An approach to coma

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Consciousness

- dependent on the function of two separate anatomical and physiological syetems:

1. The ascending reticular activationg sys(ARAS) projecting from brainstem to thalamus.{determines arousal}(the level of consciousness)
2. The cerebral cortex: determines the content of consciousness

Impaired functioning of either anatomical system may cause coma

Disturbed consciousness: def

- Coma- a state of unrousable unresponsiveness.
- Level of consciousness represents a continuum between being alert and deeply conmatose.
- It may be qualified using the GCS
- Coma→ GCS < 8

Table 22.10 Glasgow Coma Scale

Eye opening (*E*)	
Spontaneous	4
To speech	3
To pain	2
No response	1
Motor response (*M*)	
Obeys	6
Localizes	5
Withdraws	4
Flexion	3
Extension	2
No response	1
Verbal response (*V*)	
Orientated	5
Confused conversation	4
Inappropriate words	3
Incomprehensible sounds	2
No response	1
Glasgow Coma Scale = *E* + *M* + *V* (GCS minimum = 3; maximum	

- Glasgow Coma Scale
- EMV=minimum 3
- Maximum=15

delirium

- The term used to describe a confusional state in which reduced attention is a cardinal feature, usually with altered behavior, coginition , orientation and a fluctuating level of consciousness from agitation to hypoarousal

Stupor and obtundation

- No longer use

Principle causes

- Diffuse brain dysfunction
- Direct effect within brainstem
- Pressure effect on brainstem

Diffuse brain dysfunction

- Drug overdose
- Encephalitis, meningitis, cerebral malaria
- SAH
- CO poisoning
- Trauma to brain
- Hypo,hyperglycemia
- Organ failure- severe uraemia,hepatic encephalopathy,respira

Continue:

- Hypercalcaemia, hypoCa
- Hypoadrenalism, hypopit and hypothyroidism
- Hyponatraemia, hypernatraemia
- Metabolic acidosis
- Hypothermia, hyperpyrexia
- Seizures-post epileptic state, non-convulsive state

Continue;

- Metabolic rarities eg porphyria
- Extensive cortical damage
- Hypoxic ischaemic brain injury eg cardiac arrest

Direct effect within brain stem

- Brainstem haemorrhage, infarction or demyelination
- Brainstem neoplasm eg glioma
- Wernicke-korsakoff syndrome

Pressure effect on brainstem

- Tumor, massive hemisphere infarction with edema
- Haematoma,
- Abscess
- Cerebellar mass

mechanism

- Altered consciousness is produced by four mechanisms affecting the ARAS in the brainstem or thalamus, and / or widespread impairment of cortical function

- Brain stem lesion- a discrete brainstem or thalamic lesion, eg stroke may damage the ARAS
- Brainstem compression: a supratentorial mass lesion within the brain compresses the brainstem, inhibiting the ARAS, ge coning from a brain tumour or haemorrhage. Mass lesion within the post fossa→ prone to cause and hydrocephalous

Diffuse brain dysfunction

- Diffuse brain dysfunction: generalized severe metabolic or toxic disorders(eg alcohol, sedatives, uraemia, hypercapnia,depress cortical and ARAS function)

Massive cortical damage

- Unlike brainstem lesion, extensive damage of the cerebral cortex and cortical connections is required to cause coma, eg meningitis or hypoxic-ischaemic damage after cardiac arrest
- A single focal hemisphere or cerebellar lesion does not reduce coma unless it compresses the brain stem.
- Cerebral edema frequently surrounds masses, increasing their pressure effects.

Commonest causes of coma are:

- Metabolic disorder 35%
- Drug and toxin- 25%
- Mass lesion 20%
- Others- including trauma, stroke and CNS infectiona

