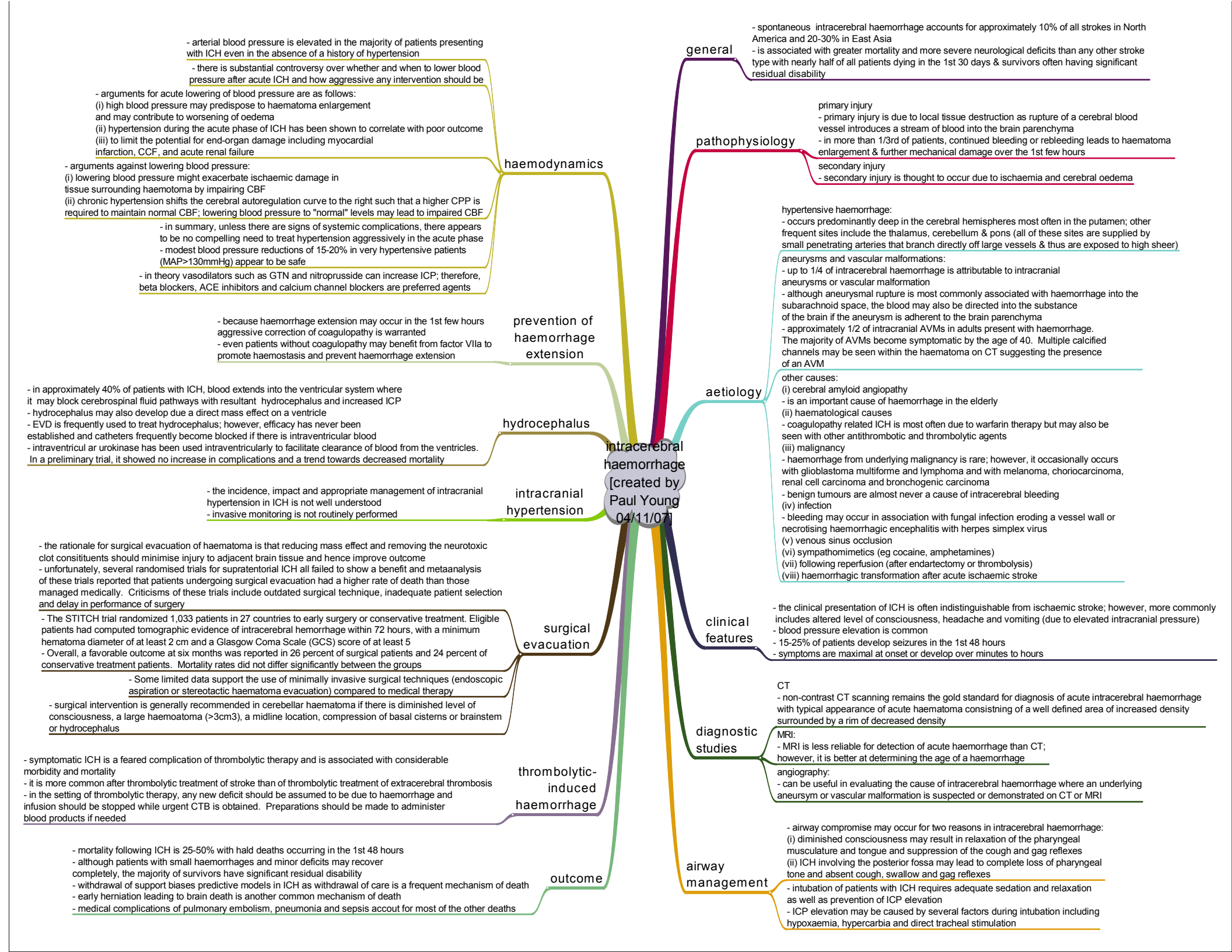
intracerebral haemorrhage [created by Paul Young 04/11/07]
general
- spontaneous intracerebral haemorrhage accounts for approximately 10% of all strokes in North America and 20-30% in East Asia
- is associated with greater mortality and more severe neurological deficits than any other stroke type with nearly half of all patients dying in the 1st 30 days & survivors often having significant residual disability
pathophysiology
primary injury
- primary injury is due to local tissue destruction as rupture of a cerebral blood vessel introduces a stream of blood into the brain parenchyma
- in more than 1/3rd of patients, continued bleeding or rebleeding leads to haematoma enlargement & further mechanical damage over the 1st few hours
secondary injury
- secondary injury is thought to occur due to ischaemia and cerebral oedema
aetiology
hypertensive haemorrhage:
- occurs predominantly deep in the cerebral hemispheres most often in the putamen; other frequent sites include the thalamus, cerebellum & pons (all of these sites are supplied by small penetrating arteries that branch directly off large vessels & thus are exposed to high sheer)
aneurysms and vascular malformations:
- up to 1/4 of intracerebral haemorrhage is attributable to intracranial aneurysms or vascular malformation
- although aneurysmal rupture is most commonly associated with haemorrhage into the subarachnoid space, the blood may also be directed into the substance of the brain if the aneurysm is adherent to the brain parenchyma
- approximately 1/2 of intracranial AVMs in adults present with haemorrhage. The majority of AVMs become symptomatic by the age of 40. Multiple calcified channels may be seen within the haematoma on CT suggesting the presence of an AVM
other causes:
(i) cerebral amyloid angiopathy
- is an important cause of haemorrhage in the elderly
(ii) haematological causes
- coagulopathy related ICH is most often due to warfarin therapy but may also be seen with other antithrombotic and thrombolytic agents
(iii) malignancy
- haemorrhage from underlying malignancy is rare; however, it occasionally occurs with glioblastoma multiforme and lymphoma and with melanoma, choriocarcinoma, renal cell carcinoma and bronchogenic carcinoma
- benign tumours are almost never a cause of intracerebral bleeding
(iv) infection
- bleeding may occur in association with fungal infection eroding a vessel wall or necrotising haemorrhagic encephalitis with herpes simplex virus
(v) venous sinus occlusion
(vi) sympathomimetics (eg cocaine, amphetamines)
(vii) following reperfusion (after endartectomy or thrombolysis)
(viii) haemorrhagic transformation after acute ischaemic stroke
clinical features
- the clinical presentation of ICH is often indistinguishable from ischaemic stroke; however, more commonly includes altered level of consciousness, headache and vomiting (due to elevated intracranial pressure)
- blood pressure elevation is common
- 15-25% of patients develop seizures in the 1st 48 hours
- symptoms are maximal at onset or develop over minutes to hours
diagnostic studies
CT
