Coma

ZSMU

Department of general practice – family medicine

Neural basis of consciousness

- **Consciousness** *cannot be readily defined in terms of anything else*

- *A state of awareness of self and surrounding*

- **Mental Status** =

  Arousal + Content

Anatomy of Mental Status

- Ascending reticular activating system (ARAS)
  - Activating systems of upper brainstem, hypothalamus, thalamus
  - Determines the level of arousal
- Cerebral hemispheres and interaction between functional areas in cerebral hemispheres
  - Determines the intellectual and emotional functioning
- Interaction between cerebral hemispheres and activating systems

The content of consciousness

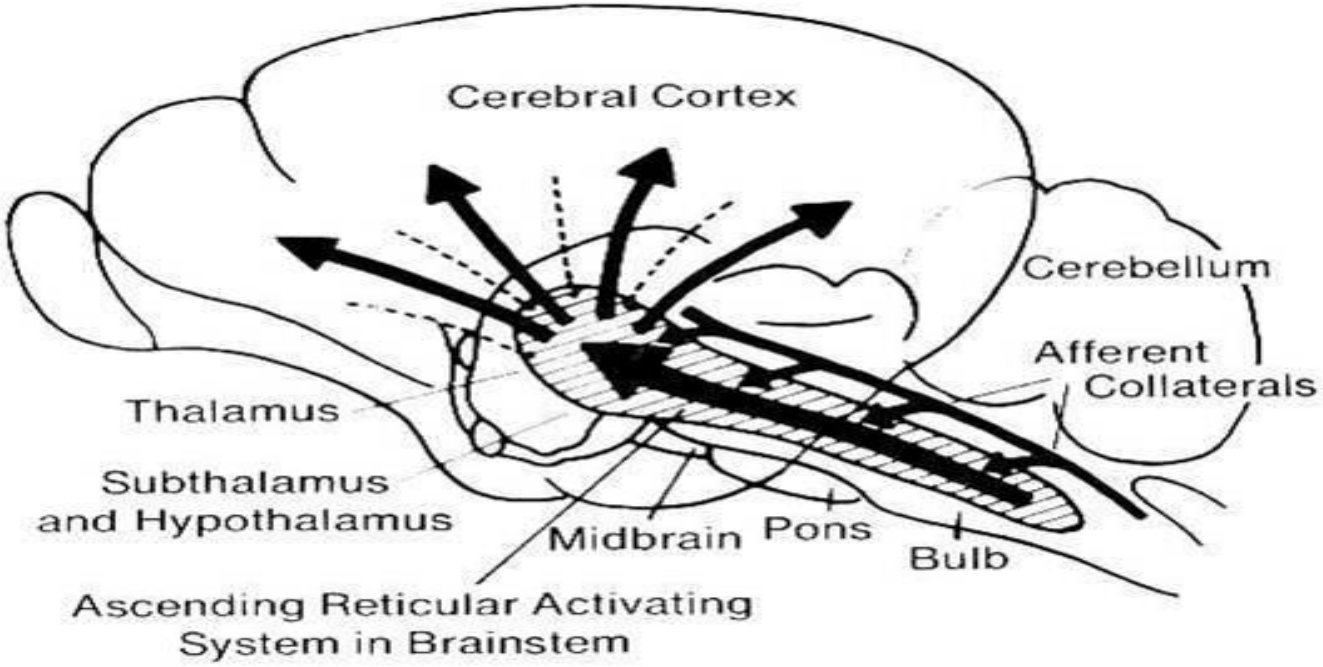
- Sum of patient's intellectual (cognitive) functions and emotions (affect)

  Sensations, emotions, memories, images, ideas (SEMII)
- Depends upon the activities of the cerebral cortex, the thalamus & their interrelationship

Lesions of these structures will diminish the content of consciousness (without changing the state of consciousness)

The state of consciousness (arousal)

- The ascending RAS, from the lower border of the pons to the ventromedial thalamus
- The cells of origin of this system occupy a paramedian area in the brainstem



Altered Mental Status

Abnormal change in level of arousal or altered content of a patient's thought processes

- **Change in the level of arousal or alertness**
  - inattentiveness, lethargy, stupor, and coma.
- **Change in content**
  - "Relatively simple" changes: e.g. speech, calculations, spelling
  - More complex changes: emotions, behavior or personality
  - Examples: *confusion*, *disorientation*, *hallucinations*, *poor comprehension*, or *verbal expressive difficulty*

Definitions of levels of arousal (conciousness)

- Alert (Conscious) - Appearance of wakefulness, awareness of the self and environment
- Lethargy - mild reduction in alertness
- Obtundation - moderate reduction in alertness. Increased *response time* to stimuli.
- Stupor - Deep sleep, patient can be aroused only by vigorous and repetitive stimulation. Returns to deep sleep when not continually stimulated.
- Coma (Unconscious) - Sleep like appearance and behaviorally unresponsive to all external stimuli (*Unarousable unresponsiveness*, *eyes closed*)

Semicoma was defined as complete loss of consciousness with a response only at the reflex level (now obsolete)

Psychogenic unresponsiveness

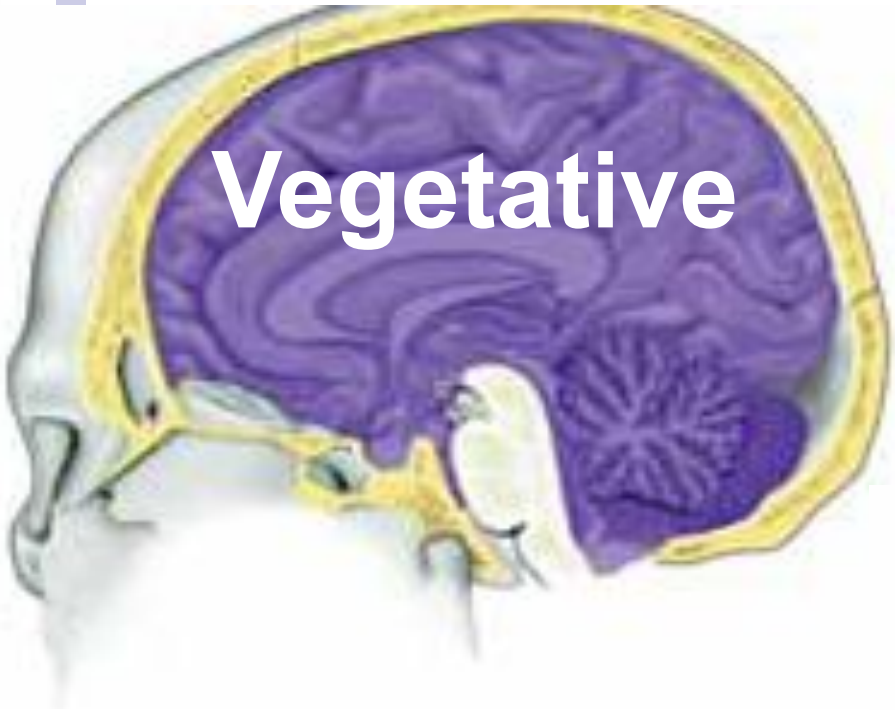
- The patient, although apparently unconscious, usually shows some response to external stimuli
- An attempt to elicit the corneal reflex may cause a vigorous contraction of the orbicularis oculi
- Marked resistance to passive movement of the limbs may be present, and signs of organic disease are absent

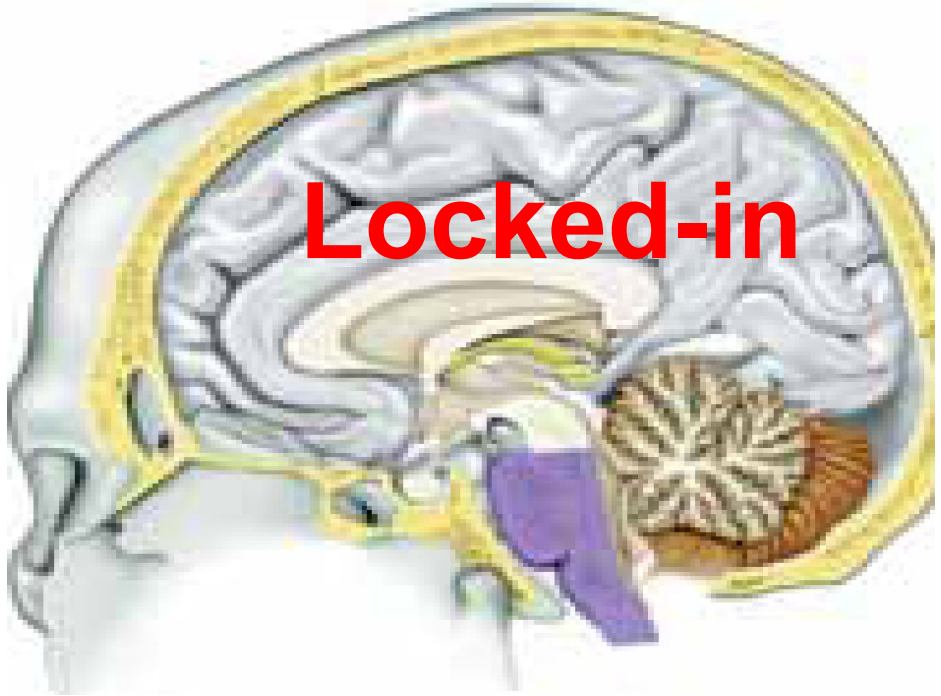
Vegetative state *(coma vigil, apallic syndrome)*

- Patients who survive coma do not remain in this state for > 2–3 weeks, but develop a persistent unresponsive state in which sleep–wake cycles return.
- After severe brain injury, the brainstem function returns with sleep–wake cycles, eye opening in response to verbal stimuli, and normal respiratory control.