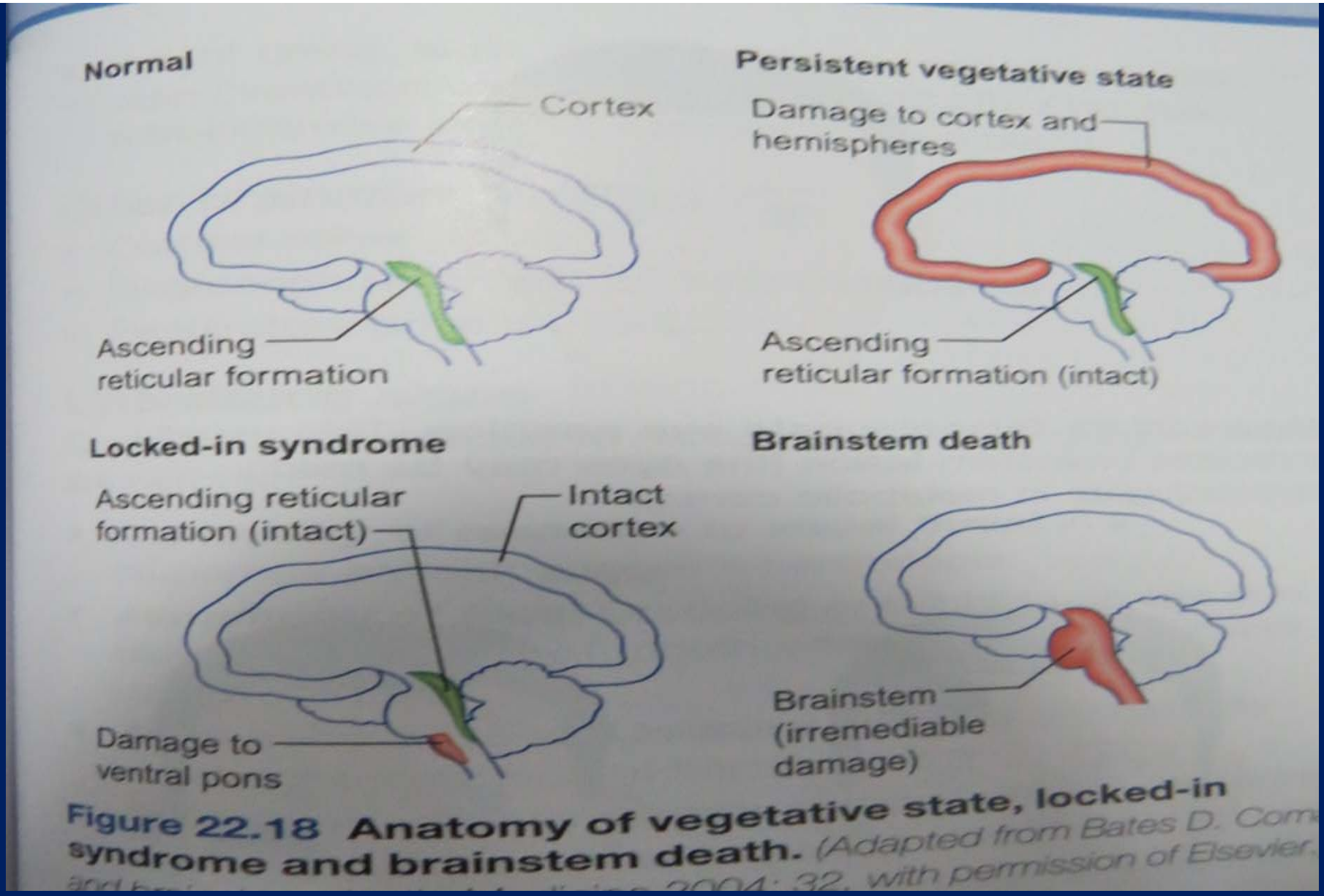


Figure 22.18 Anatomy of vegetative state, locked-in syndrome and brainstem death. *(Adapted from Bates D. Com... and brain... Medicine 2004; 32, with permission of Elsevier.*

Immediate assessment and management

- Check the airway, breathing and circulation
- Stix for blood glucose: if hypo- give glucose (25ml 50%)
- Treat seizures with buccal midazolam and if not terminated, intravenous phenytoin
- If there is fever and meninism: give IV antibiotics

  check ICT malaria or blood film

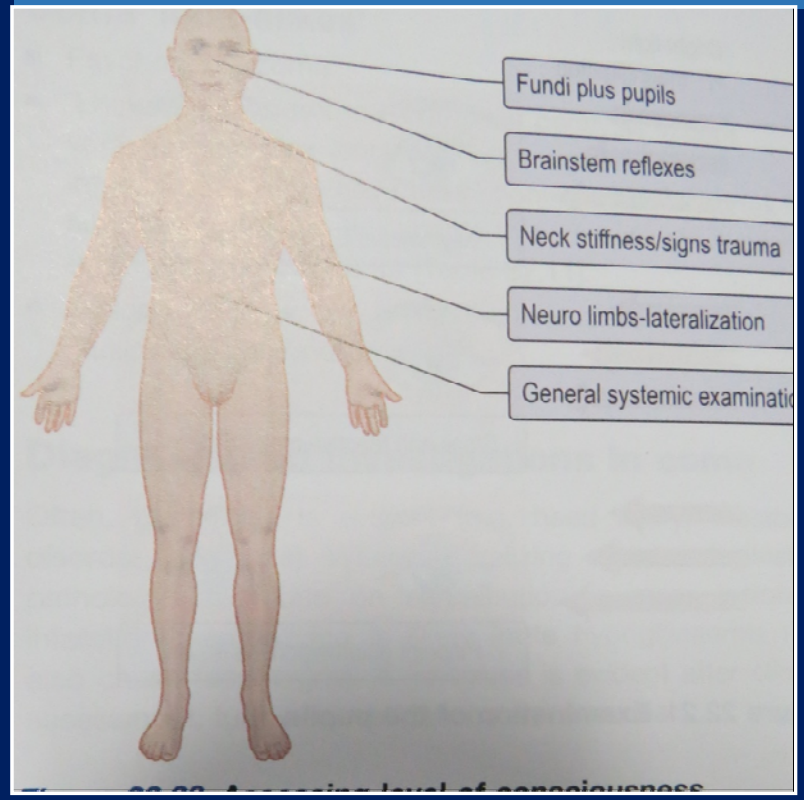
If alcohol

- Thiamine- dose IV parbrinex 1 pair 250mg 8hrly for W-K Syn
- Naloxone
- flumazenil

Obtain as much history as possible

- Limited history is one of the problems faced in assessing the unconscious patient.
- What were the circumstances?
- Ask paramedics, police, and witnesses
- Contact the patient's relatives, friends and GP and
- Obtain past hospital notes, drug details, bottles, identifications data

General and neurological examination



Fundi plus pupils
Brain stem reflex
Neck stiffness/ sign of trauma
Neuro limbs – lateralization
General system examination

General and neurological examination(2)

- Temp, check for meningism
- Sniff the patient's breath for ketones, alcohol and hepatic fetor
- Survey the skin for signs of trauma or spinal injury,
- rash (meningococcal sepsis) jaundice or stigmata of chronic liver diseases, cyanosis, injection marks
- Respiratory pattern-

Respiratory pattern

- Cheyne-stroke (alternating hyperpnoea and periods of apnoea indicating bilateral cerebral or upper brainstem dysfunction or acidotic (Kussmaul respiration(deep,sighing hyperventilation seen in diabetic ketoacidosis and uraemia)

neuro

- Aim: GCS, Brainstem function, lateralization of pathology
- GCS: repeated regularly →progressively declining
- Use painful stimulus→ nail bed pressure or central area, record best response
- Shout commands

fundi

- Look for papilloedema and subhyaloid retinal haemorrhage (SAH)

Brainstem function

- Pupils
- Record size and reaction to light
- Asymmetric -3rd N palsy
- Small and reactive_metabolic
- Pinpoint- opiate/ pontine