- non-contrast CT scanning remains the gold standard for diagnosis of acute intracerebral haemorrhage with typical appearance of acute haematoma consistning of a well defined area of increased density surrounded by a rim of decreased density
MRI:
- MRI is less reliable for detection of acute haemorrhage than CT; however, it is better at determining the age of a haemorrhage
angiography:
- can be useful in evaluating the cause of intracerebral haemorrhage where an underlying aneurysm or vascular malformation is suspected or demonstrated on CT or MRI
airway management
- airway compromise may occur for two reasons in intracerebral haemorrhage:
(i) diminished consciousness may result in relaxation of the pharyngeal musculature and tongue and suppression of the cough and gag reflexes
(ii) ICH involving the posterior fossa may lead to complete loss of pharyngeal tone and absent cough, swallow and gag reflexes
- intubation of patients with ICH requires adequate sedation and relaxation as well as prevention of ICP elevation
- ICP elevation may be caused by several factors during intubation including hypoxaemia, hypercarbia and direct tracheal stimulation
outcome
- mortality following ICH is 25-50% with hald deaths occurring in the 1st 48 hours
- although patients with small haemorrhages and minor deficits may recover completely, the majority of survivors have significant residual disability
- withdrawal of support biases predictive models in ICH as withdrawal of care is a frequent mechanism of death
- early herniation leading to brain death is another common mechanism of death
- medical complications of pulmonary embolism, pneumonia and sepsis accout for most of the other deaths
thrombolytic-induced haemorrhage
- symptomatic ICH is a feared complication of thrombolytic therapy and is associated with considerable morbidity and mortality
- it is more common after thrombolytic treatment of stroke than of thrombolytic treatment of extracerebral thrombosis
- in the setting of thrombolytic therapy, any new deficit should be assumed to be due to haemorrhage and infusion should be stopped while urgent CTB is obtained. Preparations should be made to administer blood products if needed
surgical evacuation
- the rationale for surgical evacuation of haematoma is that reducing mass effect and removing the neurotoxic clot consitituents should minimise injury to adjacent brain tissue and hence improve outcome
- unfortunately, several randomised trials for supratentorial ICH all failed to show a benefit and metaanalysis of these trials reported that patients undergoing surgical evacuation had a higher rate of death than those managed medically. Criticisms of these trials include outdated surgical technique, inadequate patient selection and delay in performance of surgery
- The STITCH trial randomized 1,033 patients in 27 countries to early surgery or conservative treatment. Eligible patients had computed tomographic evidence of intracerebral hemorrhage within 72 hours, with a minimum hematoma diameter of at least 2 cm and a Glasgow Coma Scale (GCS) score of at least 5
