can include:**

Tachycardia, unusual sleepiness, dilated pupils, seizures, vomiting, cardiac arrhythmias, hypotension, muscle aches or pains, or dizziness.

Management

- Patients who were experiencing minor symptoms can be managed at home in conjunction with a doctor or with a regional poison control center.
- Treatment is largely supportive and involves discontinuation of all serotonergic therapies, control of agitation with benzodiazepines and defervescence.
- Hypertonicity, rigidity, and high-grade fever are evident in severe cases of serotonin toxicity where patients require endotracheal intubation and ventilatory support.
- Muscular rigidity is thought to be the primary cause of hyperthermia and death so patients with severe symptoms should be rapidly cooled.
- If benzodiazepines do not sufficiently control muscle spasm or temperature is greater than 41.1 C, intubation with neuromuscular blockade and paralysis is considered.
- In cases refractory to supportive care, cyproheptadine which does not have an IV formulation and must, therefore, be administered via a nasogastric or orogastric tube.(Anti serotonergic agent)

Mood Stabilisers



Lithium



Lithium

- 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 water 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

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

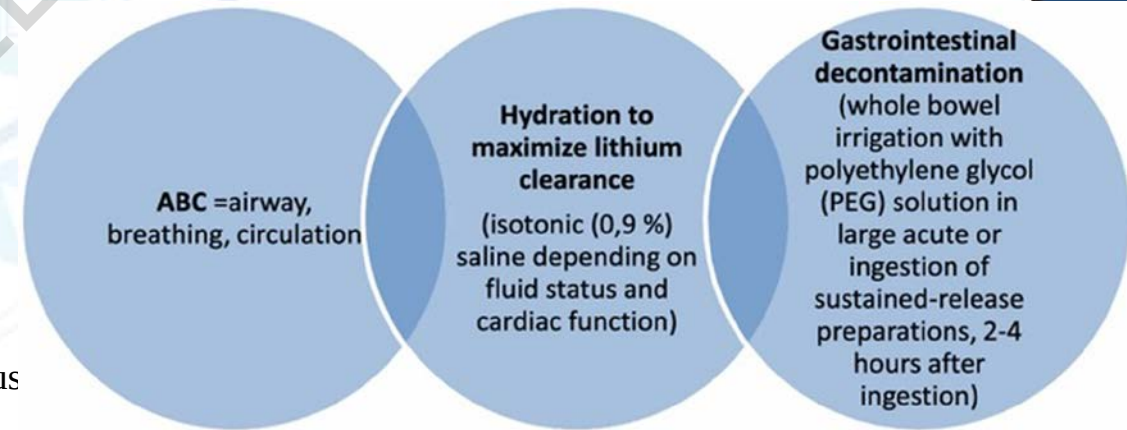
Oliguria and Renal Failure

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

Mechanism Of Action:

Enhancement of GABA activity

Modulation of voltage sensitive sodium channels

Half Life- 10 – 16 hrs

Indications:

Acute Mania

Bipolar Depression

Schizoaffective Disorder

Management for toxicity

1. Therapy for patients with valproate toxicity is mainly supportive and includes initial stabilization and resuscitation
2. Measures include maintenance of airway, breathing, and circulation.
3. Establish early IV access as intravenous fluid administration for the patients presenting with hypotension.
4. Patients with severe respiratory depression may require endotracheal intubation and mechanical ventilation.
5. Benzodiazepines should be administered if there is a seizure due to valproate toxicity.

6. **Gastrointestinal decontamination** is performed with a single activated charcoal single dose if the patient presents within 2 hours of valproate overdose. As valproate is available in enteric-coated and extended-release preparations, which have slow absorption, activated charcoal can still be given more than 2 hours after ingestion

7. Consider the use of **L-carnitine** for patients presenting with an acute overdose of valproate presenting with altered mental status.

8. **Serum ammonia levels should be simultaneously measured**, and when serum ammonia levels start decreasing, L-carnitine therapy can stop.

9. **Hemodialysis with extracorporeal treatment is indicated for valproate concentration >1300 mg/L (>9000 micromol/L)**, presence of shock, or cerebral edema.

Benzodiazepines



NPTEL



Benzodiazepines

- Benzodiazepines can have short or long durations of action. This affects the onset and course of withdrawal.
- **Short-acting benzodiazepines include oxazepam, alprazolam and temazepam.** Withdrawal typically **begins 1-2 days** after the last dose, and continues for 2-4 weeks or longer.
- **Long-acting benzodiazepines include diazepam ,clonazepam and nitrazepam.** Withdrawal typically **begins 2-7 days** after the last dose, and continues for 2-8 weeks or longer

Symptoms include:

Anxiety

Insomnia

Restlessness

Agitation and irritability

Poor concentration and memory

Muscle tension and aches.

