DELIRIUM: Identification, Prevention and Management

PRESENTER:
DR RENU MOUNIKA T
POST GRADUATE

MODERATOR:
DR E ANANDA REDDY
PROFESSOR
DEPARTMENT OF PSYCHIATRY

- 71 years old male Mr. K, with history of asthma, BPH and Hypertension admitted to surgery ward 3 days ago for bilateral lower extremity cellulitis
- At the time of admission he was cooperative and oriented but over the past 24 hours has become occasionally confused, agitated, uncooperative and somnolent
- He appears to be talking to someone in his room and trying to pull out the IV line
- His current medications include: lisinopril, naproxen, cimetidine, albuterol/ipratropium inhaler, levofloxacin, oxygen via nasal canula

- He has no known psychiatric history
- He drinks 1-2 glasses of wine daily night
- During interview, he is difficult to rouse and falls asleep several times
- He struggles to maintain focus on questions and it is difficult to perform the mental status examination
- He believes he is at home and that the interviewer is his cousin

WHAT IS DELIRIUM..?

Delirium is a transient organic mental syndrome of acute onset, characterized by

- Global impairment of cognitive functions
- Reduced level of consciousness
- Attentional abnormalities
- Increased or decreased psychomotor activity
- Disordered sleep wake cycle

Delirium is also known as….

- Acute confusional state
- Acute Brain failure
- Hepatic encephalopathy
- Organic brain syndrome
- Toxic or metabolic encephalopathy
- Sundowning
- Intensive Care Unit Psychosis

RECOGNITION OF DELIRIUM

- Delirium is commonly unrecognized
  - Physicians miss 57-83% of cases
  - Hospital admission 14-24% (overall prevalence 1-2%)
- The incidence of delirium arising during a hospital stay ranges from 06-56%
- Delirium should always be considered when there is an acute or subacute deterioration in behavior, cognition or function
- 30-40% of delirium cases are preventable

PREDISPOSING FACTORS

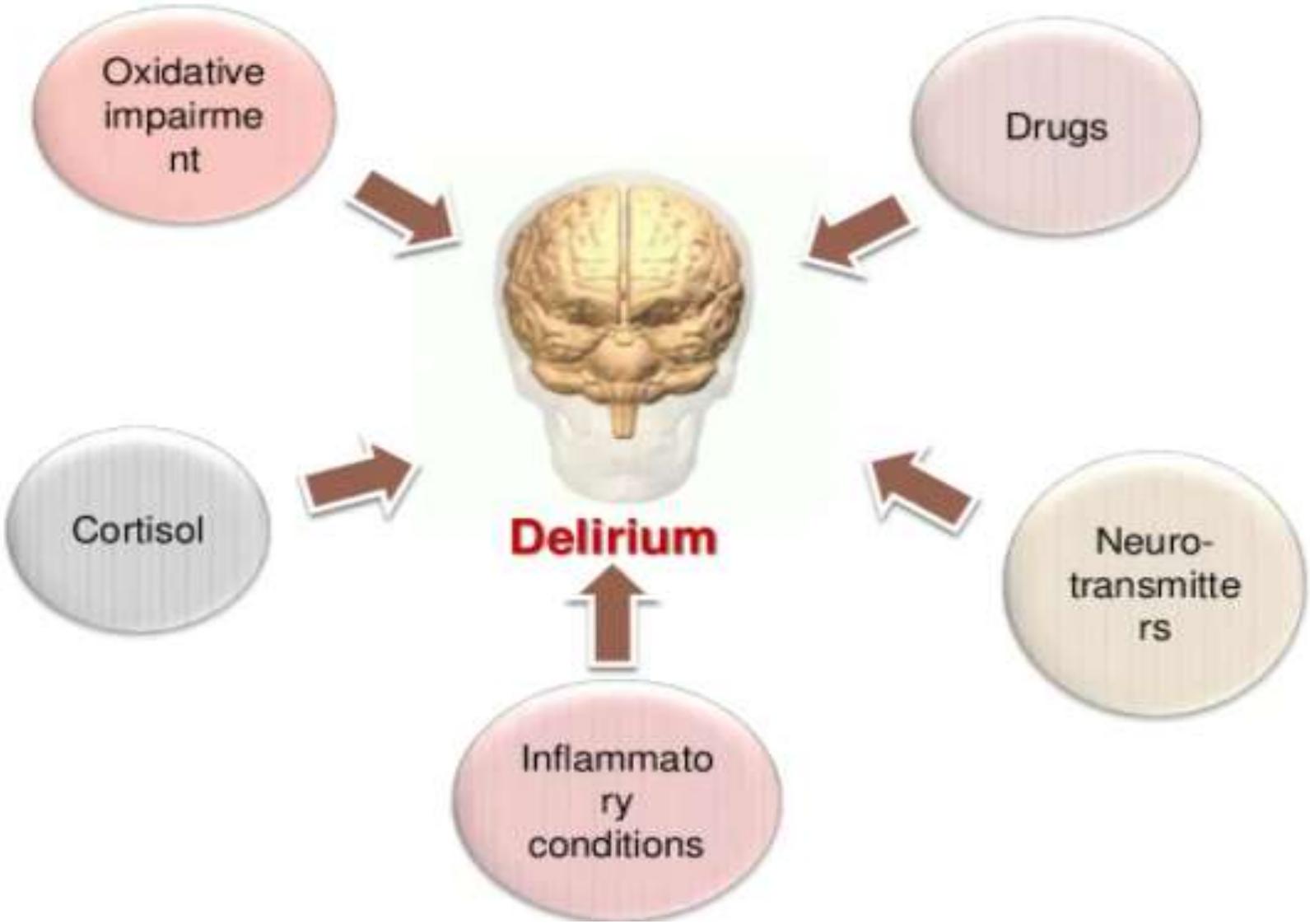
- Advanced age
- Dementia
- Functional impairment in activities of daily living
- Medical comorbidity
- History of alcohol abuse
- Sensory impairment