- Overall, a favorable outcome at six months was reported in 26 percent of surgical patients and 24 percent of conservative treatment patients. Mortality rates did not differ significantly between the groups
- Some limited data support the use of minimally invasive surgical techniques (endoscopic aspiration or stereotactic haematoma evacuation) compared to medical therapy
- surgical intervention is generally recommended in cerebellar haematoma if there is diminished level of consciousness, a large haemoatoma (>3cm3), a midline location, compression of basal cisterns or brainstem or hydrocephalus
intracranial hypertension
- the incidence, impact and appropriate management of intracranial hypertension in ICH is not well understood
- invasive monitoring is not routinely performed
hydrocephalus
- in approximately 40% of patients with ICH, blood extends into the ventricular system where it may block cerebrospinal fluid pathways with resultant hydrocephalus and increased ICP
- hydrocephalus may also develop due a direct mass effect on a ventricle
- EVD is frequently used to treat hydrocephalus; however, efficacy has never been established and catheters frequently become blocked if there is intraventricular blood
- intraventricul ar urokinase has been used intraventricularly to facilitate clearance of blood from the ventricles. In a preliminary trial, it showed no increase in complications and a trend towards decreased mortality
prevention of haemorrhage extension
- because haemorrhage extension may occur in the 1st few hours aggressive correction of coagulopathy is warranted
- even patients without coagulopathy may benefit from factor VIIa to promote haemostasis and prevent haemorrhage extension
haemodynamics
- arterial blood pressure is elevated in the majority of patients presenting with ICH even in the absence of a history of hypertension
- there is substantial controversy over whether and when to lower blood pressure after acute ICH and how aggressive any intervention should be
- arguments for acute lowering of blood pressure are as follows:
(i) high blood pressure may predispose to haematoma enlargement and may contribute to worsening of oedema
(ii) hypertension during the acute phase of ICH has been shown to correlate with poor outcome
(iii) to limit the potential for end-organ damage including myocardial infarction, CCF, and acute renal failure
- arguments against lowering blood pressure:
(i) lowering blood pressure might exacerbate ischaemic damage in tissue surrounding haemotoma by impairing CBF
(ii) chronic hypertension shifts the cerebral autoregulation curve to the right such that a higher CPP is required to maintain normal CBF; lowering blood pressure to "normal" levels may lead to impaired CBF
- in summary, unless there are signs of systemic complications, there appears to be no compelling need to treat hypertension aggressively in the acute phase
- modest blood pressure reductions of 15-20% in very hypertensive patients (MAP>130mmHg) appear to be safe
- in theory vasodilators such as GTN and nitroprusside can increase ICP; therefore, beta blockers, ACE inhibitors and calcium channel blockers are preferred agents