# examination of the comatose patient [created by Paul Young 28/11/07]

## limb movements & postural changes in coma

- choreoathetotic or ballistic movements suggest a basal ganglion lesion
- myoclonic movements indicate a metabolic disorder usually of post anoxic origin
- asterixis is usually seen with metabolic encephalopathies

decerebrate rigidity (extensor posturing)
- is characterised by stiff extension of limbs, internal rotation of the arms & plantar flexion of the ankles. These movements may be unilateral, bilateral, spontaneous or in response to a noxious stimulus
- while animal studies suggest that the lesion is usually in the midbrain, in humans such posturing may be due to:
(i) midbrain lesions
(ii) metabolic disorders including hypoglycaemia, hepatic coma and drug intoxication

## respiratory rate & pattern in coma

- as a general rule, at lighter levels of impaired consciousness tachypnoea predominates while respiratory depression increases with depth of coma
- precise localising value of respiratory rate & patterns is uncertain

| Abnormality | Significance |
|---|---|
| Bradypnoea | (i) Drug-induced coma<br>(ii) Hypothyroid coma |
| Tachypnoea | (i) Central neurogenic hyperventilation (midbrain lesions)<br>(ii) metabolic encephalopathy |
| Cheyne Stokes respiration (hyperpnoea alternating regularly with apnoea) | (i) deep cerebral lesions<br>(ii) metabolic encephalopathy |
| Apneustic breathing (an inspiratory pause) | (i) pontine lesions |
| Ataxic breathing (usuall progresses to agonal gasps and terminal apnoea) | (i) medullary lesions |

## body temperature in coma

- hypothermia is frequently observed with alcohol or barbiturate intoxication, sepsis, drowning, hypoglycaemia, myxoedema coma & exposure to cold
- severe hyperthermia may be seen in pontine haemorrhage, intracranial infections, heat stroke & anticholinergic drug toxicity

## distinguishing structural & metabolic encephalopathy

| Feature | Structural | Metabolic |
|---|---|---|
| Consciousness | Usually fixed level of depressed conscious or may deteriorate progressively | Milder alteration of conscious state with waxing and waning of altered sensorium |
| Fundoscopy | May be abnormal | Usually normal |
| Pupils | May be abnormal either in size or response to light | Usually preserved light response (except in certain overdoses) |
| Eye movements | May be affected | Usually preserved |
| Motor findings | Asymmetrical involvement | Abnormalities usually symmetrical |
| Involuntary movements | Not common | Asterixis, tremor & myoclonus frequently seen |

## eye signs in coma

ophthalmoscopy findings:
(i) papilloedema suggests the presence of intracranial hypertension but it is frequently absent when the lesion is acute
(ii) subhyaloid & vitreous haemorrhages are seen in patients with SAH

eye movements:
- horizontal eye movements to the contralateral side are initiated in the ipsilateral frontal lobe & closely coordinated in the contralateral pons. To facilitate conjugate eye movements, yoking of the 3rd, 4th & 6th cranial nerve nuclei is achieved by the medial longitudinal fasciculus (ie to look left the movement originates in the right frontal lobe & is coordinated by the left pons)
- vertical eye movements are under bilateral control of the cortex & upper midbrain
- full & conjugate eye movements in response to oculocephalic & oculovestibular stimuli demonstrates the functional integrity of a large segment of the brainstem
- upward rolling of the eyes after corneal stimulation (Bell's phenomenon) implies intact midbrain & pontine function)
- the presence of spontaneous roving eye movements excludes brainstem pathology as a cause of coma
- ocular bobbing, an intermittent downward jerking of eye movement is seen in pontine lesions due to loss of horizontal gaze and unopposed midbrain controlled vertical gaze activity
- in a paralytic frontal lobe pathology the eye will deviate towards the side of the lesion while in pontine pathologies, the eyes will deviate away from the lesion
- skew deviation (vertical separation of the ocular axes) occurs with pontine & cerebellar disorders

pupils:
- the presence of normal pupils (2-5mm, equal in size & demonstrating both direct & consensual light reflexes) confirms the integrity of the pupillary pathway (retina, optic nerve, optic chiasma & tracts, midbrain & 3rd cranial nerve nuclei & nerves)
- the size of the pupil is a balance between the opposing influences of both sympathetic & parasympathetic systems

| Abnormality | Cause | Neuroanatomical basis |
|---|---|---|
| Miosis (<2mm) | | |
| Unilateral | (i) Horner's syndrome<br>(ii) Local pathology | Sympathetic paralysis<br>Trauma to sympathetics |
| Bilateral | (i) Pontine lesions<br>(ii) Thalamic haemorrhage<br>(iii) metabolic encephalopathy<br>(iv) drug ingestion<br>- organophosphates<br>- narcotics<br>- barbiturates | } sympathetic paralysis<br><br>cholinesterase inhibition<br>central effect |
| Mydriasis (>5mm) | | |
| Unilateral fixed | (i) midbrain lesion<br>(ii) uncal herniation | 3rd nerve damage<br>stretch of 3rd nerve against the petroclinoid ligament |
| Bilateral fixed | (i) massive midbrain haemorrhage<br>(ii) hypoxic brain injury<br>(iii) drug ingestion<br>- atropine<br>- tricyclics<br><br>- sympathomimetics | Bilateral 3rd nerve damage<br>Mesencephalic damage<br><br>Paralysis of parasympathetics<br>Prevent local catecholamine uptake by nerve endings<br>Stimulation of sympathetics |