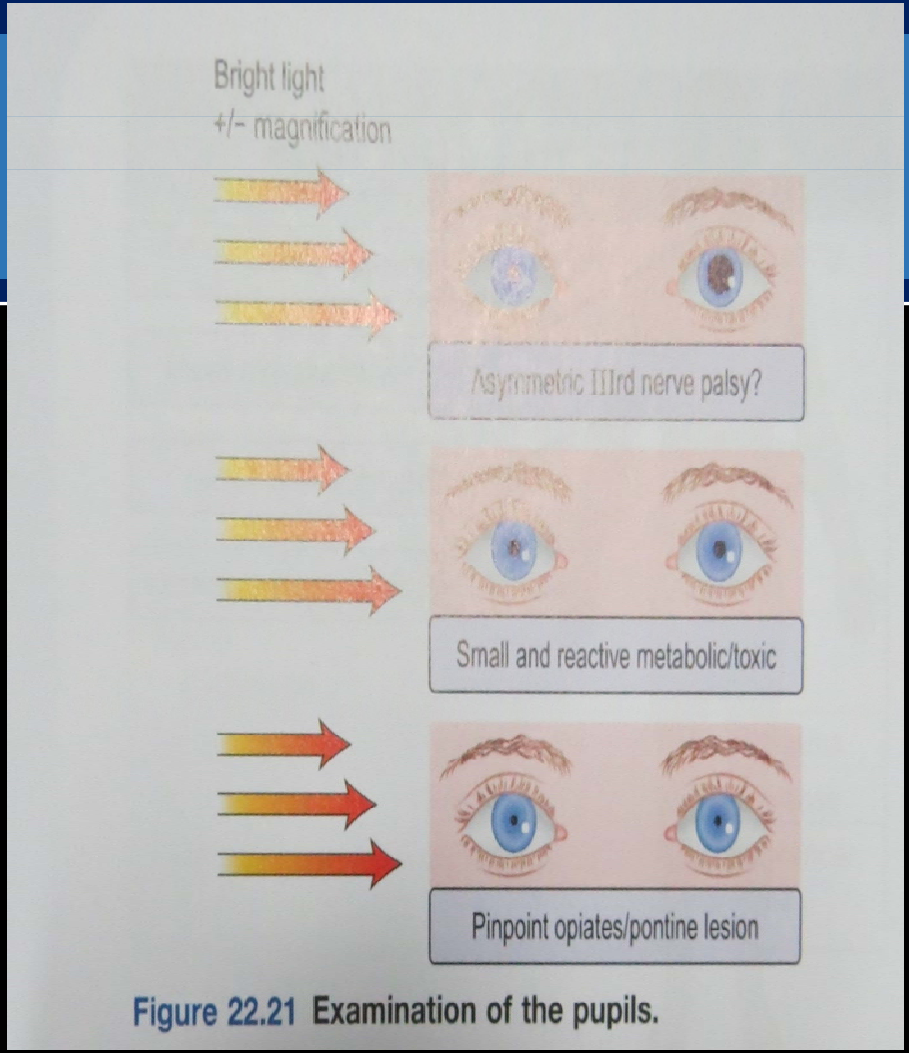


Figure 22.21 Examination of the pupils.

- 3rd nerve –potential neurosurgical emergency
- Bilateral mid point Reactive pupils – normal pupils – sedative except opiate
- Bilateral light-fixed, dilated- cardinal sign of brain death, it can occur in deep coma of any cause esp barbiturate intoxication, hypothermia

- Bilateral pin point light-fixed pupils – pontine lesions(haemorrhage) and opiate
- Mydriatic drugs and previous pupillary surgery can cause diagnostic difficulty

Eye movement and position

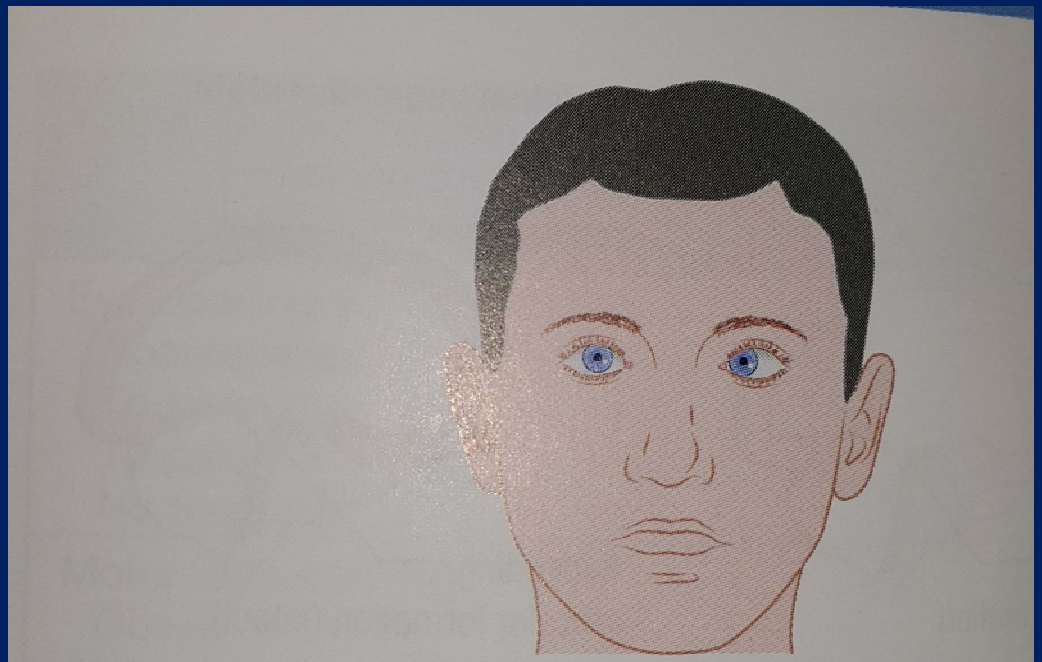


Figure 22.23 Dysconjugate eye position. This u
indicates brainstem lesion (the eyes may be mild
dysconjugate in metabolic coma).

- Dysconjugate eye position:
- This usually indicates brainstem lesion (the eyes may be mildly dysconjugate in metabolic coma)
- Divergent ocular axes eg skew deviation(one side up and one side down)