PRECIPITATING FACTORS

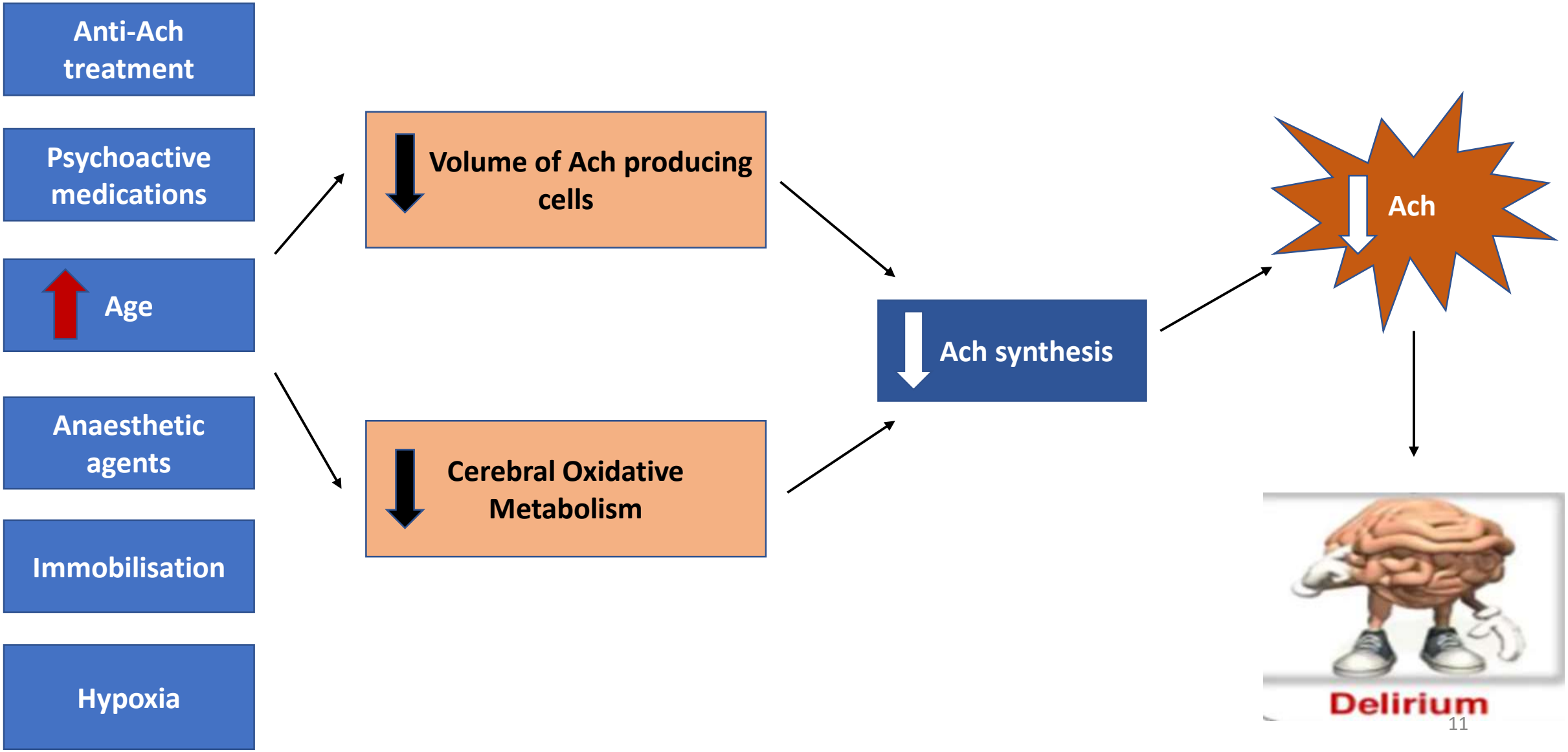
- Acute myocardial events
- Acute pulmonary events
- Bed rest
- Fluid and electrolyte disturbance
- Drug withdrawal
- Infection

- Multiple Medications
- Uncontrolled pain
- Urinary retention, faecal impaction
- Indwelling devices (urinary catheters)
- Severe anaemia
- Use of restraints
- Intracranial events