Signs & Symptoms of Benzodiazepines discontinuation

- Return of original anxiety symptoms (Recurrence)
- Worsening of original anxiety symptoms (Rebound)
- Emergence Of New symptoms (Withdrawal)

Disturbances Of Mood and Cognition

Anxiety, Apprehension , dysphoria ,pessimism , Irritability

Disturbances of Sleep

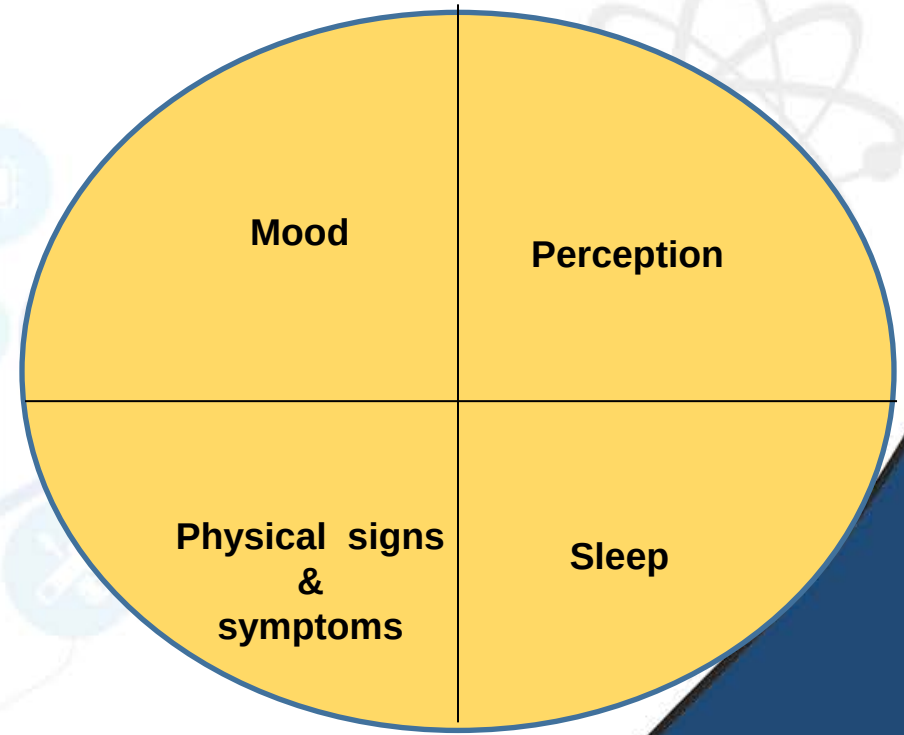
Insomnia, altered sleep-wake cycle and daytime drowsiness.

Physical Signs and symptoms

Tachycardia, elevated blood pressure , hyperreflexia , muscle tension , agitation , muscle ,joint pain , grand mal seizures.

