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 (ii) Hypothyroid coma
Tachypnoea	(i) Central neurogenic hyperventilation (midbrain lesions) (ii) metabolic encephalopathy
Cheyne Stokes respiration (hyperpnoea alternating regularly with apnoea)	(i) deep cerebral lesions (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 (ii) Local pathology	Sympathetic paralysis Trauma to sympathetics
Bilateral	(i) Pontine lesions (ii) Thalamic haemorrhage (iii) metabolic encephalopathy (iv) drug ingestion - organophosphates - narcotics - barbiturates	} sympathetic paralysis cholinesterase inhibition central effect
Mydriasis (>5mm)		
Unilateral fixed	(i) midbrain lesion (ii) uncal herniation	3rd nerve damage stretch of 3rd nerve against the petroclinoid ligament
Bilateral fixed	(i) massive midbrain haemorrhage (ii) hypoxic brain injury (iii) drug ingestion - atropine - tricyclics - sympathomimetics	Bilateral 3rd nerve damage Mesencephalic damage Paralysis of parasympathetics Prevent local catecholamine uptake by nerve endings Stimulation of sympathetics