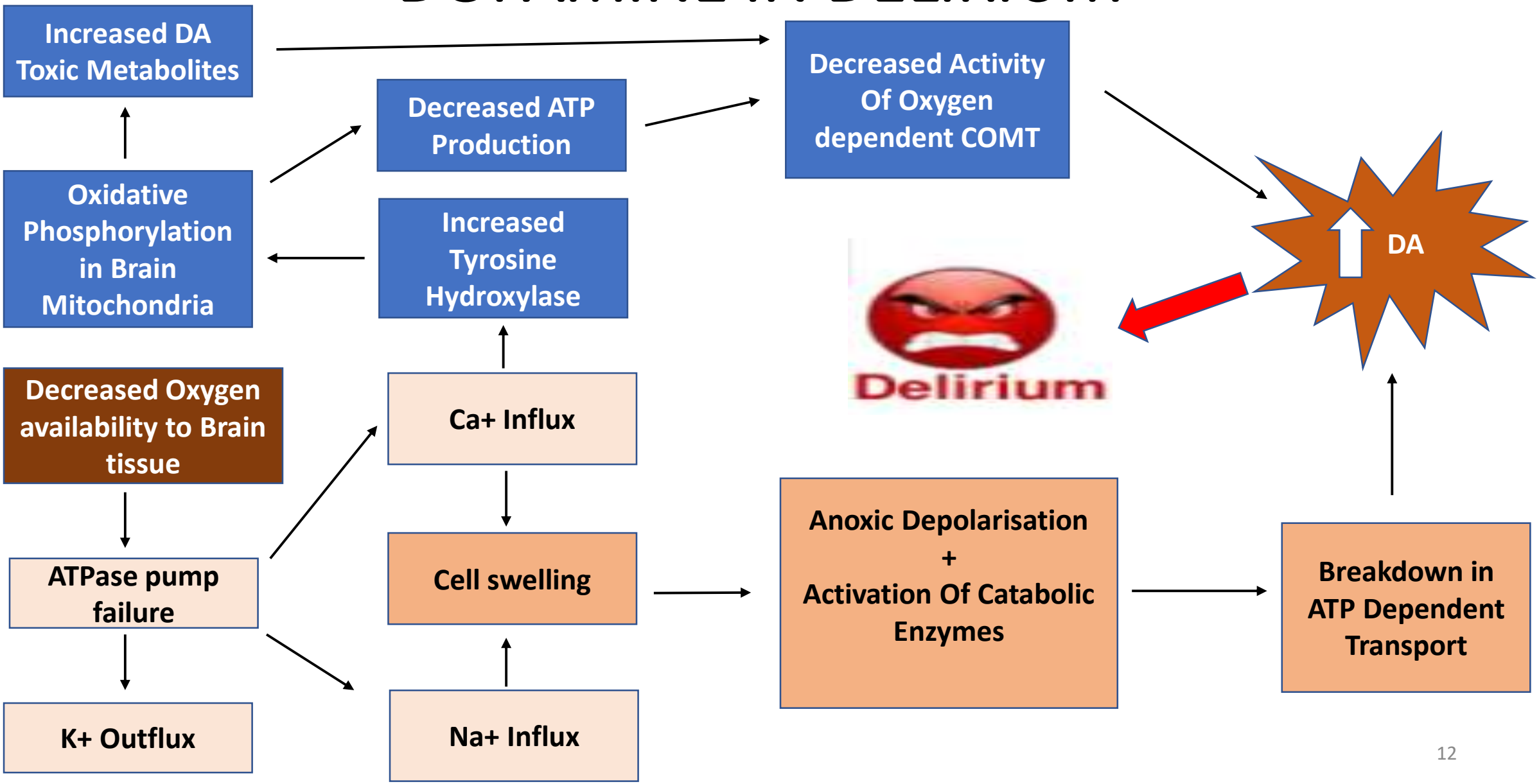
PATHO-PHYSIOLOGY



ACETYLCHOLINE IN DELIRIUM



DOPAMINE IN DELIRIUM



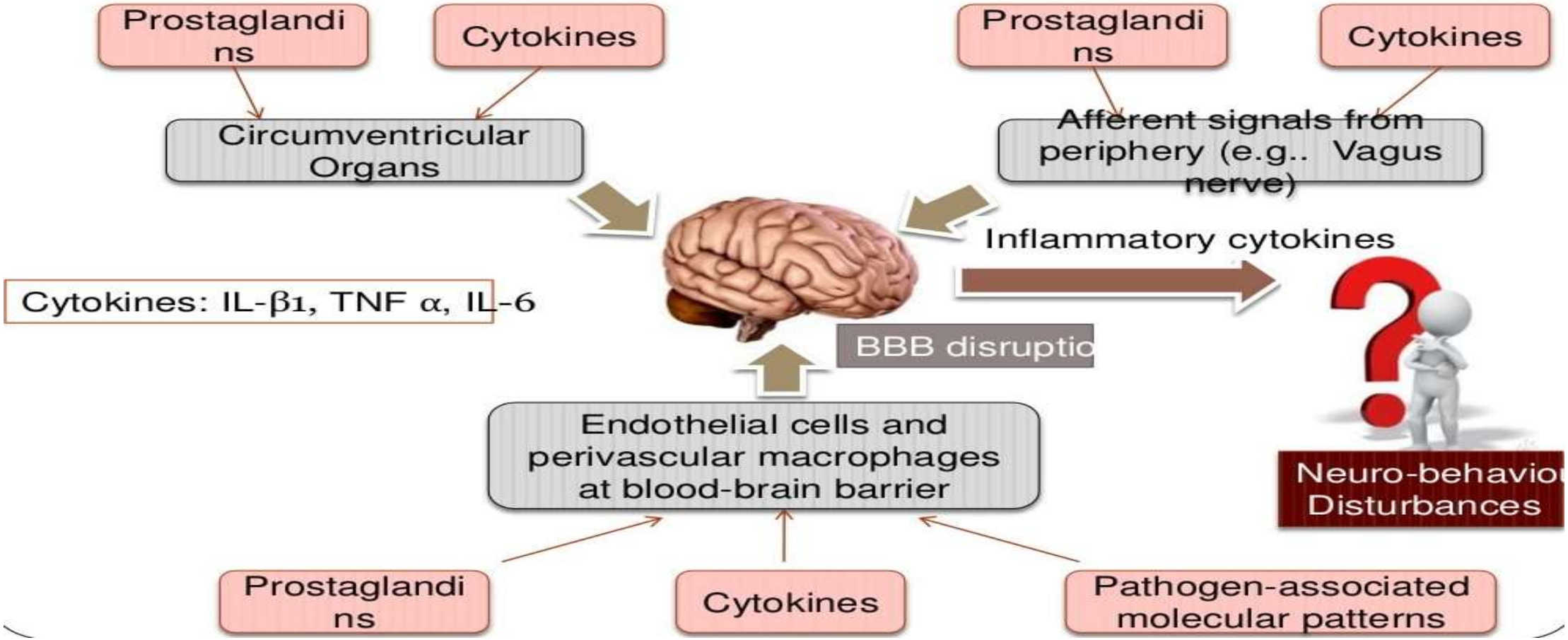
SEROTONIN IN DELIRIUM

- Serotonin is a major CNS neurotransmitter
- Production depends on transport of tryptophan across the blood-brain barrier
- Tryptophan competes with the amino acid phenylalanine for transport across the blood-brain barrier
- Disturbance of the tryptophan:phenalanine ratio may increase or decrease the level of serotonin resulting in delirium
- Disturbance of the tryptophan:phenalanine ratio has been observed in post traumatic states and other medical and surgical conditions.

GABA AND GLUTAMATE IN DELIRIUM

- GABA and glutamate have both been implicated in the development of delirium
- Glutamate is metabolized into GABA, which is an inhibitory neurotransmitter
- In hepatic encephalopathy, there is increased ammonia levels, which is the precursor of GABA
- Benzodiazepine and alcohol withdrawal are associated with reduced GABA activity which can cause delirium