Conjugate gaze deviation

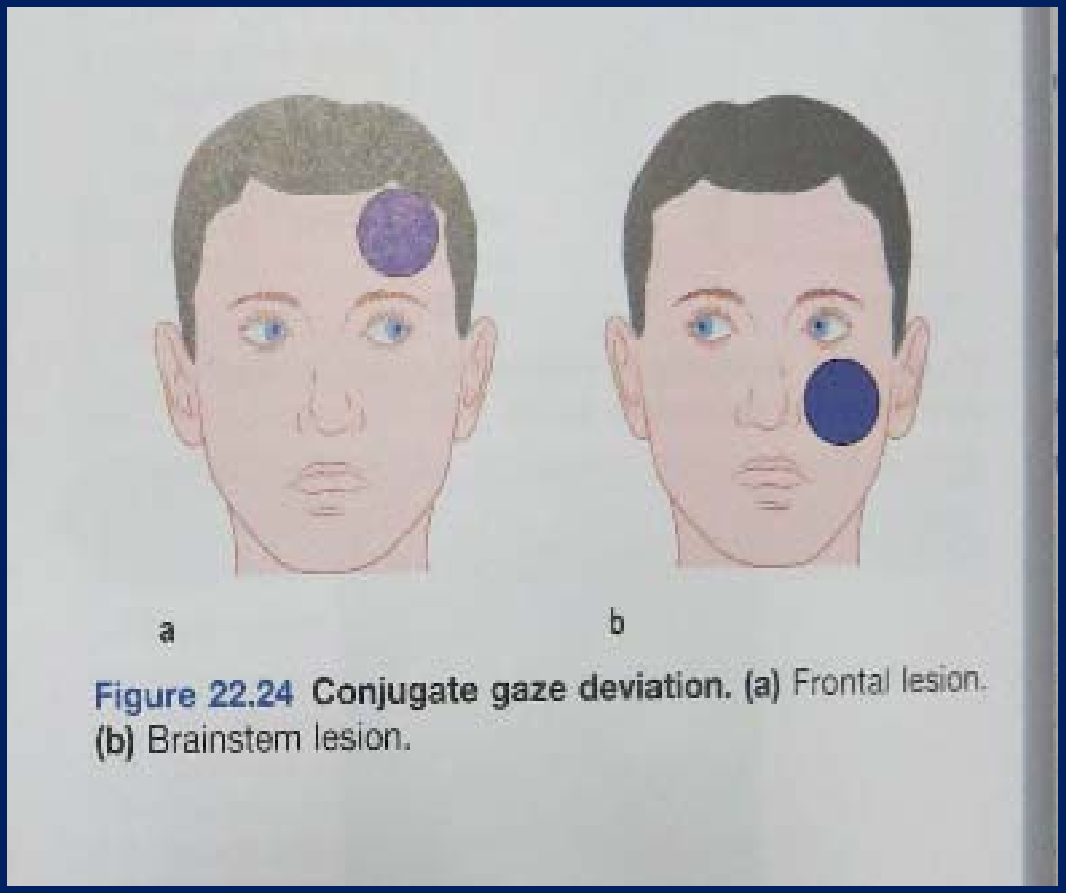


Figure 22.24 Conjugate gaze deviation. (a) Frontal lesion. **(b)** Brainstem lesion.

- Towards the lesion in the frontal lobe and the normal limbs (unopposed activity of the intact frontal eye fields drives eyes to the opposite side)

Conjugate gaze deviation

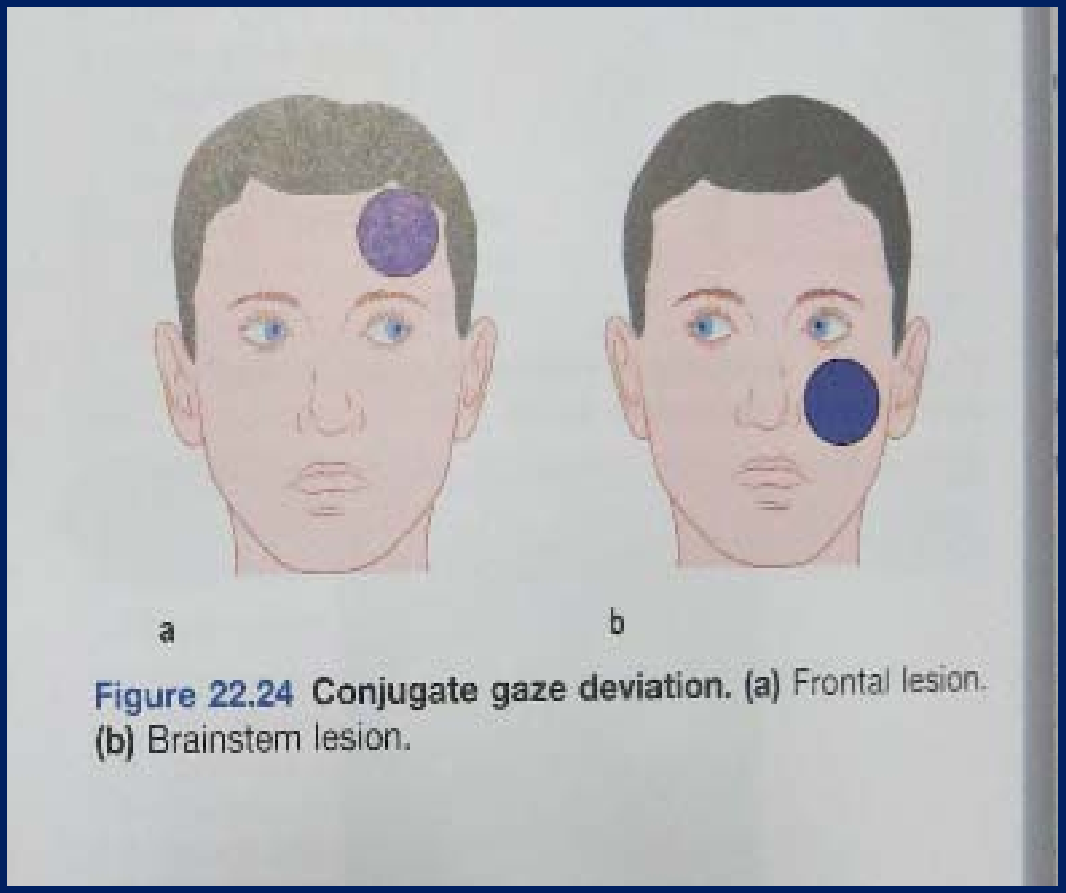


Figure 22.24 Conjugate gaze deviation. (a) Frontal lesion. **(b)** Brainstem lesion.