Perceptual Disturbances

Hyperacusis , depersonalisation , blurred vision , illusions and hallucinations



Signs of Benzodiazepines Intoxication

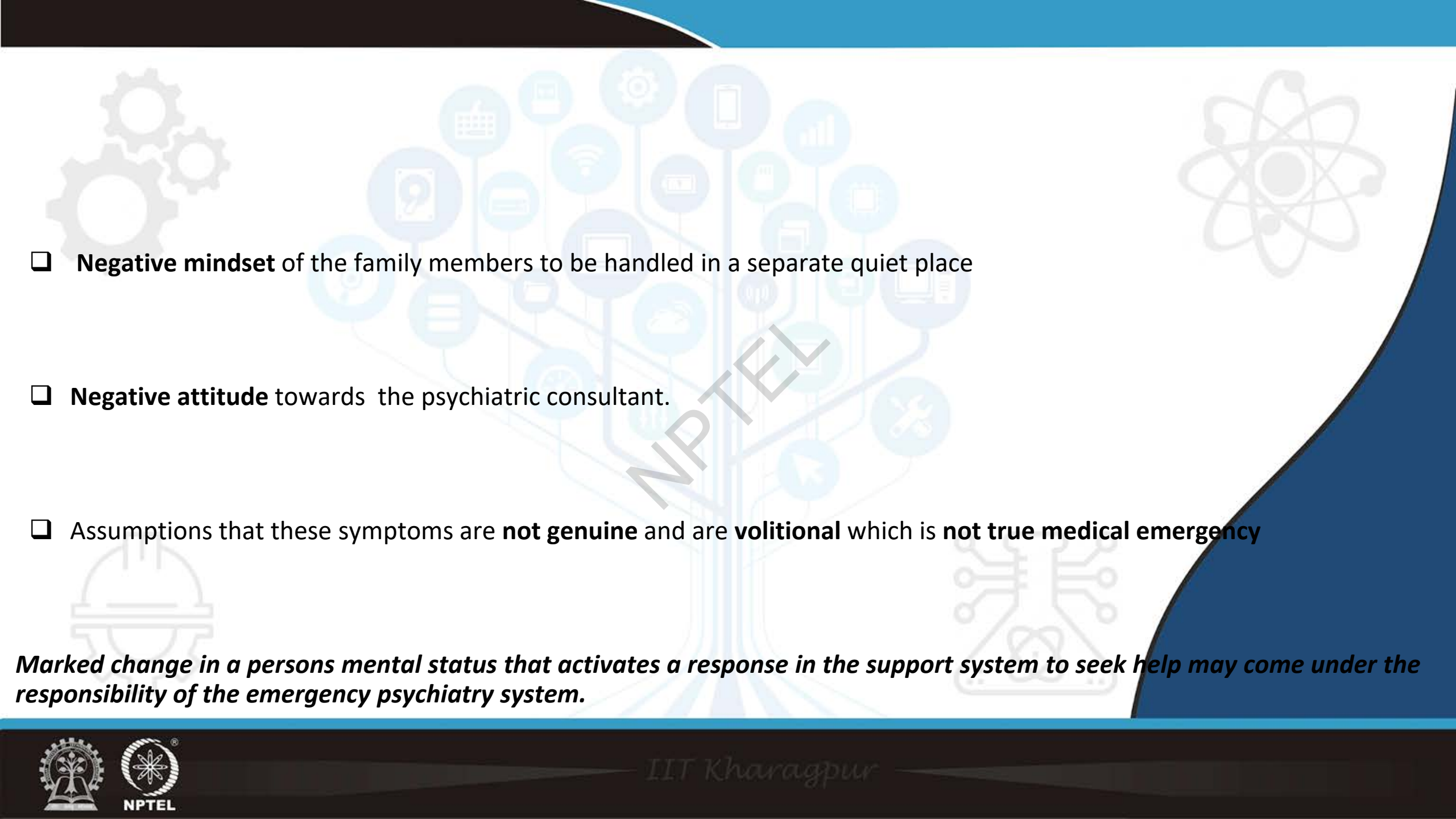
- Inco-ordinaton
- Dysarthria
- Nystagmus
- Impaired memory
- Gait disturbances.
- In severe cases stupor coma and death occurs

Guidelines for Benzodiazepines Withdrawal

- Evaluate and treat medical and psychiatric condition
- Obtain drug history and urine and blood samples.
- To determine the dose required for stabilisation from history obtained, clinical presentation.
[Most symptoms occur on first 3 days of abstinence and seizure on 2nd or 3rd day]
- Hospitalise if there is poor social support, polysubstance dependence and /or medical problems.
- Long acting benzodiazepines preferred.
- After stabilizing reduce dosage by 30 % on second and third day
- Tapering of the dose should be done depending upon the symptoms present by counteracting them
- Psychological intervention assist patients in managing related anxiety.

Factors which demand psychiatric emergency care

- ☐ Decreased Accessibility
- ☐ Unpredictibleness
- ☐ lack of health insurance
- ☐ Poverty
- ☐ Limited education
- ☐ Belongingness from vulnerable population
- ☐ No devotion of separate space for Psychiatric emergencies services in ER
- ☐ Psychiatric patients regarded as nuisance and hindrance to work at Hospital
- ☐ Agitated patients require seclusion and restraints.

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- ☐ **Negative mindset** of the family members to be handled in a separate quiet place
 - ☐ **Negative attitude** towards the psychiatric consultant.
 - ☐ Assumptions that these symptoms are **not genuine** and are **volitional** which is **not true medical emergency**

Marked change in a persons mental status that activates a response in the support system to seek help may come under the responsibility of the emergency psychiatry system.

CONCLUSIONS

-In this lecture we have discussed regarding the concepts of suicide , violence/aggression, catatonia , withdrawal /intoxication due to substances, serotonin syndrome, neurolept malignant syndrome , lithium toxicity.

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**THANK
YOU !**