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 edema
  (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 include:
  (i) Lowering blood pressure might exacerbate ischemic damage in tissue surrounding hematoma 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
- Because hemorrhage extension may occur in the first few hours, aggressive correction of coagulopathy is warranted
- Even patients without coagulopathy may benefit from factor VIIa to promote hematostasis and prevent hemorrhage extension

- The rationale for surgical evacuation of hematoma is that reducing mass effect and removing the neurotoxic clot constituents should minimize injury to adjacent brain tissue and hence improve outcome
- Unfortunately, several randomized 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 Criticisms of these trials include outdated surgical technique, inadequate patient selection and delay in surgery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% of surgical patients and 24% of conservative treatment patients. Mortality rates did not differ significantly between the groups
- In some limited data, support the use of minimally invasive surgical techniques (endoscopic aspiration or stereotactic hematoma evacuation) compared to medical therapy
- Surgical intervention is generally recommended in cerebellar hematoma if there is diminished level of consciousness, a large hematoma (>3cm3), a midline location, compression of basal cisterns or brainstem or hydrocephalus
- 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 hemorrhage and infusion should be stopped while urgent CTB is obtained. Preparations should be made to administer blood products if needed

- Spontaneous intracerebral hemorrhage accounts for approximately 10% of all strokes in North America and 20-30% in East Asia
- It is associated with greater mortality and more severe neurological deficits than any other stroke type with nearly half of all patients dying in the first 30 days & survivors often having significant residual disability

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 hematoma enlargement & further mechanical damage over the first few hours

Pathophysiology:
- Secondary injury is thought to occur due to ischemia and cerebral edema
- Hypertensive hemorrhage:
  - 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 shear)

Aneurysms and vascular malformations:
- Up to 1/4 of intracerebral hemorrhage 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 intracerebral AVMs in adults present with hemorrhage
- The majority of AVMs become symptomatic by the age of 40. Multiple calcified channels may be seen within the hematoma on CT suggesting the presence of an AVM

Other causes:
- Cerebral amyloid angiopathy
  - Is an important cause of hemorrhage in the elderly
- Haematological causes
  - Coagulopathy related ICH is most often due to warfarin therapy but may also be seen with other antithrombotic and thrombolytic agents
- Malignancy
  - Haemorrhage from underlying malignancy is rare; however, it occasionally occurs with glioastoma multiforme and lymphoma and with melanoma, chorocarcinoma, renal cell carcinoma and bronchogenic carcinoma
- Benign tumours are almost never a cause of intracerebral bleeding
- Infection
  - Bleeding may occur in association with fungal infection eroding a vessel wall or necrotising haemorrhagic encephalitis with herpes simplex virus
- Venous sinus occlusion
- Sympathomimetics (eg cocaine, amphetamines)
- Following reperfusion (after endarterectomy or thrombolysis)
- Haemorrhagic transformation after acute ischaemic stroke

Surgical evacuation
- Non-contrast CT scanning remains the gold standard for diagnosis of acute intracerebral haemorrhage with typical appearance of acute haematoma consisting of a well defined area of increased density surrounded by a rim of decreased density
- CT angiography is less reliable for detection of acute haemorrhage than CT; however, it is better at determining the age of a haemorrhage

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 first 48 hours
- Symptoms are maximal at onset or develop over minutes to hours
- 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

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