- Away from the lesion in the brainstem and towards the weak limbs (PPRF in the pons controls lateral gaze to the ipsilateral side)

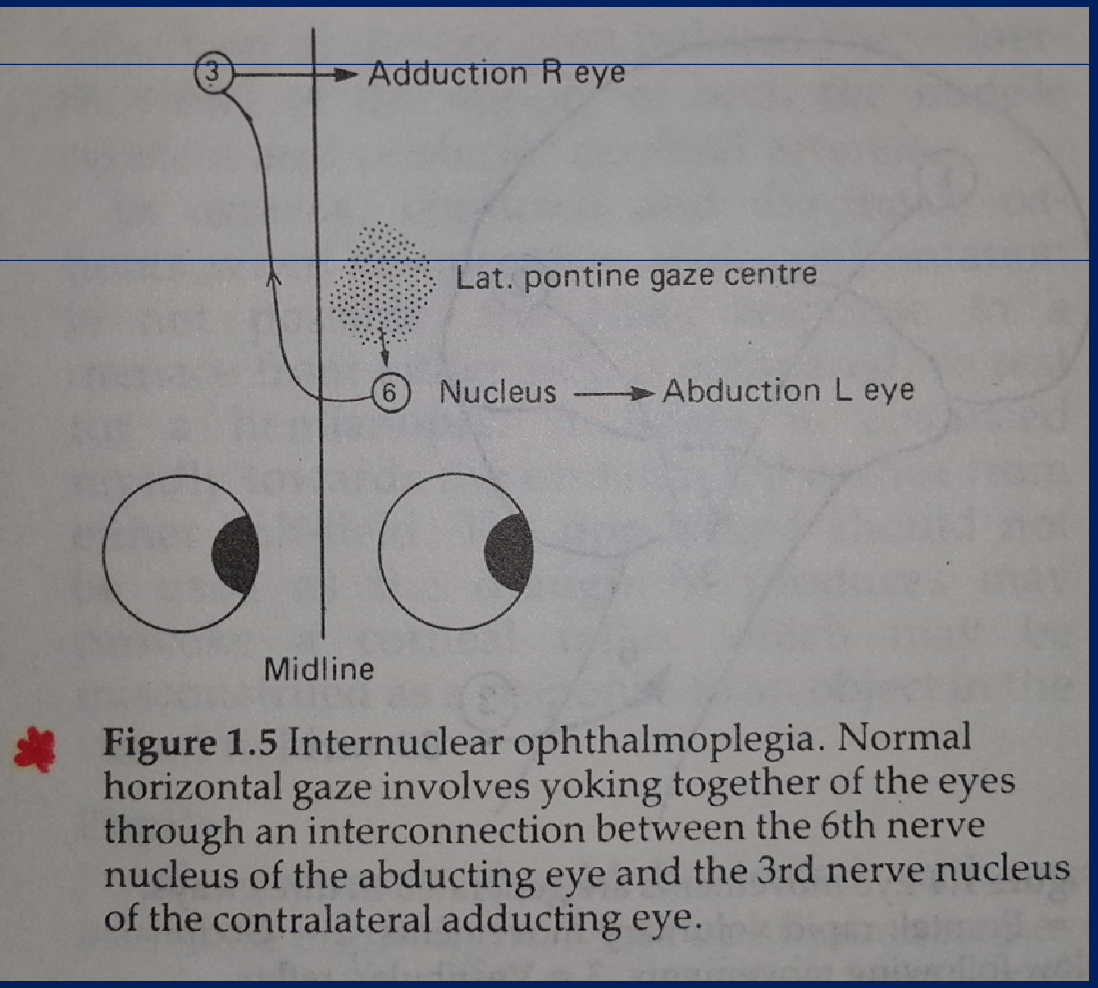


Figure 1.5 Internuclear ophthalmoplegia. Normal horizontal gaze involves yoking together of the eyes through an interconnection between the 6th nerve nucleus of the abducting eye and the 3rd nerve nucleus of the contralateral adducting eye.

- Paramedium pontine reticular formation (PPRF)

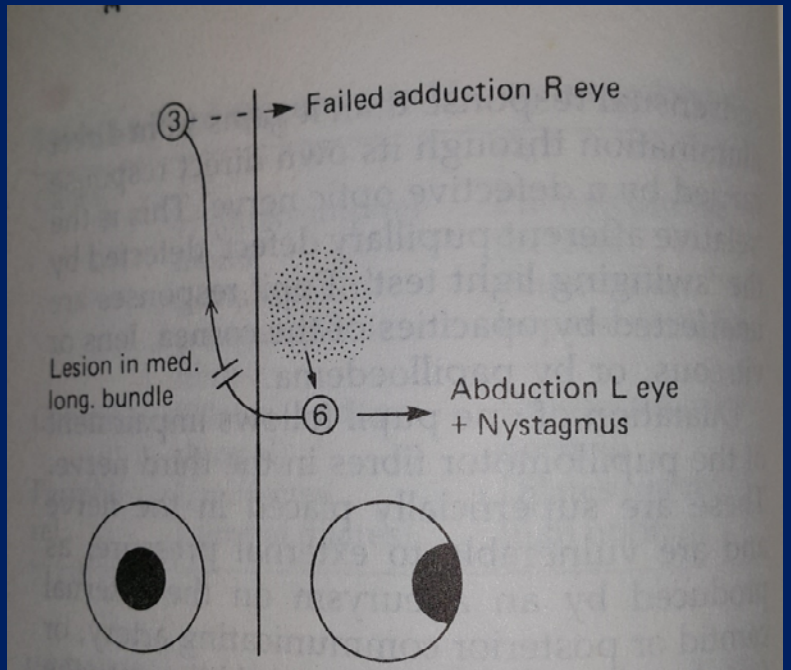


Figure 1.6 Interruption of the medial longitudinal bundle leads to nystagmus in the abducting eye (ataxic nystagmus) and defective adduction of the other eye (internuclear ophthalmoplegia), a common sign of brain-stem involvement in multiple sclerosis.