Locked in syndrome

- Patient is awake and alert, but unable to move or speak.
- **Pontine lesions** affect lateral eye movement and motor control
- Lesions often spare vertical eye movements and blinking.


Vegetative


Locked-in

Confusional state

- Major defect: lack of attention
  - *Disorientation* to time > place > person
  - Patient thinks *less clearly* and *more slowly*
  - *Memory* faulty (difficulty in repeating numbers (digit span)
- Misinterpretation of external stimuli
- Drowsiness may alternate with hyper -excitability and irritability

Delirium

- **Markedly abnormal mental state**
  - **Severe confusional state**
  - **PLUS Visual hallucinations &/or delusions**

    (complex systematized dream like state)

- Marked: disorientation, fear, irritability, misperception of sensory stimuli
- Pt. out of true contact with environment and other people
- ***Common causes*:**

1. Toxins
2. metabolic disorders
3. partial complex seizures
4. head trauma
5. acute febrile systemic illnesses

To cause coma, as defined as a state of unconsciousness in which the eyes are closed and sleep–wake cycles absent

- Lesion of the cerebral hemispheres extensive and bilateral
- Lesions of the brainstem: above the lower 1/3 of the pons and destroy both sides of the paramedian reticulum

The use of terms other than coma and stupor to indicate the degree of impairment of consciousness is beset with difficulties and more important is the use of coma scales (**Glasgow Coma Scale**)

Glasgow Coma Scale (GCS)

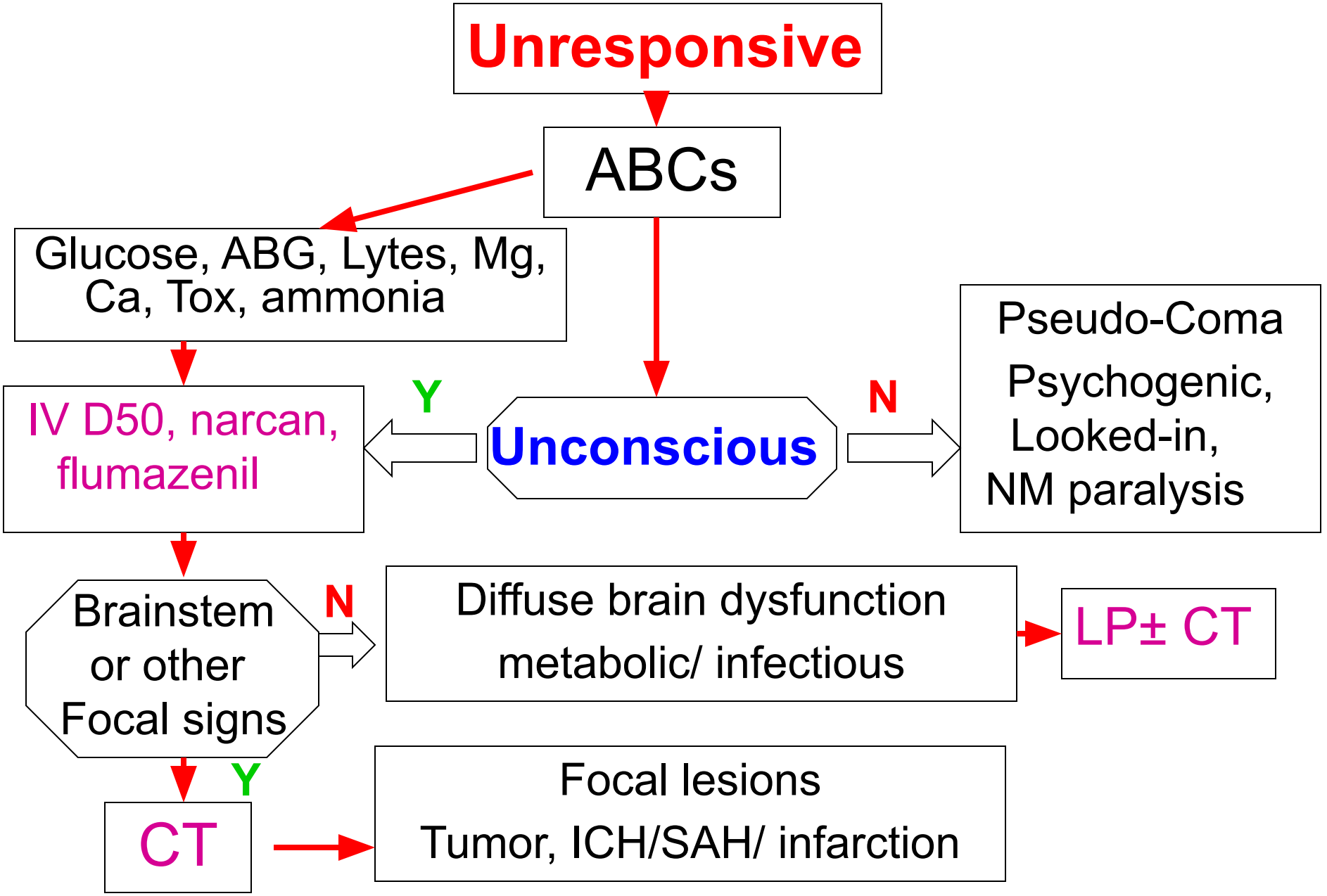
Best eye response (E)	Best verbal response (V)	Best motor response (M)
4 Eyes opening spontaneously	5 Oriented	6 Obeys commands
3 Eye opening to speech	4 Confused	5 Localizes to pain
2 Eye opening in response to pain	3 Inappropriate words	4 Withdraws from pain
1 No eye opening	2 Incomprehensible sounds	3 Flexion in response to pain
	1 None	2 Extension to pain
		1 No motor response

- Individual elements as well as the sum of the score are important.
- Hence, the score is expressed in the form "GCS 9 = E2 V4 M3 *at 07:35*

Generally, comas are classified as:

- Severe, with GCS ≤ 8
- Moderate, GCS 9 - 12
- Minor, GCS ≥ 13.

Approaches to DD



Approaches to DD

General examination:

On arrival to ER immediate attention to:

1. Airway
2. Circulation
3. establishing IV access
4. Blood should be withdrawn: estimation of glucose # other biochemical parameters # drug screening

- **Attention is then directed towards:**

1. Assessment of the patient
2. Severity of the coma
3. Diagnostic evaluation

- **All possible information from:**

1. Relatives
2. Paramedics
3. Ambulance personnel
4. Bystanders

particularly about the mode of onset

- **Previous medical history**:
  1. Epilepsy
  2. DM, Drug history
- **Clues** obtained from the patient's
  1. Clothing or
  2. Handbag
- **Careful examination for**
  1. Trauma requires complete exposure and ‘log roll’ to examine the back
  2. Needle marks

- If head trauma is suspected, the examination must await adequate stabilization of the neck.
- Glasgow Coma Scale: the severity of coma is essential for subsequent management.
- Following this, particular attention should be paid to brainstem and motor function.

Temperature

Hypothermia

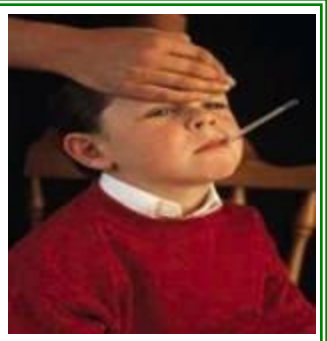
- Hypopituitarism, Hypothyroidism
- Chlorpromazine
- Exposure to low temperature environments, cold-water immersion

  Risk of hypothermia in the elderly with inadequately heated rooms, exacerbated by immobility.

