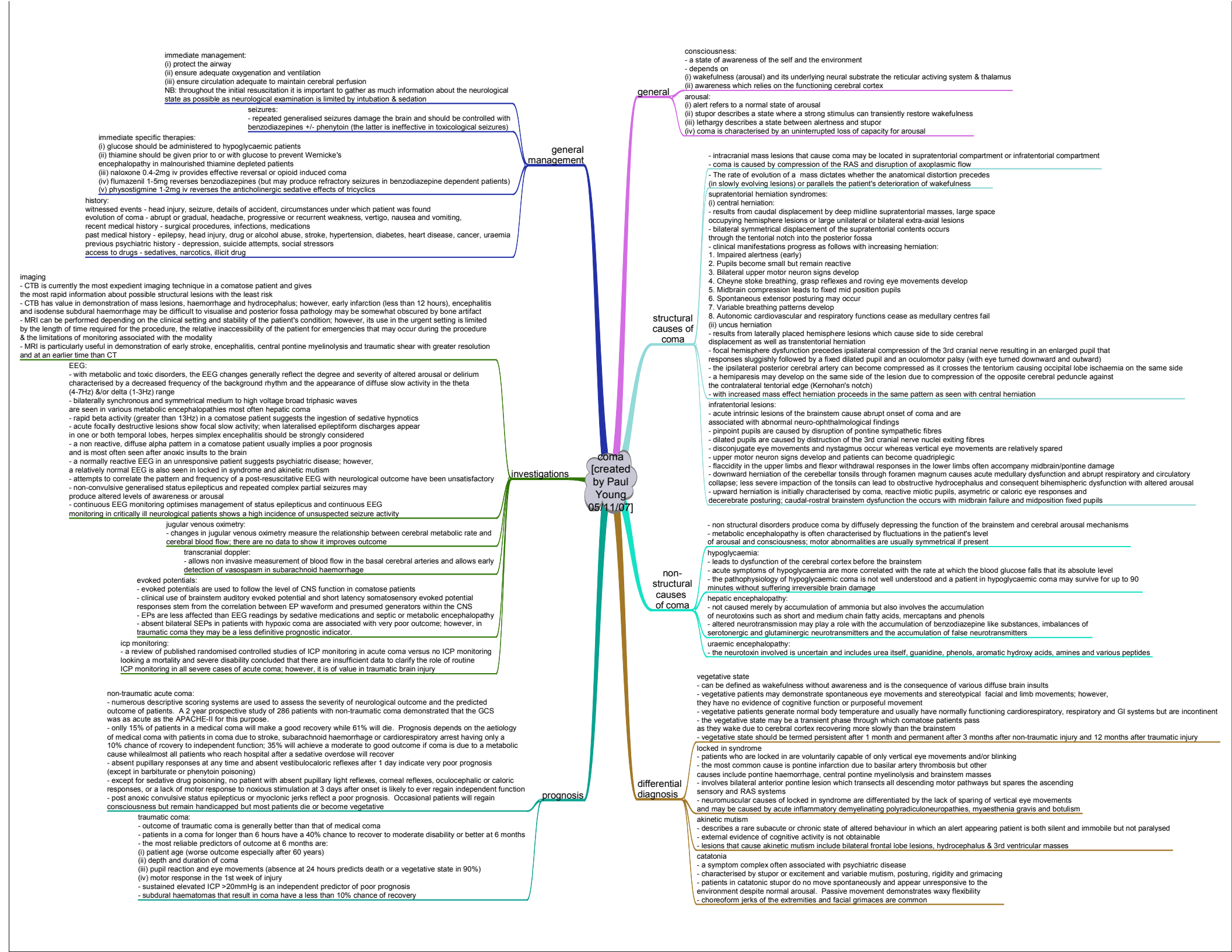
coma [created by Paul Young 05/11/07]
general
consciousness:
- a state of awareness of the self and the environment
- depends on
(i) wakefulness (arousal) and its underlying neural substrate the reticular activing system & thalamus
(ii) awareness which relies on the functioning cerebral cortex
arousal:
(i) alert refers to a normal state of arousal
(ii) stupor describes a state where a strong stimulus can transiently restore wakefulness
(iii) lethargy describes a state between alertness and stupor
(iv) coma is characterised by an uninterrupted loss of capacity for arousal
general management
immediate management:
(i) protect the airway
(ii) ensure adequate oxygenation and ventilation
(iii) ensure circulation adequate to maintain cerebral perfusion
NB: throughout the initial resuscitation it is important to gather as much information about the neurological state as possible as neurological examination is limited by intubation & sedation
seizures:
- repeated generalised seizures damage the brain and should be controlled with benzodiazepines +/- phenytoin (the latter is ineffective in toxicological seizures)
immediate specific therapies:
(i) glucose should be administered to hypoglycaemic patients
(ii) thiamine should be given prior to or with glucose to prevent Wernicke's encephalopathy in malnourished thiamine depleted patients
(iii) naloxone 0.4-2mg iv provides effective reversal or opioid induced coma
(iv) flumazenil 1-5mg reverses benzodiazepines (but may produce refractory seizures in benzodiazepine dependent patients)
(v) physostigmine 1-2mg iv reverses the anticholinergic sedative effects of tricyclics
history:
witnessed events - head injury, seizure, details of accident, circumstances under which patient was found
evolution of coma - abrupt or gradual, headache, progressive or recurrent weakness, vertigo, nausea and vomiting,
recent medical history - surgical procedures, infections, medications
past medical history - epilepsy, head injury, drug or alcohol abuse, stroke, hypertension, diabetes, heart disease, cancer, uraemia
previous psychiatric history - depression, suicide attempts, social stressors
access to drugs - sedatives, narcotics, illicit drug
structural causes of coma
- intracranial mass lesions that cause coma may be located in supratentorial compartment or infratentorial compartment
- coma is caused by compression of the RAS and disruption of axoplasmic flow
- The rate of evolution of a mass dictates whether the anatomical distortion precedes (in slowly evolving lesions) or parallels the patient's deterioration of wakefulness
supratentorial herniation syndromes:
(i) central herniation:
- results from caudal displacement by deep midline supratentorial masses, large space occupying hemisphere lesions or large unilateral or bilateral extra-axial lesions
- bilateral symmetrical displacement of the supratentorial contents occurs through the tentorial notch into the posterior fossa
- clinical manifestations progress as follows with increasing herniation:
1. Impaired alertness (early)
2. Pupils become small but remain reactive
3. Bilateral upper motor neuron signs develop
4. Cheyne stoke breathing, grasp reflexes and roving eye movements develop
5. Midbrain compression leads to fixed mid position pupils
6. Spontaneous extensor posturing may occur
7. Variable breathing patterns develop
8. Autonomic cardiovascular and respiratory functions cease as medullary centres fail
(ii) uncus herniation
- results from laterally placed hemisphere lesions which cause side to side cerebral displacement as well as transtentorial herniation