move in

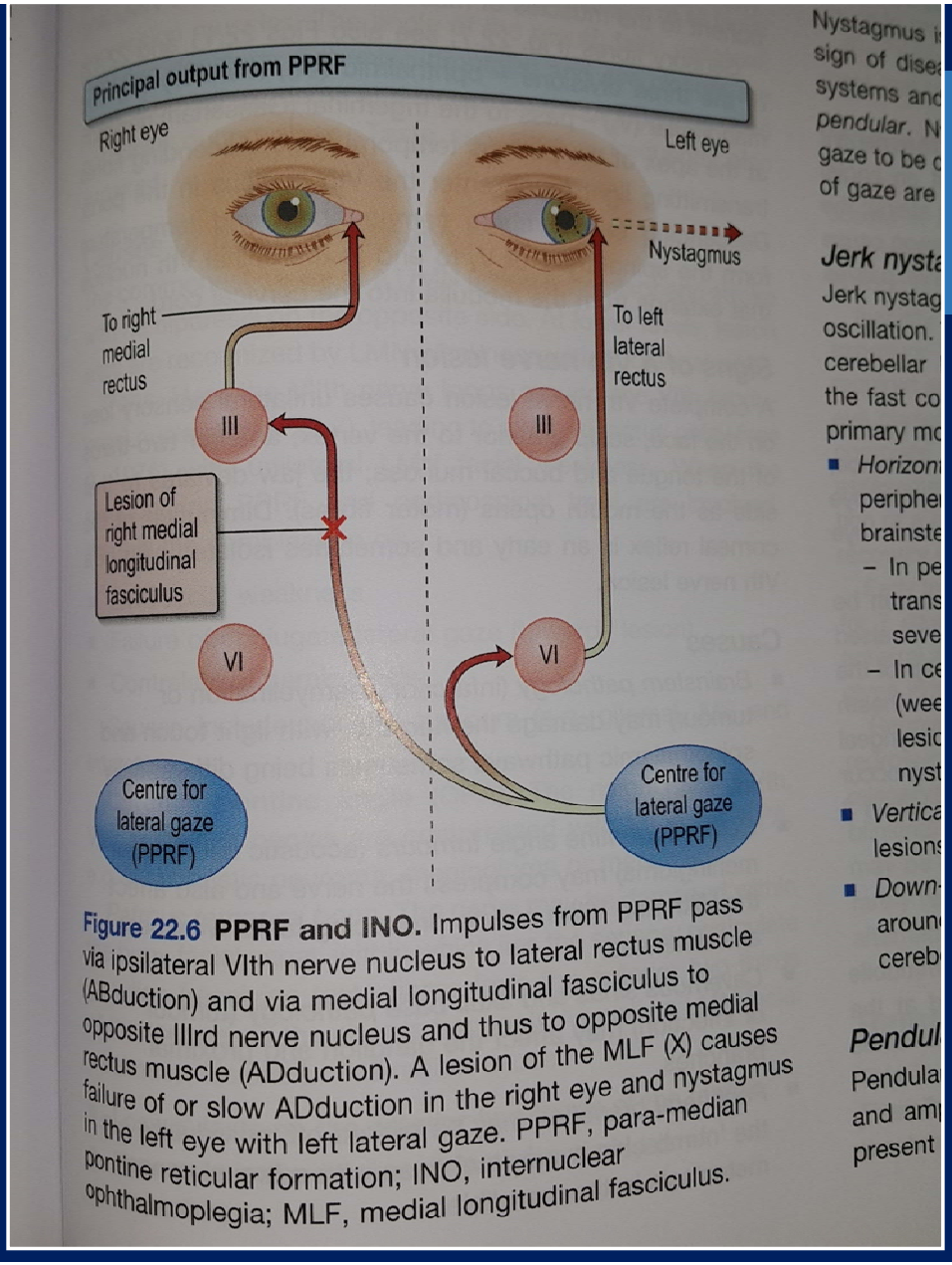


Figure 22.6 PPRF and INO. Impulses from PPRF pass via ipsilateral VIth nerve nucleus to lateral rectus muscle (ABduction) and via medial longitudinal fasciculus to opposite IIIrd nerve nucleus and thus to opposite medial rectus muscle (ADduction). A lesion of the MLF (X) causes failure of or slow ADduction in the right eye and nystagmus in the left eye with left lateral gaze. PPRF, para-median pontine reticular formation; INO, internuclear ophthalmoplegia; MLF, medial longitudinal fasciculus.

Impulses from PPRF pass via ipslateral Vth CN to lateral rectus muscle(Abduction)

A lesion of th MLF (X) causes failure of or slow ADDuction in the right eye and nystagmus in the left eye with left lateral gazes

Doll's eye movement

Passive head turning produces conjugate ocular deviation away from the direction of rotation (doll's head reflex)