- C/P: generalized rigidity and muscle fasciculation but true shivering may be absent. (a low-reading rectal thermometer is required).
- Hypoxia and hypercarbia are common.
- Treatment:

1. Gradual warming is necessary
2. May require peritoneal dialysis with warm fluids.

Hyperthermia (febrile Coma)



- Infective: encephalitis, meningitis
- Vascular: pontine, subarachnoid hge
- Metabolic: thyrotoxic, Addisonian crisis
- Toxic: belladonna, salicylate poisoning
- Sun stroke, heat stroke
- Coma with 2ry infection: UTI, pneumonia, bed sores.

Hyperthermia or heat stroke

Loss of thermoregulation dt. prolonged exertion in a hot environment

- Initial ↑ in body temperature with profuse sweating followed by
- hyperpyrexia, an abrupt cessation of sweating, *and then*
- rapid onset of coma, convulsions, and death

- This may be exacerbated by certain drugs, ‘Ecstasy’ abuse—involving a loss of the thirst reaction in individuals engaged in prolonged dancing.

Other causes

- Tetanus
- Pontine hge
- Lesions in the floor of the third ventricle
- Neuroleptic malignant syndrome
- Malignant hyperpyrexia with anaesthetics.

Heat stroke neurological sequelae

- Paraparesis.
- Cerebellar ataxia.
- Dementia (rare)

Pulse

- Bradycardia: brain tumors, opiates, myxedema.
- Tachycardia: hyperthyroidism, uremia

Blood Pressure

- High: hypertensive encephalopathy
- Low: Addisonian crisis, alcohol, barbiturate

Skin

- Injuries, Bruises: traumatic causes
- Dry Skin: DKA, Atropine
- Moist skin: Hypoglycemic coma
- Cherry-red: CO poisoning
- Needle marks: drug addiction
- Rashes: meningitis, endocarditis

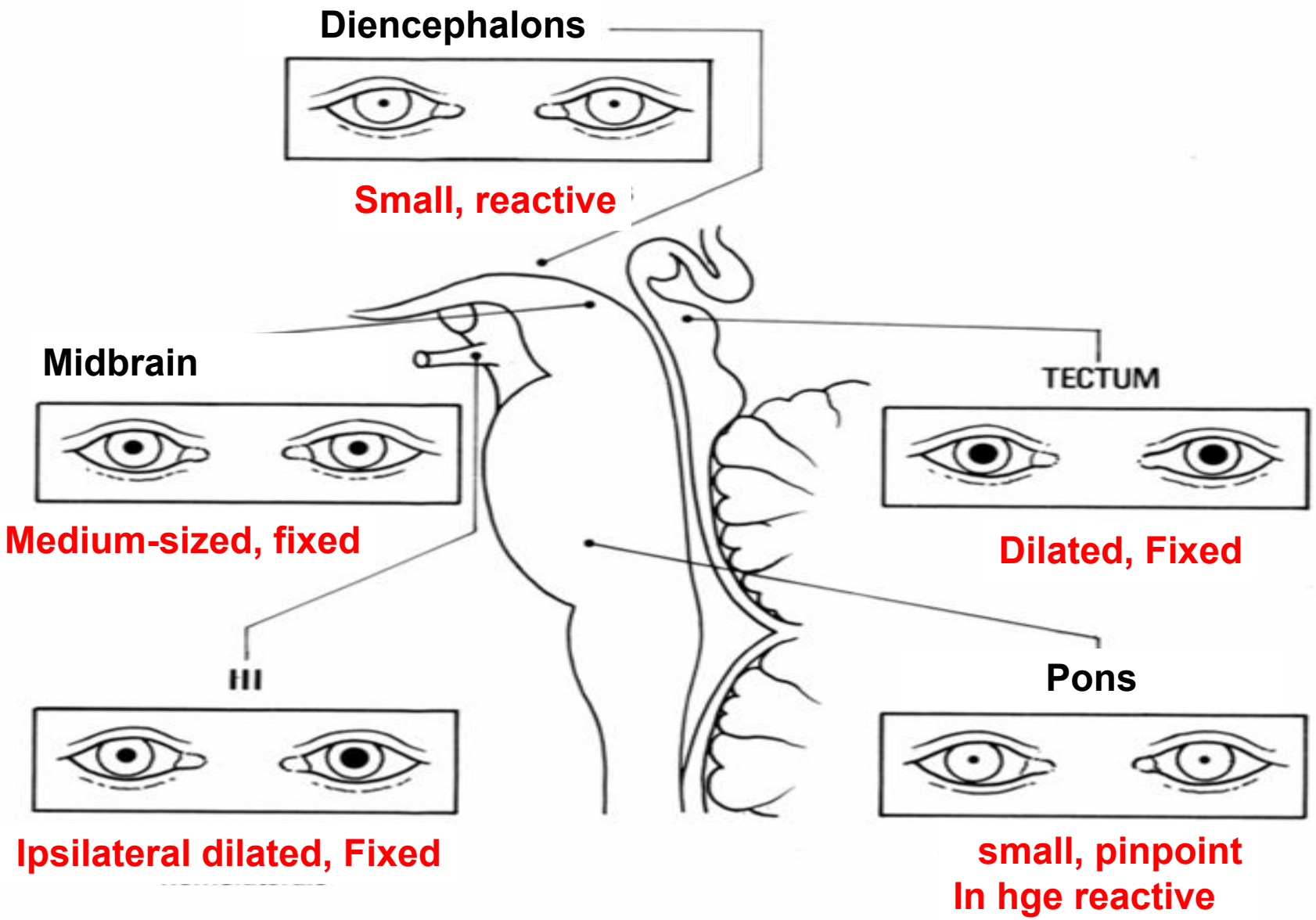
Pupils

- Size, inequality, reaction to a bright light.
- An important general rule: most metabolic encephalopathies give small pupils with preserved light reflex.
- Atropine, and cerebral anoxia tend to dilate the pupils, and opiates will constrict them.

Structural lesions *are more commonly associated with pupillary asymmetry and with loss of light reflex.*

- **Midbrain tectal lesions** : round, regular, medium-sized pupils, do not react to light
- **Midbrain nuclear lesions**: medium-sized pupils, fixed to all stimuli, often irregular and unequal.
- **Cranial n III distal to the nucleus**: Ipsilateral fixed, dilated pupil.

- **Pons (Tegmental lesions)** : bilaterally small pupils, {in pontine hge, may be pinpoint, although reactive} assess the light response using a magnifying glass
- **Lateral medullary lesion**: ipsilateral Horner's syndrome.
- **Occluded carotid artery** causing cerebral infarction: Pupil on that side is often small


Diencephalons
Small, reactive
Midbrain
Medium-sized, fixed
III
Ipsilateral dilated, Fixed
TECTUM
Dilated, Fixed
Pons
small, pinpoint
In hge reactive

Ocular movements

- The position of the eyes at *rest*
- Presence of *spontaneous eye movement*
- The reflex responses to oculocephalic and oculovestibular maneuvers
- In diffuse cerebral disturbance but intact brainstem function, slow roving eye movements can be observed
- Frontal lobe lesion may cause deviation of the eyes *towards* the side of the lesion

- Lateral pontine lesion can cause conjugate deviation to the *opposite* side
- Midbrain lesion Conjugate deviation *downwards*
- Structural brainstem lesion disconjugate ocular deviation

The oculocephalic (doll's head) response

rotating the head from side to side and observing the position of the eyes.

- If the eyes move *conjugately* in the *opposite direction* to that of head movement, the response is positive and indicates an *intact pons* mediating a normal vestibulo-ocular reflex

Caloric oculovestibular responses These are tested by the installation of ice-cold water into the external auditory meatus, having confirmed that there is no tympanic rupture.

- A normal response in a conscious patient is the development of *nystagmus* with the *quick phase away* from the stimulated side This requires intact cerebropontine connections

Odour of breath

- Acetone: DKA
- Fetor Hepaticus: in hepatic coma
- Urineferous odour: in uremic coma
- Alcohol odour: in alcohol intoxication

Respiration

- **Cheyne–Stokes respiration**: (hyperpnoea alternates with apneas) is commonly found in comatose patients, often with *cerebral* disease, but is relatively *non-specific*.

  Rapid, regular respiration is also common in comatose patients and is often found with pneumonia or acidosis.