INFLAMMATORY PROCESS IN DELIRIUM



CORTISOL IN DELIRIUM

STRESS

Sympathetic activity

Other stress hormones

CORTISOL

Memory

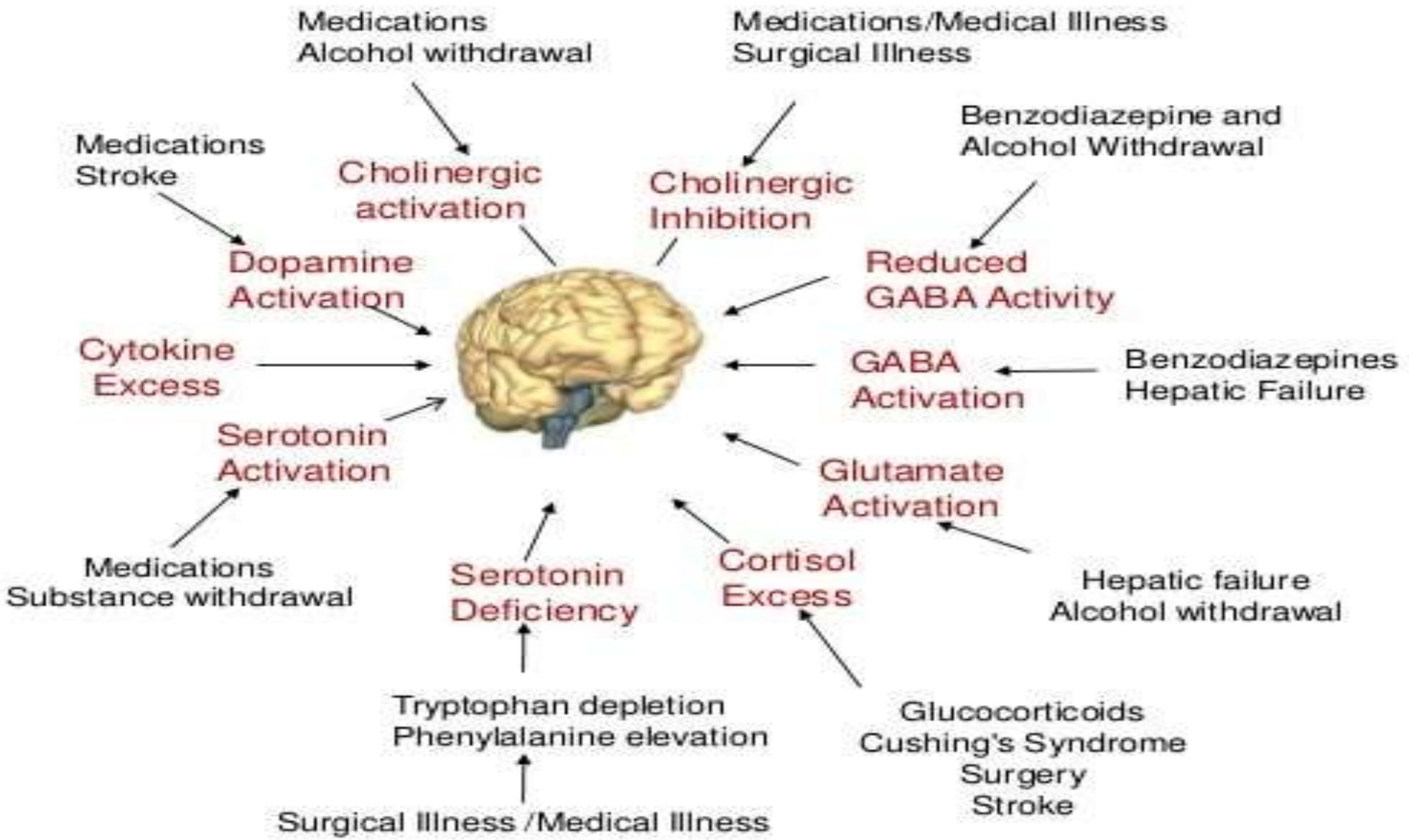
Mood

Hippocampal and Limbic Pathway Dysfunction

- Cell Death
- Atrophy
- Mitochondrial Dysfunction

Hippocampus

Summary of Patho-physiology



DSM 5 CRITERIA - DELIRIUM

- Disturbance in attention and awareness
- Develops over short period; fluctuates
- Additional disturbance in cognition
- Not accounted for by other neurocognitive disorders
- Caused by a general medical condition
- Can be multiple etiologies

DSM 5 CRITERIA

- Classification of delirium
  - Delirium due to another medical condition
  - Substance intoxication delirium
  - Substance withdrawal delirium
  - Delirium due to multiple etiologies
  - Medication induced delirium

CLINICAL FEATURES

- Temporal course
  - Abrupt or acute onset
    - Within days
  - Fluctuation in symptom severity
    - Waxing and waning
    - Worse at night
    - May result in diagnostic uncertainty

- Diffuse cognitive impairment
  - Attentional deficits
    - Reduced ability to focus, sustain or shift attention
    - “Clouding of consciousness”
  - Memory impairment
    - Long and short term
  - Disorientation
    - Commonly to time and place
    - Rarely to person
  - Executive dysfunction

- Thought disturbances
  - Disorganized

- Language disturbances
  - Word finding problems
  - Dysgraphia
  - Dysarthria
  - Dysnomia

- Perceptual disturbances
  - Misperceptions
  - Hallucinations

- Psychomotor abnormalities
  - Hyper, hypo or mixed

- Sleep-wake cycle disturbance
  - Insomnia
  - Frequent napping or drowsiness during the day
  - Reversal of sleep/wake cycle

- Delusions
  - Usually paranoid and not systematized

- Affective lability

- Neurologic abnormalities

TYPES OF DELIRIUM

- Hyperactive
  - Better recognized
  - More attention to treatment
  - Associated with improved outcome
- Hypoactive
  - Little recognized
  - Depression is primary differential
  - Appear sluggish and lethargic as well as confused
  - Associated with poor outcomes
- Mixed