Disappear in deep coma,in brainstem lesions and brain death

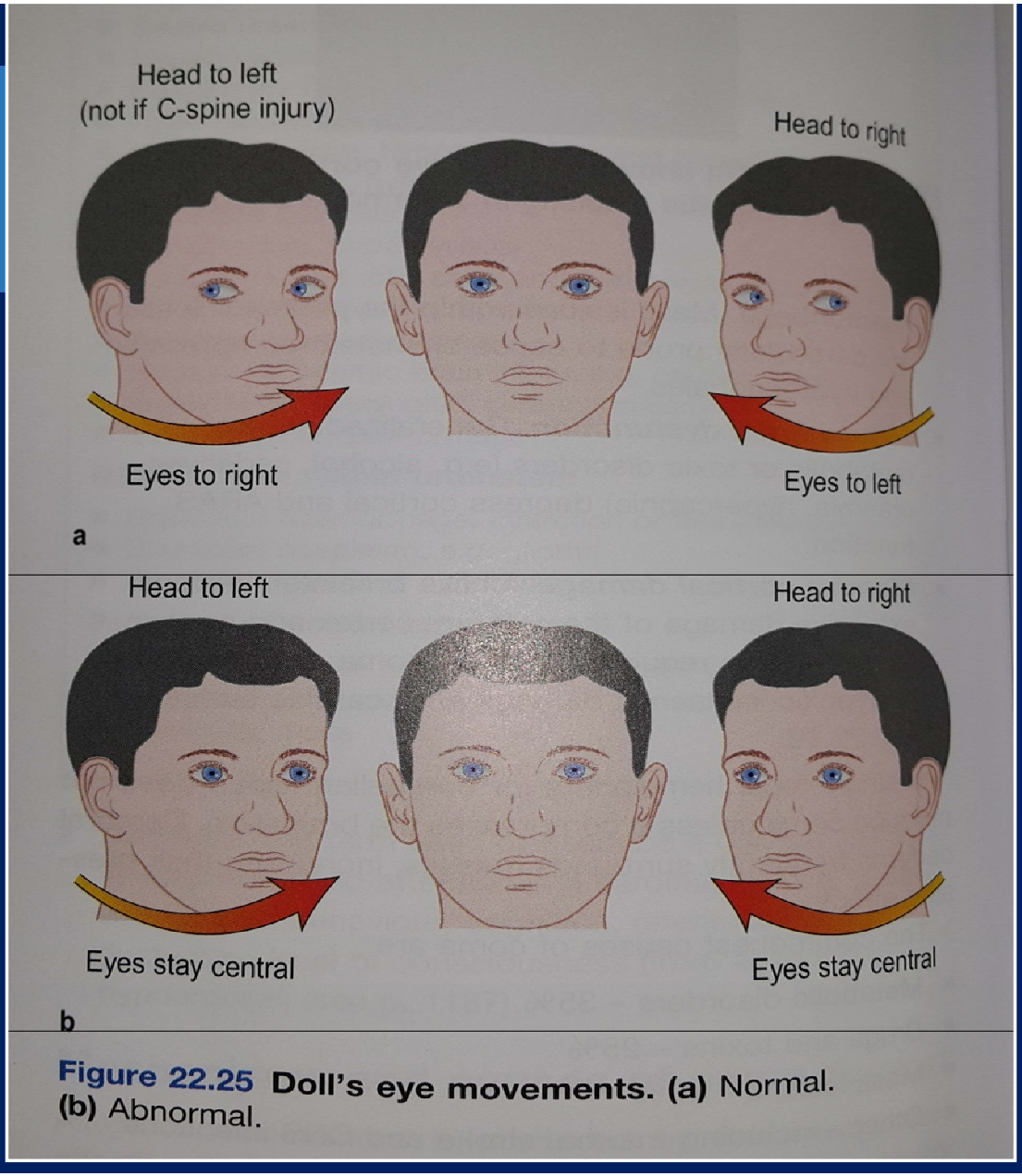


Figure 22.25 Doll's eye movements. (a) Normal. **(b)** Abnormal.

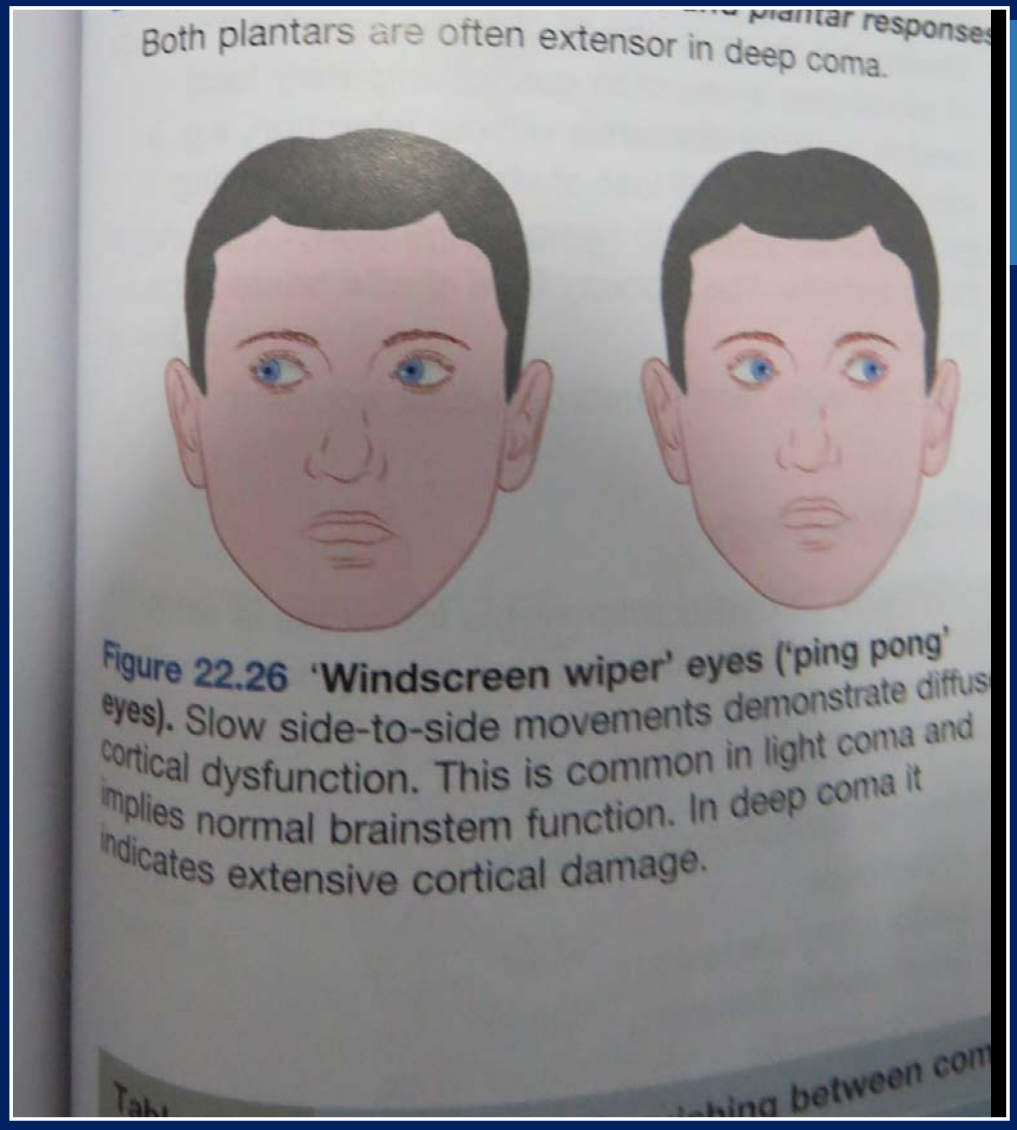

Both plantars are often extensor in deep coma.

Figure 22.26 'Windscreen wiper' eyes ('ping pong' eyes). Slow side-to-side movements demonstrate diffus cortical dysfunction. This is common in light coma and implies normal brainstem function. In deep coma it indicates extensive cortical damage.