- **Central neurogenic hyperventilation**

  Brainstem tegmentum (mostly *tumors*):

  ↑ *PO2*, ↓ *PCO2*, and

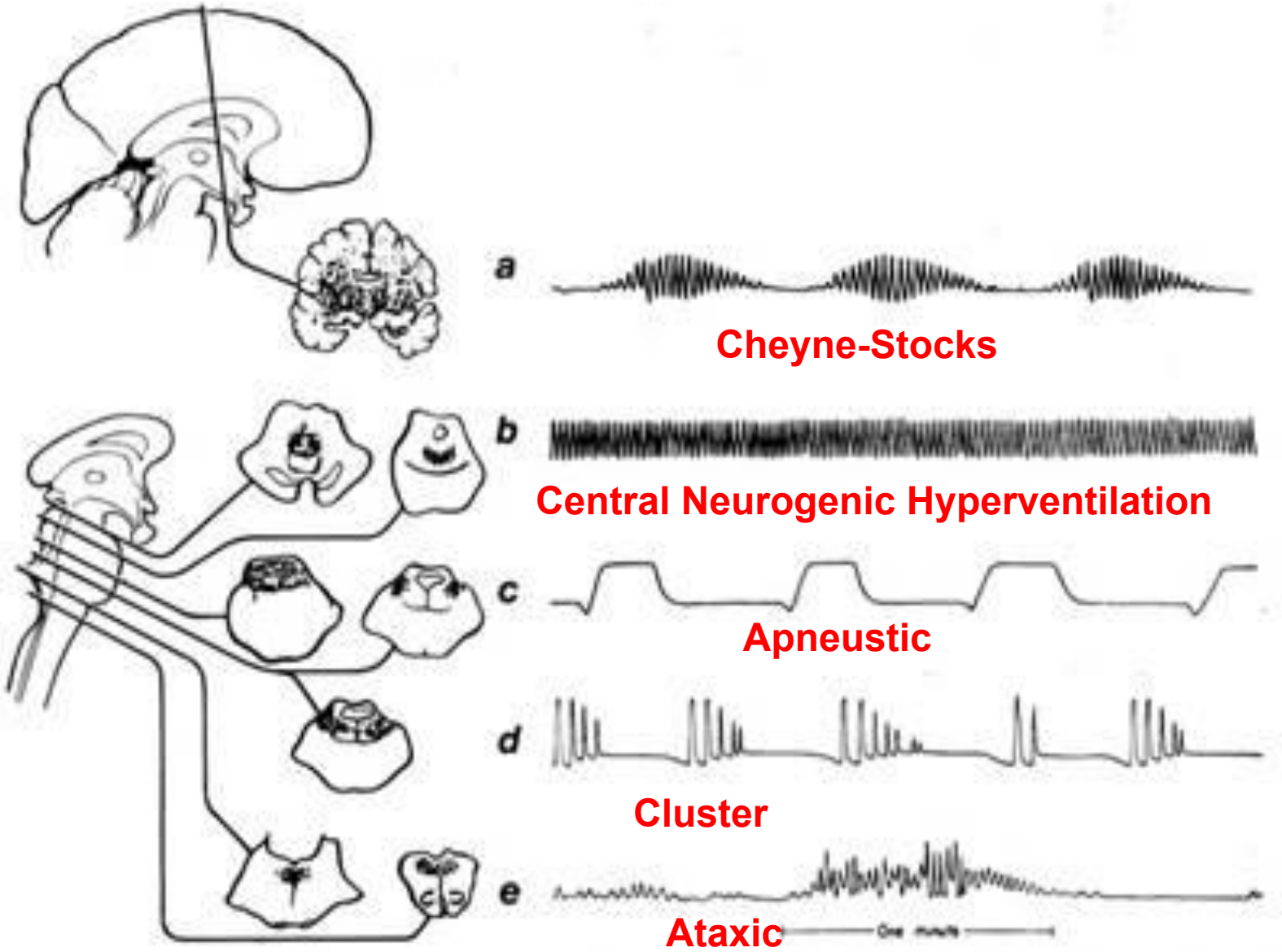
  Respiratory alkalosis in the absence of any evidence of pulmonary disease

Sometimes complicates hepatic encephalopathy

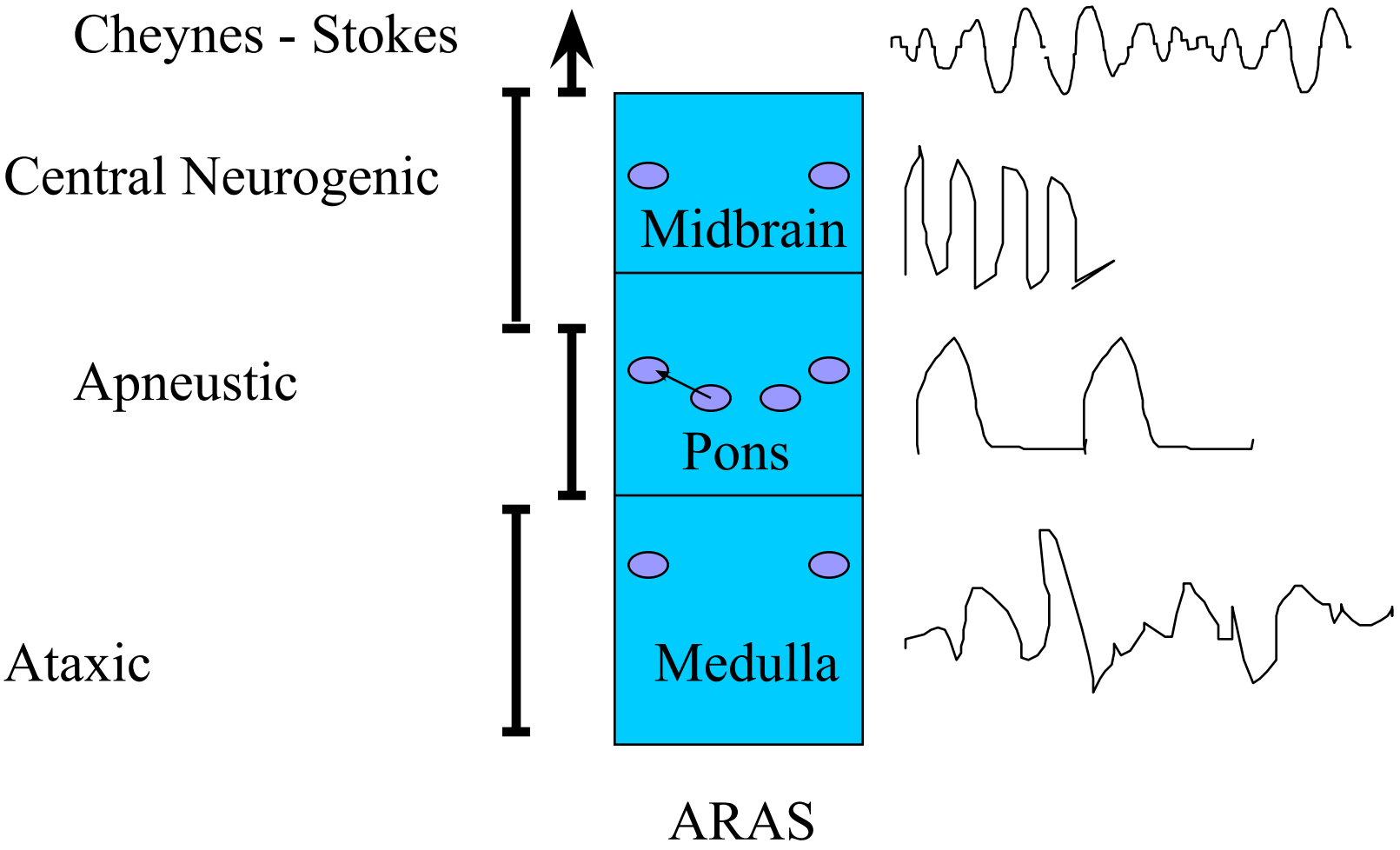
- **Apneustic breathing**

  Brainstem lesions *Pons* may also give with a *pause at full inspiration*
- **Ataxic:**

  Medullary lesions: *irregular respiration* with *random deep and shallow breaths*


a
Cheyne-Stocks
b
Central Neurogenic Hyperventilation
c
Apneustic
d
Cluster
e
Ataxic