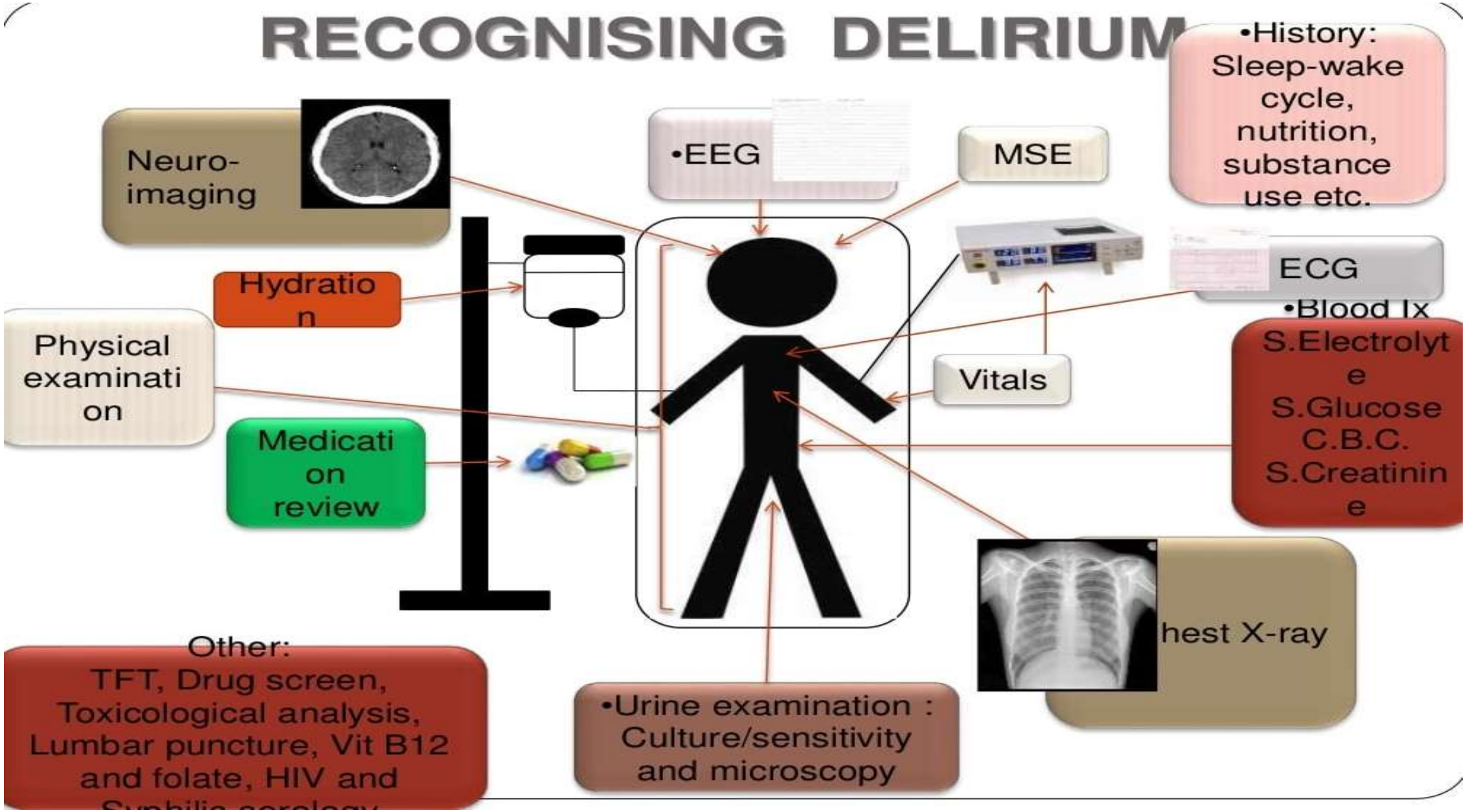
PAEDIATRIC DELIRIUM

- Childhood delirium has a different course and clinical profile than adults and geriatric patients
- The clinical manifestations between children and adults might differ, which may be due to their young age and developmental changes
- PD shows a more distinct course with a more acute onset, less circadian variety in symptoms and less sleep-wake cycle disturbances, as compared to adults

ASSESSMENT OF DELIRIUM

- Confusion Assessment Method (CAM)
- Delirium Rating Scale (DRS)
- Intensive Care Delirium Screening Checklist (ICDSC)
- Memorial Delirium Assessment Scale (MDAS)
- Delirium Symptom Interview (DSI)
- Clock drawing Test

RECOGNISING DELIRIUM



Differential Diagnosis of Delirium

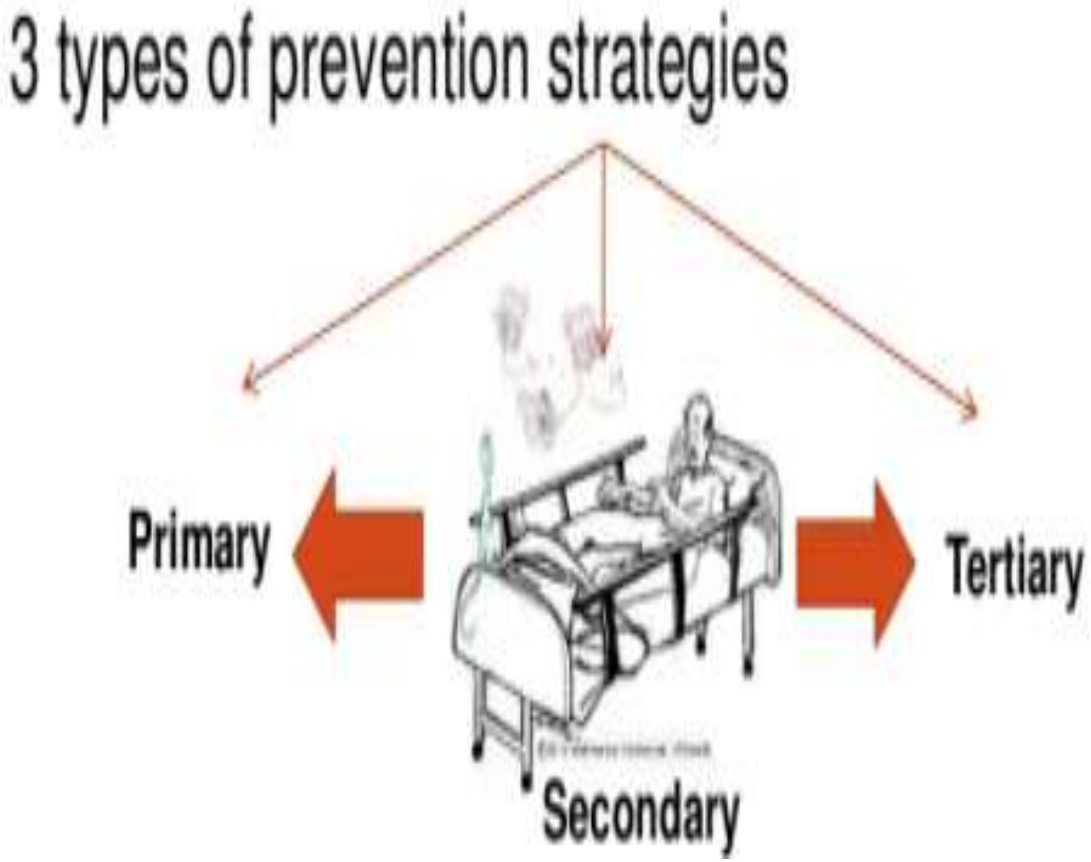
	Delirium	Dementia	Depression
Onset	Abrupt	Slow and insidious	Variable
Daily Course	Fluctuating	Usually stable	Usually stable
Length	Hours to weeks	Years	Variable
Consciousness	Reduced	Clear	Clear
Alertness	Increased or decreased	Usually normal	Normal
Activity	Increased or decreased	Variable	Variable
Attention	Impaired	Usually normal	Usually normal
Orientation	Impaired	Impaired	Normal