Windscreen wiper eyes(ping pong eyes)
slow side to side movements
demonstrate diffuse cortical dysfunction
Common in light coma
It indicates extensive cortical damaged

Other brain stem reflexes

- Corneal reflex
- Gag/ cough reflex(via ET tube if incubated)
- Respiratory derive

Lateralizing sign

- Coma makes it difficult to recognize lateralizing sign
- Helpful points:
- Asymmetry of response to visual threat in a stuporose patient suggest hemianopia
- Asymmetry of face drooping or dribbling on one side blowing in and out of mouth when the paralysed cheek does not move

- Asymmetry of tone => unilateral flaccidity or spasticity may be the only sign of hemiparesis
- Asymmetry of decerebrate and decorticate posturing
- Asymmetrical response to painful stimuli
- Asymmetry of tendon reflexes and plantar responses- both planter are often extensor in deep coma

Coma look-alikes

- Psychogenic coma
- Loaked-in syndrome: complete paralysis except vertical eye movements/blinking in verntral pontine infarction
- Patients have a functioning cerebral cortex and are fully aware but unable to communicate except through eye movements
- Severe paralysis, eg myaesthenic crisis or severe Guillain-barre syndrome

Features distinguishing between coma and related states

Table 22.11 Features distinguishing between coma and related states

Feature	Vegetative	Minimally conscious	Locked-in	Coma	Brain death
Awareness	Absent	Present	Present	Absent	Absent
Sleep-wake cycles	Present	Present	Present	Absent	Absent
Noxious stimuli	Response	Avoidance	Response (eyes only)	Response ±	Response: nil
Glasgow Coma Scale	E4, M1–4, V1–2	E4, M1–5, V1–4	E4, M1, V1	E1–2, M1–4, V1–2	E1, M1–3, V1
Motor function	No purposeful movement	Consistent or inconsistent sounds/movements	Vertical eye movements/ blinking	No purposeful movement	None/only reflex spinal movement
Respiration	Preserved	Preserved	Preserved	Variable	Absent
EEG	Slow waves, usually	Data insufficient	Normal, usually	Slow waves, usually	Typically absent
Cerebral metabolism (PET)	Severely reduced	Data insufficient	Mildly reduced	Reduced	Severely reduced/ absent
Prognosis	Varies: usually continued VS or death	Varies	Varies: full recovery unlikely	Recovery, VS, or death	Already dead

EEG, electroencephalography; PET, positron emission tomography; VS, vegetative state.
EEG and PET are not required to confirm brain death.
Reproduced from Royal College of Physicians 2003. *The vegetative state: guidance on diagnosis and management. Report of a Working Party.* London: RCP, with permission.

Diagnosis and Investigation in coma

- Often the cause is evident- trauma, metabolic, overdose
- Lateralizing signs or brainstem pathology→Mass lesion or nfarct/ haemorrhage (careful : hypoglycemia may cause focal sign)
- No cause→ further Ix are essential

Blood and urine

- Drug screen
- Biochemistry (U&E, glucose, calcium,LFT)
- Metabolic and endocrine- hypothyroid,corticol
- Arterial blood gases- acidosis, CO_2 increase
- Other- cerebral malaria, porphyria

Brain imaging

- CT
- MRI useful if CT normal
- CT is quick and effective (mass and haemorrhage) (infarct may be missed in early stages and where brainstem only affected)

CSF examination

- LP- only after careful assessment
- If mass suspect- CI (CT choice)
- Role_ meningoencephalitis, other infections and SAH(CT normal)

EEG

- Metabolic coma
- Encephalitis
- Non convulsive status epilepticus

General management

- Comatose patients need careful nursing, meticulous attention to the airway, and frequent monitoring of vital functions.
- Longer-term essentials are-
- Skin care,eye care, fluids, feeding, sphinceters

Management of coma

- Skin care- turning to avoid pressure sores and pressure palsies
- Oral hygiene- mouthwashes, suction
- Eye care- prevention of corneal damage (lid taping, irrigation)
- Fluids- nasogastric or IV
- Feeding- via a fine bore nasogastric tube or via peg
- Sphincters- catheterization when essential (use penile urinary sheath if possible in men) rectal evacuation

prognosis

- Depend on the cause of coma and extent of brain damage sustained
- Metabolic and toxic – best Px
- Following hypoxic ischaemic brain injury only 11% make a good recovery and following stroke the prognosis is worse still with only 7% recovering

- Of those patients who do not recover consciousness, a substantial proportion will remain in a vegetative or minimally responsive state.

Vegetative state

- Is usually a consequence of extensive cortical damage. Brainstem function is intact so breathing is normal without the need for mechanical ventilation and the patient appears awake with eye opening and sleep-wake cycles.

  However there is no sign of awareness or response to environmental stimuli except reflex movements. Patients may remain in this state for years.

Permanent (PVS)

- There is no recovery after 12 months where trauma is the cause and after 6 months for all other causes
- Prolonged support of patients after this time presents a number of ethical issues – families may apply to the courts for withdrawal of feeding in PVS

Minimally conscious state (MCS)

- It describes patients with some limited awareness eg apparent, vague pain perception. A patient may emerge from VS into MCS
- Distinguishing VS from MCS requires careful specialist assessment over a long period. Functional brain imaging has recently been used for this purpose.

Brain stem death

- Brain death means the irreversible loss of the capacity for consciousness combined with the irreversible loss of the capacity to breathe.
- Both of these are essentially functions of the brainstem.
- Death if thought of in this way, can arise either from causes outside the brain(respiratory and cardiac arrest) or from causes within the cranial cavity.
- With the evident of mechanical ventilation it became possible to support such a dead patient temporarily although in all cases cardiovascular failure eventually supervenes and progresses to asystole

- Before deciding on a diagnosis of brainstem death, it is essential

that certain preconditions and exclusions are fulfilled

preconditions

- The patient must be in apneic coma (ie unresponsive and on a ventilator, with no spontaneous respiratory efforts)
- Irremediable structural brain damage due to a disorder that can cause brainstem death must have been diagnosed with certainty eg head injury, ICH

exclusion

- Possibility of apnoea due to → sedatives or neuromuscular block
- Hypothermia
- Significant metabolic or endocrine disturbances
- Profound abn plasma E, AB abn, glucose

Confirmation test for BS death

- Oculo-cephalic reflexes should be absent. In a comatose patient whose brainstem is intact the eyes will rotate relative to the orbit (doll's eye) in brainstem dead, the eyes remain stationary relative to the orbit
- Fixed and unresponsive to bright light (DLR, CLR)
- Corneal reflexes- absent

BS death (continued)

- No vestibule ocular reflexes on caloric testing
- No motor response within the cranial nerve territory to painful stimuli applied centrally or peripherally
- Spinal reflex movements may be present
- No gag or cough reflex in response to Ph, Lar, or tracheal stimaulation
- No spontaneous respiration

- Thank you