Abnormal breathing patterns in coma

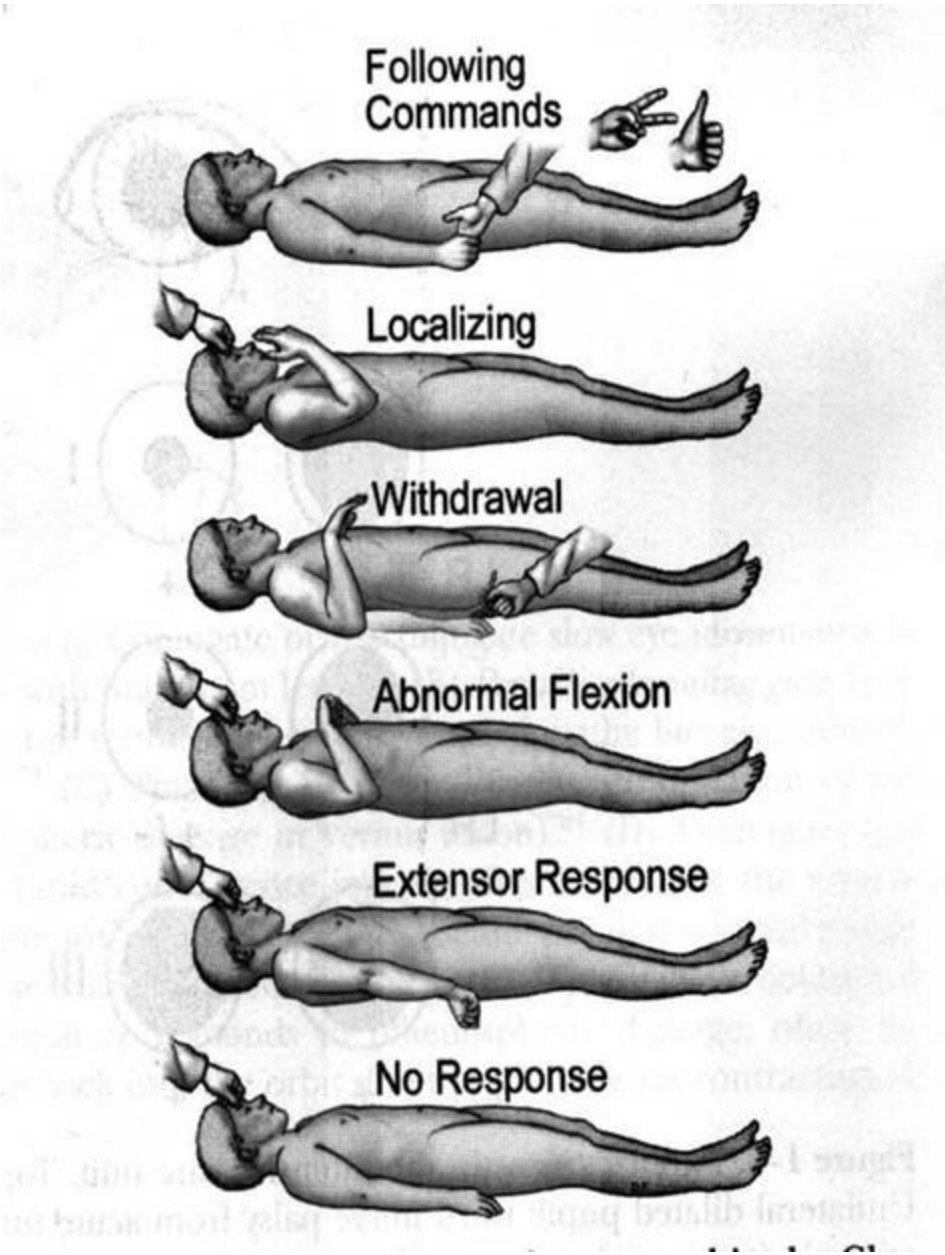


Motor function

- Particular attention should be directed towards asymmetry of tone or movement.
- The plantar responses are usually extensor, but asymmetry is again important.
- The tendon reflexes are less useful.
- The motor response to painful stimuli should be assessed carefully (part of GCS)

- Painful stimuli: supraorbital nerve pressure and nail-bed pressure. *Rubbing of the sternum should be **avoided** (bruising and distress to the relatives)*
- Patients may localize or exhibit a variety of responses, asymmetry is important

- *Flexion* of the *upper* limb with extension of the lower limb (decorticate response) and extension of the upper and lower limb (decerebrate response) indicate a more severe disturbance and prognosis.



Signs of lateralization

- Unequal pupils
- Deviation of the eyes to one side
- Facial asymmetry
- Turning of the head to one side
- Unilateral hypo-hypertonia
- Asymmetric deep reflexes
- Unilateral extensor plantar response (Babinski)
- Unilateral focal or Jacksonian fits

Head and neck

- **The head**

1. Evidence of injury
2. Skull should be palpated for depressed fractures.

- **The ears and nose:** haemorrhage and leakage of CSF
- **The fundi:** papilloedema or subhyaloid or retinal haemorrhages

- **Neck**: In the presence of trauma to the head, associated trauma to the neck should be assumed until proven otherwise.
- Positive Kernig's sign : a meningitis or SAH. *If established as safe to do so, the cervical spine should be gently flexed*
- Neck stiffness may occur:

1. ↑ ICP
2. incipient tonsillar herniation

Causes of COMA

CNS causes of coma

Cerebrovascular disease is a frequent cause of coma.

- Mechanism:

Impairment of perfusion of the RAS

- With hypotension
- Brainstem herniation (parenchymal hge, swelling from infarct, or more rarely, extensive brainstem infarction)

Subarachnoid haemorrhage

Loss of consciousness is common with SAH

- only about 1/2 of patients recover from the initial effects of the haemorrhage.
- Causes of coma:

1. Acute ↑ICP and
2. Later, vasospasms, hyponatraemia

Parenchymal haemorrhage

May cause a rapid decline in consciousness, from

1. Rupture into the ventricles
2. or subsequent herniation and brainstem compression.

- Cerebellar haemorrhage or infarct with

1. Subsequent oedema
2. Direct brainstem compression, early decompression can be lifesaving.

Hypotension

- The critical blood flow in humans required to maintain effective cerebral activity is about 20 ml/100 g/min and any fall below this leads rapidly to cerebral insufficiency.
- The causes:

1. syncope in younger patients
2. cardiac disease in older patients.

Hypertensive encephalopathy

- Now rare with better control of blood pressure.
- C/P: impaired consciousness, grossly raised blood pressure, papilloedema.
- Neuropathologically: fibrinoid necrosis, arteriolar thrombosis, microinfarction, and cerebral oedema (failure of autoregulation)

Raised intracranial pressure

- Mass effects: tumours, abscesses, haemorrhage, subdural, extradural haematoma, brainstem herniation→ distortion of the RAS.
- C/P: depends on normal variation in the tentorial aperture, site of lesion, and the speed of development.

- Herniation and loss of consciousness
  Lesions located deeply, laterally, or in the temporal lobes > located at a distance, such as the frontal and occipital lobes.
- Rate of growth: slowly growing tumours may achieve a substantial size and distortion of cerebral structure without impairment of consciousness, in contrast to small rapidly expanding lesions

- **Central herniation** involves downward displacement of the upper brainstem
- **Uncal herniation** in which the medial temporal lobe herniates through the tentorium

- Central herniation: small pupils are followed by midpoint pupils, and irregular respiration gives way to hyperventilation as coma deepens.
- Uncal herniation: a unilateral dilated pupil, due to compression of the III nerve, and asymmetric motor signs. As coma deepens, the opposite pupil loses the light reflex and may constrict briefly before enlarging.
- Rarely, Upward herniation can occur with posterior fossa masses

Head injury

- The leading cause of death below the age of 45, head injury accounts for 1/2 of all trauma deaths
- A major cause of patients presenting with coma.
- A history is usually available and, if not, signs of injury such as bruising of the scalp or skull fracture lead one to the diagnosis

- Alcohol on the breath provides a direct clue to a cause of coma, evidence of head injury need not necessarily imply that this is the cause.
- Epileptic seizure, may have resulted in a subsequent head injury

- Damage can be diffuse or focal.
- Rotational forces of the brain cause surface cortical contusions and even lacerations, most obvious frontotemporally because of the irregular sphenoidal wing and orbital roof.
- Subdural bleeding due to tearing of veins

- Diffuse axonal injury is now seen as the major consequence of head injury and associated coma.
- Mild degrees of axonal injury also occur with concussion and brief loss of consciousness

- Secondary damage can occur from parenchymal haemorrhage, brain oedema, and vascular dilatation, all of which will lead to ↑ICP→ ↓perfusion pressure, which can be accentuated by systemic hypoxia and blood loss.
- Subdural and extradural haematomata may cause impairment of consciousness following apparent recovery are important to diagnose, as they are readily treatable surgically.