- Hypoactive delirium can resemble depression. However clouding of consciousness is absent in depression

- Hyperactive delirium can mimic acute psychosis. However in delirium hallucinations are predominantly visual and delusion are not well systematized.

- An EEG can differentiate it from both depression and psychosis

PREVENTION OF DELIRIUM



- Multicomponent approaches to reduce the risk factors
- Difficult for a single person to implement and often led by teams of physicians, nurse, care givers and others
- It can be prevented or at least moderated by addressing modifiable risk factors

Yale Delirium Prevention Trial

RISK FACTOR	INTERVENTION
Cognitive impairment	Orientation protocol, cognitively stimulating activities 3x/day
Sleep deprivation	Non-pharmacologic protocol, noise reduction, schedule adjustments
Immobility	Ambulation or active ROM exercises; minimize equipment
Visual impairment	Glasses or magnifying lens, adaptive equipment
Hearing impairment	Portable amplifying devices, earwax disimpaction
Dehydration	Early recognition and volume

PRINCIPLES OF MANAGEMENT

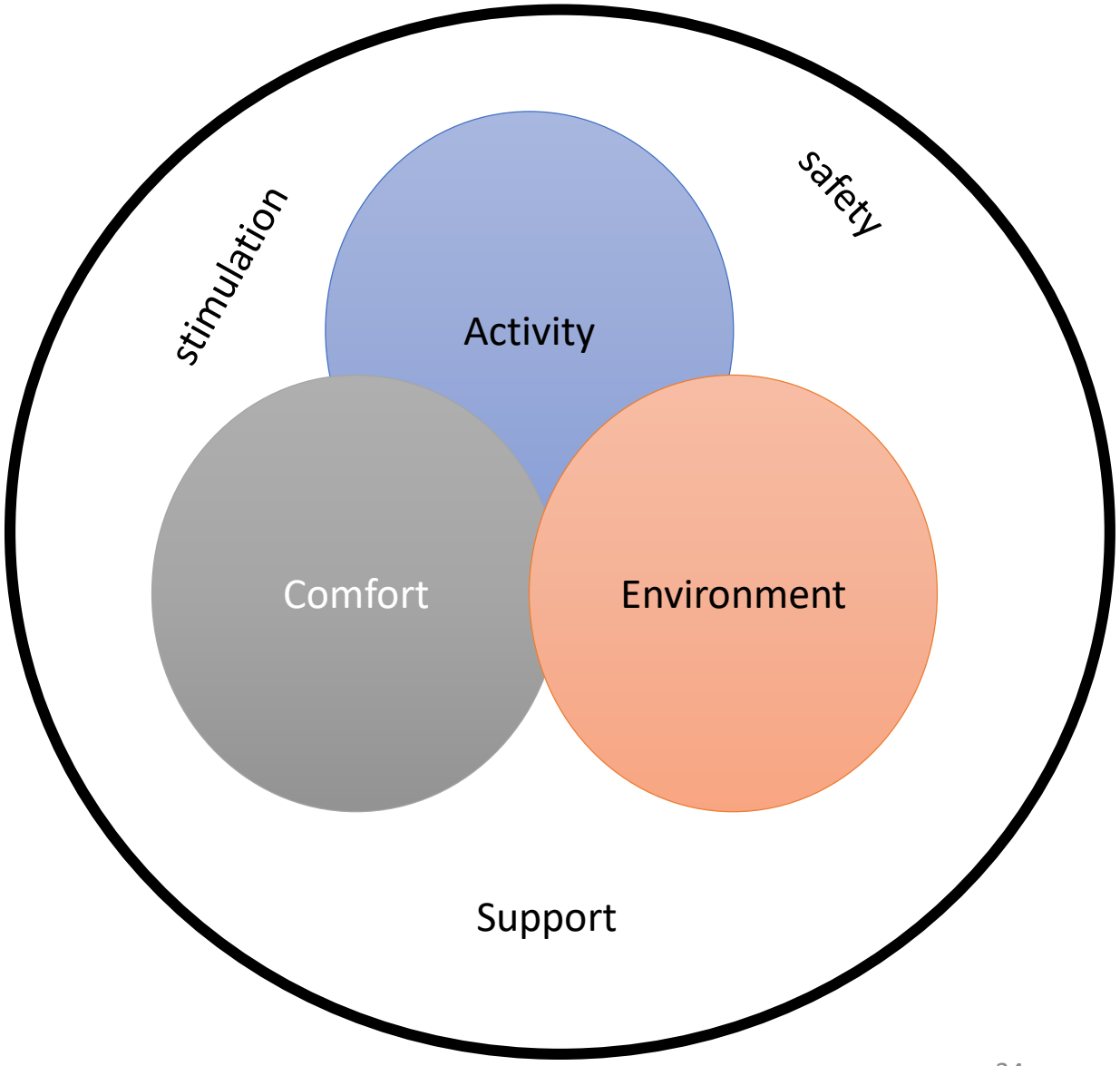
1. Determine the cause and treat it
2. Avoid exacerbation
3. Provide supportive care
4. Manage the behavior by pharmacological and non-pharmacological methods
5. Restoration of cognitive and self care functions
6. Psycho-education of the family members