- focal hemisphere dysfunction precedes ipsilateral compression of the 3rd cranial nerve resulting in an enlarged pupil that responses sluggishly followed by a fixed dilated pupil and an oculomotor palsy (with eye turned downward and outward)
- the ipsilateral posterior cerebral artery can become compressed as it crosses the tentorium causing occipital lobe ischaemia on the same side
- a hemiparesis may develop on the same side of the lesion due to compression of the opposite cerebral peduncle against the contralateral tentorial edge (Kernohan's notch)
- with increased mass effect herniation proceeds in the same pattern as seen with central herniation
infratentorial lesions:
- acute intrinsic lesions of the brainstem cause abrupt onset of coma and are associated with abnormal neuro-ophthalmological findings
- pinpoint pupils are caused by disruption of pontine sympathetic fibres
- dilated pupils are caused by distruction of the 3rd cranial nerve nuclei exiting fibres
- disconjugate eye movements and nystagmus occur whereas vertical eye movements are relatively spared
- upper motor neuron signs develop and patients can become quadriplegic
- flaccidity in the upper limbs and flexor withdrawal responses in the lower limbs often accompany midbrain/pontine damage
- downward herniation of the cerebellar tonsils through foramen magnum causes acute medullary dysfunction and abrupt respiratory and circulatory collapse; less severe impaction of the tonsils can lead to obstructive hydrocephalus and consequent bihemispheric dysfunction with altered arousal
- upward herniation is initially characterised by coma, reactive miotic pupils, asymetric or caloric eye responses and decerebrate posturing; caudal-rostral brainstem dysfunction the occurs with midbrain failure and midposition fixed pupils
non-structural causes of coma
- non structural disorders produce coma by diffusely depressing the function of the brainstem and cerebral arousal mechanisms
- metabolic encephalopathy is often characterised by fluctuations in the patient's level of arousal and consciousness; motor abnormalities are usually symmetrical if present
hypoglycaemia:
- leads to dysfunction of the cerebral cortex before the brainstem
- acute symptoms of hypoglycaemia are more correlated with the rate at which the blood glucose falls that its absolute level
- the pathophysiology of hypoglycaemic coma is not well understood and a patient in hypoglycaemic coma may survive for up to 90 minutes without suffering irreversible brain damage
hepatic encephalopathy:
- not caused merely by accumulation of ammonia but also involves the accumulation of neurotoxins such as short and medium chain fatty acids, mercaptans and phenols
- altered neurotransmission may play a role with the accumulation of benzodiazepine like substances, imbalances of serotonergic and glutaminergic neurotransmitters and the accumulation of false neurotransmitters
uraemic encephalopathy:
- the neurotoxin involved is uncertain and includes urea itself, guanidine, phenols, aromatic hydroxy acids, amines and various peptides
differential diagnosis
vegetative state
- can be defined as wakefulness without awareness and is the consequence of various diffuse brain insults
- vegetative patients may demonstrate spontaneous eye movements and stereotypical facial and limb movements; however, they have no evidence of cognitive function or purposeful movement
- vegetative patients generate normal body temperature and usually have normally functioning cardiorespiratory, respiratory and GI systems but are incontinent
- the vegetative state may be a transient phase through which comatose patients pass as they wake due to cerebral cortex recovering more slowly than the brainstem