Infections

- Systemic infections may result in coma as an event secondary to metabolic and vascular disturbance or seizure activity.
- Direct infections of the CNS, as with meningitis and encephalitis, can all be associated with coma.
- *Meningitis:* the onset is usually subacute, intense headache, associated with fever and neck stiffness. meningococcal meningitis may be rapid in onset

- Diagnosis is confirmed by identifying the changes in the CSF, from which it may be possible to isolate the causative organism.
- Prompt treatment of acute meningitis is, however, imperative and may precede diagnostic confirmation.
- *Encephalitis:* usually subacute, and often associated with fever and/or seizures, herpes simplex encephalitis may be explosive at onset, leading to coma within a matter of hours Treatment with aciclovir, precedes definitive diagnosis.

Parasitic infections

Cerebral malaria

- 25 % mortality rate.
- Associated with 2–10 % of cases of infection with *Plasmodium falciparum*.
- C/P: acute profound mental obtundation or psychosis, leading to coma with extensor plantar responses
- CSF: may show increased protein, characteristically there is no pleocytosis

- Hypoglycaemia and lactic acidosis, which may contribute to the coma.
- Treatment: intravenous *quinine*.

Steroids, *which were at one time prescribed widely for oedema, are now contraindicated as they prolong the coma*.

Septic patients

- Commonly develop an encephalopathy.
- In some patients this can be severe, with a prolonged coma.
- Lumbar puncture in such patients is usually normal or only associated with a mildly elevated protein level.
- EEG is valuable and is abnormal, ranging from diffuse theta through to triphasic waves and suppression or burst-suppression

- Although there is a high mortality, there is the potential for complete reversibility
- Presence of coma should not prevent an aggressive approach to management of such patients including, for example, haemodialysis to deal with acute renal failure

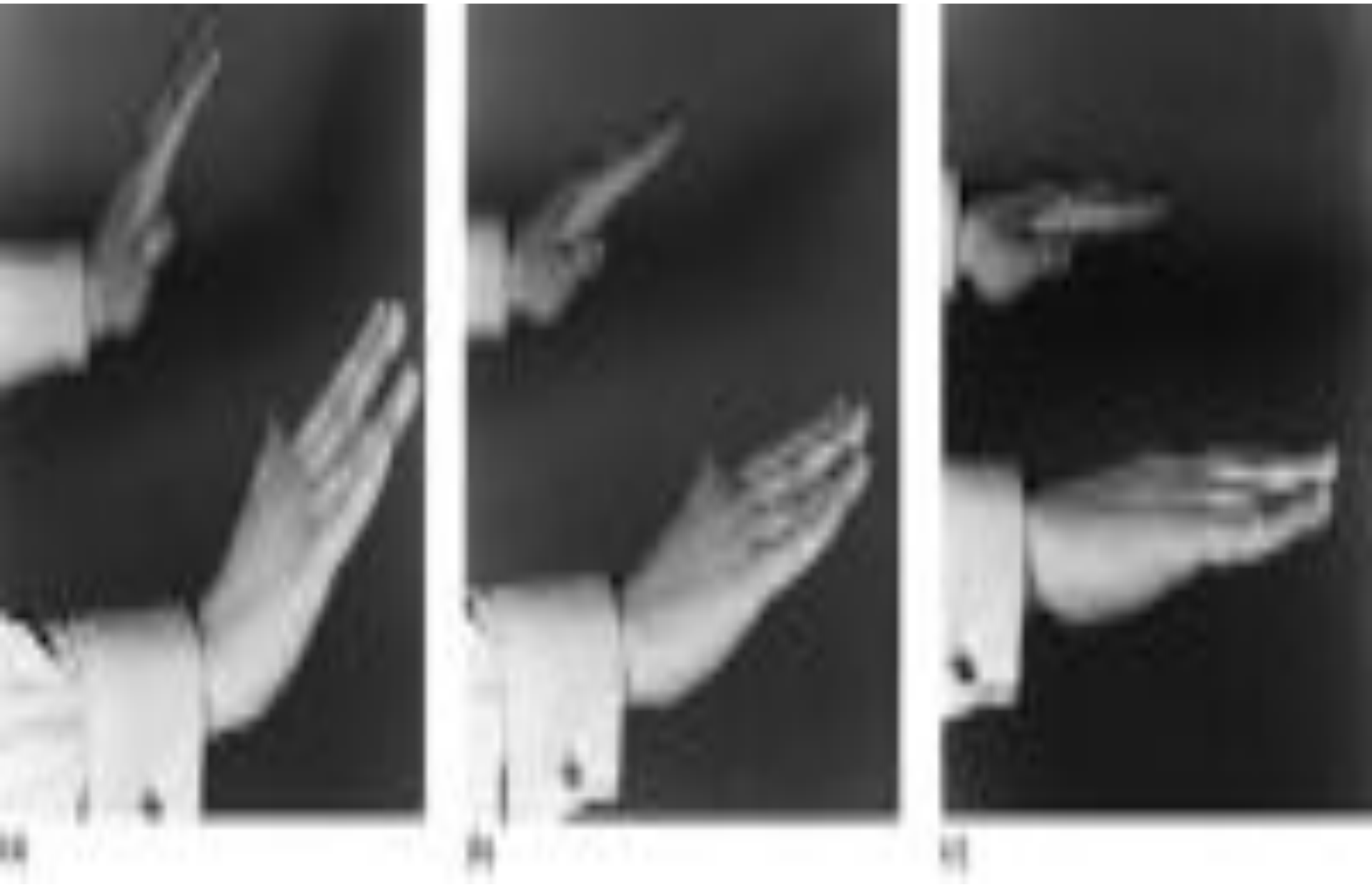
Metabolic causes of coma

Hepatic coma

The patient is known to be suffering from liver failure

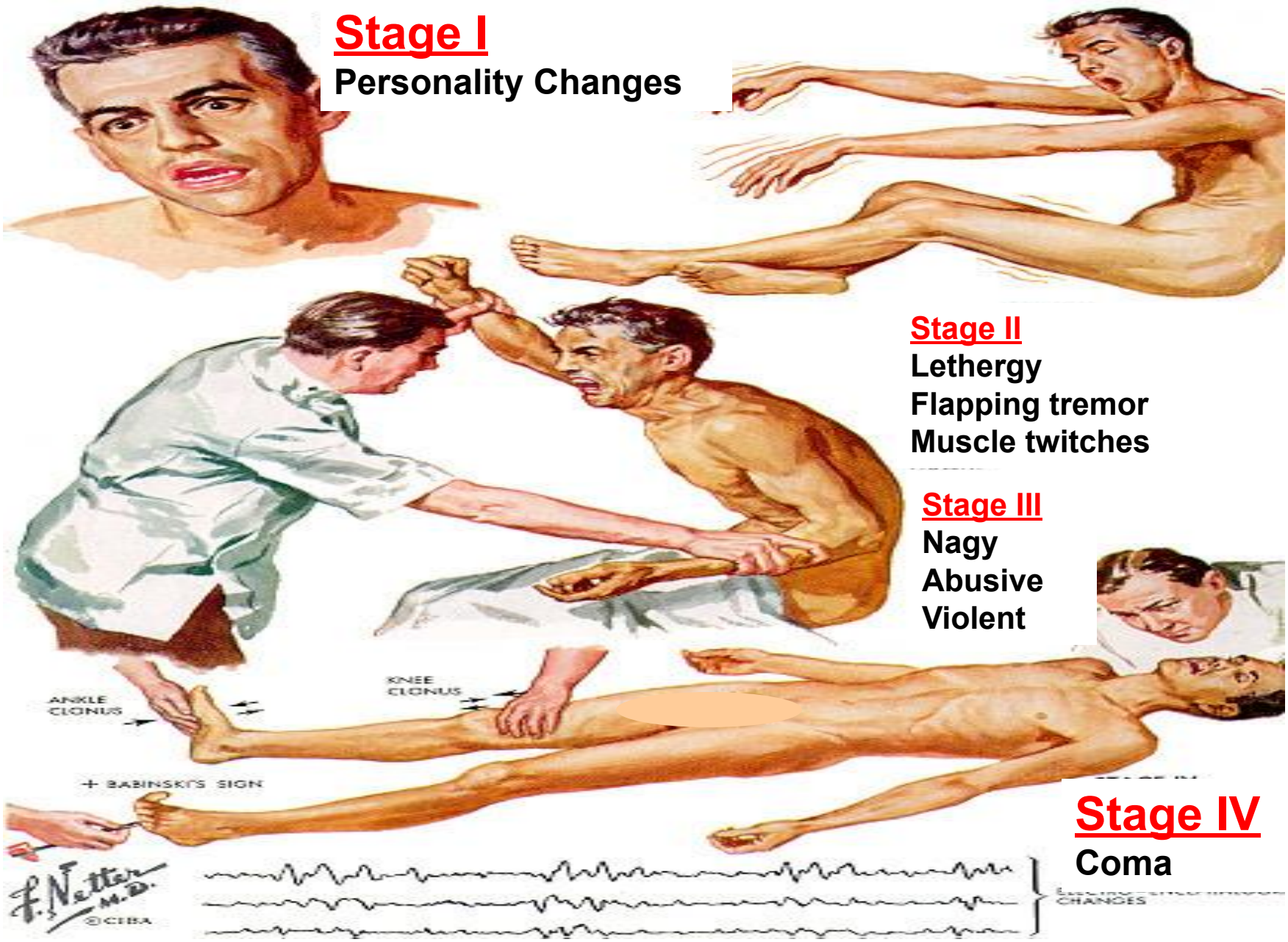
- May occur in patients with chronic liver failure and portosystemic shunting (In these cases jaundice may be absent)