NON PHARMACOLOGICAL MANAGEMENT

- Early mobilisation
- Physical therapy
- Cognitive stimulation
- Frequent Re-orientation

- Minimize disturbance
- Family engagement
- Familiar Stimuli
- Noise/Light Control

- Dietary Needs
- Bowel and Bladder Care
- Sleep Hygiene
- Sensory Correction
- Removal Of lines
- Safe Pain control



PHARMACOLOGICAL MANAGEMENT (NICE GUIDELINES)

Reserved for patients with severe agitation or behavioral disturbance who are at risk of interrupting essential medical care and risk of causing harm to themselves or others

- 1st choice: **HALOPERIDOL**
- Oral Dose : 0.25-0.5 mg
- Very agitated patients : Bolus dose of 5-10mg IV/IM
- Less anticholinergic activity
- HPL+ atypical antipsychotic use has increased to 5- 40% in recent years

- **OLANZAPINE:**
  - Orally or sublingually
  - Initial dose 1.25– 2.5 mg then adjusted, depending on response, to 1.25–20 mg per day

- **AMISULPRIDE:**
  - 50–800 mg/day
  - Dose is flexible according to clinicians experience

- **QUETIAPINE:**
  - Initially 12.5–25 mg orally once a day, increased to twice a day if needed.
  - Maximum 200 mg in 24 hours (50 mg in elderly)

BENZODIAZEPINES

- When the agitation is associated with sedative-hypnotic withdrawal (such as alcohol, benzodiazepines, barbiturates), benzodiazepines are the treatment of choice

- Insomnia is best treated with benzodiazepines with short or intermediate half-lives (Ex: Lorazepam 1 to 2 mg at bedtime)

- Benzodiazepines with long half-lives and barbiturates should be avoided unless they are being used as part of the treatment for the underlying disorder (Ex: Alcohol withdrawal)

ELECTROCONVULSIVE THERAPY (ECT)

- ECT is also a treatment for delirium when other approaches have failed

- It has been used as a last resort for delirious patients with severe agitation who are not responsive to pharmacotherapy, such as high doses of iv haloperidol

TREATMENT OF SPECIFIC CAUSES

- **Anti-cholinergic Intoxication:**
  - Physical agitation and visual hallucinations
  - Physostigmine - Drug of choice

- **Substance Intoxication:**
  - Cessation of the substance
  - BZD: Flumazenil
  - Opioid: Naloxone

- **Wernicke's Encephalopathy:**
  - Thiamine supplementation IV or IM

- **Substance Withdrawal:**
  - Aim is to reduce severity of withdrawal, preventing delirium and reducing the incidence of the seizures
  - Benzodiazepines-1st line

- **Terminally ill patients:**
  - Not all causes are reversible and realistic treatment expectations should be set after discussion with patient and care givers

RECENT STUDIES

- Recent studies show that delirium strongly predicts future new-onset dementia, as well as accelerating existing dementia.
- Newer studies on Ach enhancers
  - Studies done on Rivastigmine show promising results, but should be commenced before onset if possible
  - Some evidence for Donepezil
- 5HT antagonist - Odansetron (8mg/day) has been found effective especially in hypoactive type, but efficacy is found in both types.
- Stimulants are now found to be effective in hypoactive type - modafinil, methylphenidate, dexamphetamine

RECENT STUDIES

- Drug modifying NE transmission
  - Alpha 2 agonists - clonidine, dexmedetomidine
  - NE released especially in withdrawal states and hypoxic damage is severely neurotoxic
  - New studies on dexmedetomidine pre op and postop shows it reduces transition to delirium from 40% to 3-4% and reduced need for opiate analgesics post op

IMPACT AND OUTCOME OF DELIRIUM

CONCLUSION

- Delirium is a state characterized by an acute decline in both the level of consciousness and cognition with particular impairment in attention.
- There are multiple etiologies of delirium
- 65% unrecognized by physicians
- Early diagnosis and evaluation of cause is essential
- Treatment include pharmacological and non pharmacological aspect
- Non pharmacological approach is more essential in treatment

THANK YOU