- vegetative state should be termed persistent after 1 month and permanent after 3 months after non-traumatic injury and 12 months after traumatic injury
locked in syndrome
- patients who are locked in are voluntarily capable of only vertical eye movements and/or blinking
- the most common cause is pontine infarction due to basilar artery thrombosis but other causes include pontine haemorrhage, central pontine myelinolysis and brainstem masses
- involves bilateral anterior pontine lesion which transects all descending motor pathways but spares the ascending sensory and RAS systems
- neuromuscular causes of locked in syndrome are differentiated by the lack of sparing of vertical eye movements and may be caused by acute inflammatory demyelinating polyradiculoneuropathies, myaesthenia gravis and botulism
akinetic mutism
- describes a rare subacute or chronic state of altered behaviour in which an alert appearing patient is both silent and immobile but not paralysed
- external evidence of cognitive activity is not obtainable
- lesions that cause akinetic mutism include bilateral frontal lobe lesions, hydrocephalus & 3rd ventricular masses
catatonia
- a symptom complex often associated with psychiatric disease
- characterised by stupor or excitement and variable mutism, posturing, rigidity and grimacing
- patients in catatonic stupor do no move spontaneously and appear unresponsive to the environment despite normal arousal. Passive movement demonstrates waxy flexibility
- choreoform jerks of the extremities and facial grimaces are common
investigations
imaging
- CTB is currently the most expedient imaging technique in a comatose patient and gives the most rapid information about possible structural lesions with the least risk
- CTB has value in demonstration of mass lesions, haemorrhage and hydrocephalus; however, early infarction (less than 12 hours), encephalitis and isodense subdural haemorrhage may be difficult to visualise and posterior fossa pathology may be somewhat obscured by bone artifact
- MRI can be performed depending on the clinical setting and stability of the patient's condition; however, its use in the urgent setting is limited by the length of time required for the procedure, the relative inaccessibility of the patient for emergencies that may occur during the procedure & the limitations of monitoring associated with the modality
- MRI is particularly useful in demonstration of early stroke, encephalitis, central pontine myelinolysis and traumatic shear with greater resolution and at an earlier time than CT
EEG:
- with metabolic and toxic disorders, the EEG changes generally reflect the degree and severity of altered arousal or delirium characterised by a decreased frequency of the background rhythm and the appearance of diffuse slow activity in the theta (4-7Hz) &/or delta (1-3Hz) range
- bilaterally synchronous and symmetrical medium to high voltage broad triphasic waves are seen in various metabolic encephalopathies most often hepatic coma
- rapid beta activity (greater than 13Hz) in a comatose patient suggests the ingestion of sedative hypnotics
- acute focally destructive lesions show focal slow activity; when lateralised epileptiform discharges appear in one or both temporal lobes, herpes simplex encephalitis should be strongly considered
- a non reactive, diffuse alpha pattern in a comatose patient usually implies a poor prognosis and is most often seen after anoxic insults to the brain
- a normally reactive EEG in an unresponsive patient suggests psychiatric disease; however, a relatively normal EEG is also seen in locked in syndrome and akinetic mutism
- attempts to correlate the pattern and frequency of a post-resuscitative EEG with neurological outcome have been unsatisfactory