- Precipitation: GIT hge, infection, certain diuretics, sedatives, analgesics, general anaesthesia, high-protein food or ammonium compounds
- Subacute onset, although it can be sudden, with an initial confusional state often bilateral asterixis or flapping tremor.
- Asterixis, a -ve myoclonus jerk, results in sudden loss of a maintained posture. elicited by asking the subject to maintain extension at the wrist



- As coma supervenes, there is often decerebrate and/or decorticate posturing with extensor plantar responses
- Diagnosis: signs of liver disease hepatic fetor, and biochemical evidence of disturbed liver function. EEG with paroxysms of bilaterally synchronous slow waves in the delta range or with occasional triphasic waves

- The disturbance of consciousness due to raised ammonia, and indeed treatments to reduce ammonia
- endogenous benzodiazepine ligands may contribute to the hepatic coma, benzodiazepine antagonist, flumazenil, in hepatic coma would support this view


Stage I
Personality Changes
Stage II
Lethergy
Flapping tremor
Muscle twitches
Stage III
Nagy
Abusive
Violent
ANKLE CLONUS
KNEE CLONUS
+ BABINSKI'S SIGN
Stage IV
Coma
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CHANGES

Renal coma

- May occur in acute or chronic renal failure
- Raised blood urea alone cannot be responsible for the loss of consciousness but the
- Metabolic acidosis, electrolyte disturbances and Water intoxication due to fluid retention may be responsible

- Early symptoms Headache, vomiting, dyspnoea, mental confusion, drowsiness or restlessness, and insomnia
- Later muscular twitchings, asterixis, myoclonus, and generalized convulsions are likely to precede the coma.
- ↑ blood urea or creatinine establishes the diagnosis (DD hypertensive encephalopathy)

- Dialysis may develop iatrogenic causes of impaired consciousness.

Dialysis disequilibrium syndrome

1. Is a temporary, self-limiting disorder, but it can be fatal
2. More common in *children* and during *rapid changes* in blood solutes. *Rapid osmotic shift of water into the brain is the main problem*

3. accompanied by headache, nausea, vomiting, and restlessness before drowsiness and marked somnolence.
4. It can occur during or just after dialysis treatment, but resolves in 1 or 2 days

Dialysis encephalopathy dialysis dementia syndrome

1. Progressive dysarthria, mental changes,
2. progression to seizures, myoclonus, asterixis, and focal neurological signs
3. terminally, there may be coma

4. EEG: paroxysmal bursts of irregular, generalized spike and wave activity.
5. has been attributed to the neurotoxic effects of aluminium: aluminium-containing *antacids* and a high aluminium content in the *water*
6. *Reached its peak prevalence in the mid 1970s, before preventive action was taken.*

Disturbance of glucose metabolism

Diabetic Ketoacidosis

- Subacute onset with late development of coma.
- Marked ketoacidosis, usually above 40 mmol/l, together with ketonuria.
- Secondary lactic acidosis (DD severe anoxia or methyl alcohol or paraldehyde poisoning)
- Patients are dehydrated, rapid, shallow breathing, occasionally acetone on the breath.
- The plantar responses are usually flexor until coma supervenes.

Hyperglycaemic non-ketotic diabetic coma

- More commonly seen in the elderly.
- Coma is more common than with ketoacidosis.
- Profound cellular dehydration, risk of developing *cerebral venous thrombosis*, which may contribute to the disturbance of consciousness.
- It may be induced by drugs, acute pancreatitis, burns, and heat stroke

Hypoglycaemic coma

- Much more rapid onset.
- Symptoms appear with blood sugars of less than 2.5 mmol/l
- Initially autonomic: sweating and pallor, and then inattention and irritability progressing to stupor, coma, and frequent seizures.
- May present with a focal onset (hemiparesis)
- Plantar responses are frequently extensor.
- Patients may be hypothermic.

Diagnosis of Hypoglycemic Coma:

- The patient is known to be taking insulin.
- Spontaneous hypoglycaemia with insulinomas are usually diagnosed late.
- There may be a long history of intermittent symptoms and in relation to fasting or exercise.
- May also be *precipitated by* hepatic disease, alcohol intake, hypopituitarism, and Addison's disease

Treatment:

- Glucose, together with thiamine
- Unless treated promptly, hypoglycaemia results in irreversible brain damage. *Cerebellar Purkinje cells*, the cerebral cortex, and particularly the *hippocampus and basal ganglia* are affected
- Dementia and a cerebellar ataxia are the clinical sequelae of inadequately treated hypoglycaemia.

Other endocrine causes of coma

Pituitary failure

- Rare cause of coma and is the result of hypoglycaemia, hypotension, hypothermia, and impaired adrenocortical function
- History of fatigue, occasionally depression and loss of libido
- Patients are very sensitive to infections and to sedative drugs, which often precipitate impaired consciousness.

- Pituitary apoplexy Acute onset of hypopituitarism occurs with haemorrhagic infarction in pre-existing tumours, patients present with impaired consciousness, meningism, and opthalmoplegia

Hypothyroidism

- Mental symptoms are common, with headaches, poor concentration, and apathy; *this is frequently diagnosed as depression*.
- With progression there is increasing somnolence and, patients become sensitive to drugs and infections.
- These and cold weather, particularly in the elderly, may precipitate **myxoedemic coma**.

- Myxoedemic coma has a high mortality and is associated with hypoglycaemia and hyponatraemia.
- low-reading thermometer to detect hypothermia
- Treatment: support of ventilation and blood pressure and cautious correction of the thyroid deficiency with tri-iodothyronine

Hyperthyroidism

- Mild mental symptoms: anxiety, restlessness,reduced attention.
- 'Thyroid storm' with agitated delirium, which can progress to coma, may have bulbar paralysis
- Apathetic form of thyrotoxicosis: particularly the elderly, with depression leading to apathy, confusion, and coma without any signs of hypermetabolism

Adrenocortical failure

- Mental changes are common in Addison's disease and secondary hypoadrenalism.
- Undiagnosed Addison's disease is frequently associated with behavioural changes and fatigue.
- Infection or trauma may precipitate coma and associated hypotension, hypoglycaemia, and dehydration

- Tendon reflexes are often absent
- ↑ ICP, papilloedema
- Friedrichsen–Waterhouse syndrome acute adrenal failure due to meningococcal septicaemia a cause of sudden coma in infants.
- Acute adrenal failure due to HIV infection can occur

Disturbance of Ca and Mag metabolism

Hypercalcaemia

- Mental confusion, apathy, often with headache. If severe, stupor and even coma.
- Causes: metastatic bone disease, including multiple myeloma

Hypocalcaemia

- Primarily affects the peripheral nervous system, with tetany and sensory disturbance
- It can be associated with ↑ICP and papilloedema

Hypomagnesaemia

- Inadequate intake and prolonged parenteral feeding,
- Overshadowed by other metabolic disturbances, including hypocalcaemia, but can give rise to a similar clinical picture.

Hypermagnesaemia

- Renal insuf., overzealous replacement of mag and its use (in eclampsia) can give rise to mag intoxication, with major CNS depression.

Drugs

- Poisoning, drug abuse, and alcohol intoxication accounting for up to 30 % of those presenting through accident and emergency departments.
- 80 % require only simple observation in their management.

- **The most commonly drugs in suicide attempts are :**

1. Benzodiazepines
2. Paracetamol
3. antidepressants.

- **Narcotic overdoses** (heroin)

1. *Pinpoint pupils*
2. *Shallow respirations* , *needle marks*.
3. The coma is easily reversible with *naloxone*

- **Solvent abuse and glue sniffing** should be considered in the undiagnosed patient with coma.
- Drugs may also result in disturbed consciousness due to

1. secondary metabolic derangement
2. the acidosis associated with *ethylene glycol* and carbon monoxide poisoning

Alcohol intoxication

- Apparent from the history, flushed face, rapid pulse, and low blood pressure. The smell of alcohol on the breath.
- Intoxicated are at increased risk of hypothermia and of head injury can be the cause of coma.
- At low plasma concentrations of alcohol, mental changes, at higher levels, coma ensues, >350 mg/dl may prove fatal.

Miscellaneous causes of coma

Seizures

- Common cause of coma, with a period of unconsciousness following a single generalized seizure commonly lasting between 30 and 60 minutes.
- Following status epilepticus, there may be a prolonged period of coma. *History,* trauma to the tongue or inside of the mouth.
- Seizures secondary to metabolic disturbances may have a longer period of coma.

Extensive neurological disease

- PMLE
- severe end-stage multiple sclerosis.
- Prion disease may lead to coma over a short period of 6–8 weeks, but this is following a progressive course of widespread neurological disturbance.

Eclampsia

- In the second half of pregnancy and represents a failure of autoregulation, with raised blood pressure.
- Neuropathologically: there are ring haemorrhages around occluded small vessels with fibrinoid deposits.

- CP: seizures, cortical blindness, and coma.
- Management: control of convulsions and raised blood pressure. Parental magnesium is commonly employed, may give rise to hypermagnesaemia.

Postpartum complications of pregnancy

cerebral angiitis and venous sinus thrombosis, may also lead to coma

Investigation of coma

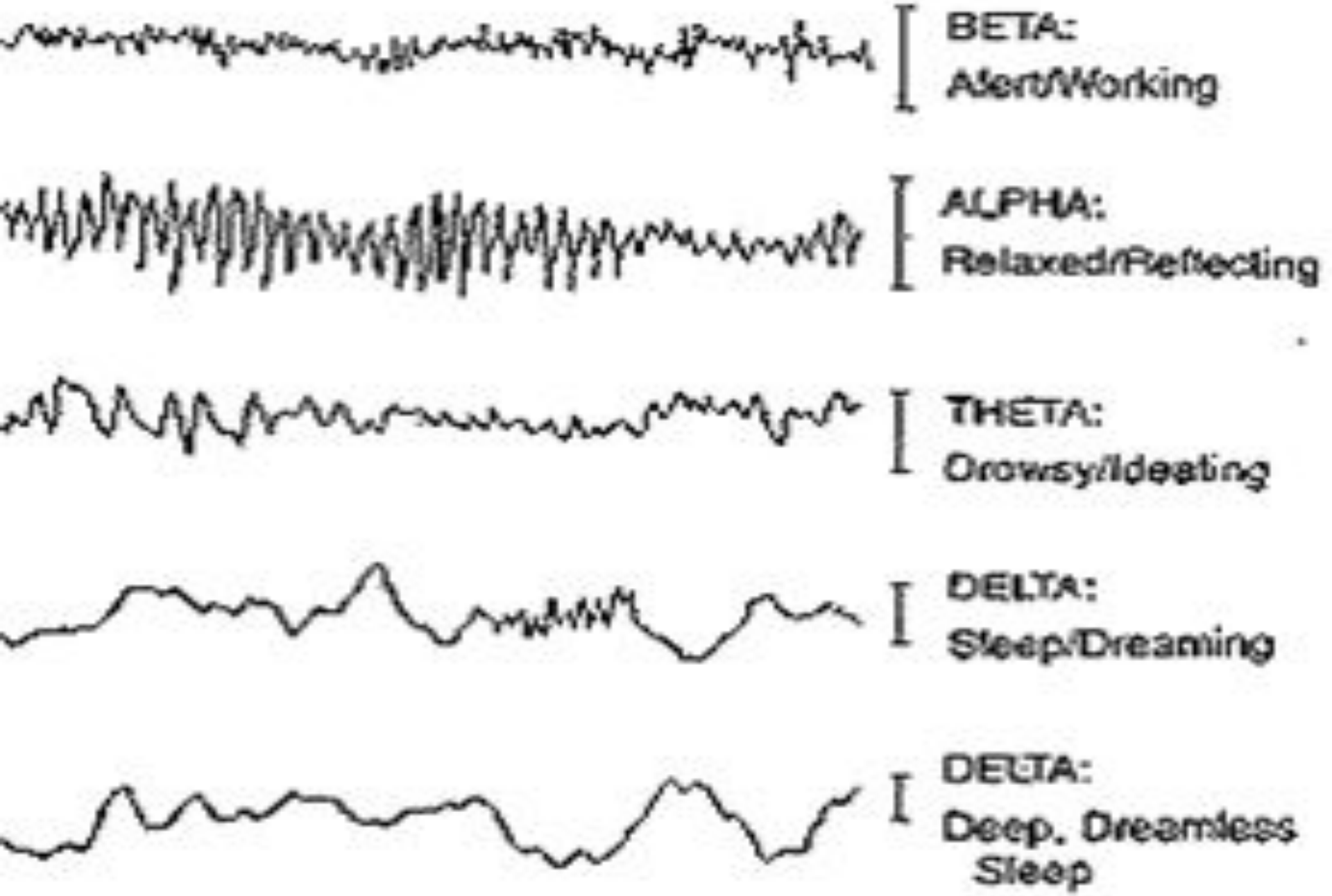
- At presentation blood will be taken for determination of glucose, electrolytes, liver function, calcium, osmolality, and blood gases.
- Blood should also be stored for a subsequent drug screen if needed

- Following the clinical examination, a broad distinction between a metabolic cause, with preserved pupillary responses, or a structural cause of coma is likely to have been established
- Although most patients with coma will require CT scanning, or indeed all with persisting coma, clearly this is of greater urgency when a structural lesion is suspected

- In the absence of focal signs, but with evidence of meningitis, a lumbar puncture may need to be performed before scanning, as a matter of clinical urgency.
- In other situations, lumbar puncture should be delayed until after the brain scan because of the risk of precipitating a pressure cone secondary to a cerebral mass lesion

- All patients will require chest radiography and ECG, detailed investigations of systemic disease will be directed by the clinical examination.
- The EEG is of value in identifying the occasional patient with subclinical status epilepticus, and is clearly of value in assessing the patient who has been admitted following an unsuspected seizure

- Fast activity is commonly found with drug overdose and slow wave abnormalities with metabolic and anoxic coma.
- An isoelectric EEG may occur with drug-induced comas, but otherwise indicates severe cerebral damage.


BETA:
Alert/Working
ALPHA:
Relaxed/Reflecting
THETA:
Drowsy/Ideating
DELTA:
Sleep/Dreaming
DELTA:
Deep, Dreamless
Sleep

Management of the unconscious patient

- Treatment of the underlying cause
- Maintenance of normal physiology: respiration, circulation, and nutrition
- Patient should be nursed on his or her side without a pillow
- Attention will clearly need to be paid to the airway, requiring an oral airway as a minimum

- Intubation, if coma is prolonged, tracheostomy
- Retention or incontinence of urine will require catheterization
- Intravenous fluid is necessary and, if coma persists, adequate nutrition is required.
- Care of Skin, frequent changing of position, special mattress, avoid urine and stool soiling and good care of bed sores

Prognosis in coma

- In general, coma carries a serious prognosis.
- This is dependent to a large extent on the underlying cause.
- Coma due to depressant drugs carries an excellent prognosis provided that resuscitative and supportive measures are available and no anoxia has been sustained
- Metabolic causes, apart from anoxia, carry a better prognosis than structural lesions and head injury

- Length of coma and increasing age are of poor prognostic significance.
- Brainstem reflexes early in the coma are an important predictor of outcome
- in general, the absence of pupillary light and corneal reflexes 6 hours after the onset of coma is very unlikely to be associated with survival

- The chronic vegetative state usually carries a uniformly poor prognosis, although a partial return of cognition, or even restoration to partial independence, has been reported very rarely.
- Although unassociated with coma, the 'locked-in' syndrome also carries a poor prognosis, with only rare recoveries reported.


THANK YOU
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