- non-convulsive generalised status epilepticus and repeated complex partial seizures may produce altered levels of awareness or arousal
- continuous EEG monitoring optimises management of status epilepticus and continuous EEG monitoring in critically ill neurological patients shows a high incidence of unsuspected seizure activity
jugular venous oximetry:
- changes in jugular venous oximetry measure the relationship between cerebral metabolic rate and cerebral blood flow; there are no data to show it improves outcome
transcranial doppler:
- allows non invasive measurement of blood flow in the basal cerebral arteries and allows early detection of vasospasm in subarachnoid haemorrhage
evoked potentials:
- evoked potentials are used to follow the level of CNS function in comatose patients
- clinical use of brainstem auditory evoked potential and short latency somatosensory evoked potential responses stem from the correlation between EP waveform and presumed generators within the CNS
- EPs are less affected than EEG readings by sedative medications and septic or metabolic encephalopathy
- absent bilateral SEPs in patients with hypoxic coma are associated with very poor outcome; however, in traumatic coma they may be a less definitive prognostic indicator.
icp monitoring:
- a review of published randomised controlled studies of ICP monitoring in acute coma versus no ICP monitoring looking a mortality and severe disability concluded that there are insufficient data to clarify the role of routine ICP monitoring in all severe cases of acute coma; however, it is of value in traumatic brain injury
prognosis
non-traumatic acute coma:
- numerous descriptive scoring systems are used to assess the severity of neurological outcome and the predicted outcome of patients. A 2 year prospective study of 286 patients with non-traumatic coma demonstrated that the GCS was as acute as the APACHE-II for this purpose.
- onlly 15% of patients in a medical coma will make a good recovery while 61% will die. Prognosis depends on the aetiology of medical coma with patients in coma due to stroke, subarachnoid haemorrhage or cardiorespiratory arrest having only a 10% chance of rcovery to independent function; 35% will achieve a moderate to good outcome if coma is due to a metabolic cause whilealmost all patients who reach hospital after a sedative overdose will recover
- absent pupillary responses at any time and absent vestibulocaloric reflexes after 1 day indicate very poor prognosis (except in barbiturate or phenytoin poisoning)
- except for sedative drug poisoning, no patient with absent pupillary light reflexes, corneal reflexes, oculocephalic or caloric responses, or a lack of motor response to noxious stimulation at 3 days after onset is likely to ever regain independent function
- post anoxic convulsive status epilepticus or myoclonic jerks reflect a poor prognosis. Occasional patients will regain consciousness but remain handicapped but most patients die or become vegetative
traumatic coma:
- outcome of traumatic coma is generally better than that of medical coma
- patients in a coma for longer than 6 hours have a 40% chance to recover to moderate disability or better at 6 months
- the most reliable predictors of outcome at 6 months are:
(i) patient age (worse outcome especially after 60 years)
(ii) depth and duration of coma
(iii) pupil reaction and eye movements (absence at 24 hours predicts death or a vegetative state in 90%)
(iv) motor response in the 1st week of injury
- sustained elevated ICP >20mmHg is an independent predictor of poor prognosis
- subdural haematomas that result in coma have a less than 10% chance of recovery