QUESTION 48 Neuro
A 73-year-old man presents with a two-day history of increasing headache and mild left-sided weakness. On examination, he is alert, with normal language and higher mental functions. He has a mild left facial weakness with a mild left hemiparesis. Sensory examination is normal. There is mild left sided hyperreflexia with an upgoing left plantar response. His blood pressure is 170/80 mmHg. His non contrast cranial computed tomography (CT) scan is shown here.

The most appropriate management is:
A. craniotomy and removal of lesion.
B. dexamethasone.
C. glyceryl trinitrate infusion.
D. mannitol infusion.
E. observation.

Intracerebral haemorrhage

**Causes**
1. Hypertension
2. Amyloid angiopathy
3. Ruptured saccular aneurysm
4. Vascular malformation
5. Hemorrhagic infarction (venous sinus thrombosis)
6. Septic embolism (bacterial endocarditis)
7. Brain tumour
8. Bleeding disorders, anticoagulants, thrombolytic therapy

**Pathogenesis**
Common sites:
Territory of penetrator arteries that branch off major intracerebral arteries, often at 90 deg angles with parent vessel
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Vessels supply
pon and midbrain (penetrators of basilar artery),
thalamus (thalamostriata penetrators off P1 and P@ segments of posterior cerebral arteries) putamen and
caudate (lenticulostriate penetrators off the M1 segment of middle cerebral artery)

**Mechanism of brain injury**
1. Primary direct mechanical injury to brain parenchyma by expanding clot
2. Increased intracranial pressure
3. Herniation secondary to mass effect

**Secondary brain injury**
Decreased blood flow local neuronal ischemia further cytotoxic edema toxic release of inflammatory mediators

Blood brain barrier breakdown and dysregulation of hemostasis via inflammatory cascade activation and MMP overexpression

**Haemorrhage enlargement**
- Haemorrhage enlarges in the 1st 6 hrs after presentation
- Frequency of significant hemorrhage enlargement (>33% vol increase) over 24 hr is 38% of patients
- Enlargement of hematoma associated with neurologic deterioration

<table>
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<tr>
<th><strong>Aims of treatment:</strong></th>
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<tr>
<td>Minimising secondary brain ischemia</td>
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<tr>
<td>Preventing hematoma enlargement</td>
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**Epidemiology**
- Framingham study: 8% of all strokes, 25% of strokes in Japan
- Incidence of ICH increases with age, doubling every 10 years after age 35
- Rate of occurrence is highest in Asian, intermediate in blacks and lowest in whites

**Risk factors**
- Hypertension
  - Doubled risk of ICH
- Smoking
- Hypcholesterolemia
  - Treatment with statins does not appear to increase risk of primary ICH
- Cerebral amyloid angiopathy

**Clinical presentation**
Neurologic symptoms do not begin abruptly and are not maximal at onset (compared to brain embolism and subarachnoid hemorrhage)

**Sx:**
- headache, vomiting and altered level of consciousness
- seizures occur in 7 – 9 % of ICH, more common in lobar hemorrhages (affecting cortical tissue)
- complain of stiff neck and meningismus if there is intraventricular blood
- stupor or coma is ominous unless pt had thalamic hemorrhage (involvement of reticular activating system), recover after blood is reabsorbed
- ECG changes: prolonged QT interval, depressed ST segment, flat or inverted T waves, U waves and tall peaked T waves
### Site of hemorrhage

<table>
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<tr>
<th>Neurological signs</th>
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<td><strong>Putaminal</strong></td>
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<tr>
<td>Hemiplegia, hemisensory loss, homonymous hemianopsia, gaze palsy, stupor and coma</td>
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<td><strong>Cerebellar</strong></td>
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<td>Usually originate in the dentate nucles, extend into the hemisphere and 4th ventricle and possibly into the pontine. Inability to walk due to imbalance, vomiting, headache, neck stiffness, gaze palsy and facial weakness. No hemiparesis. Crucial diagnosis to make since patients frequently deteriorate and require surgery.</td>
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<tr>
<td><strong>Thalamic</strong></td>
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<td>Extend in a transverse direction to the posterior limb of the internal capsule, downward to put pressure on the tectum of midbrain or may rupture into 3rd ventricle. Hemiparesis, hemisensory loss, transient homonymous hemianopsia. Upward gaze with miotic pupils that are unreactive, peering at the tip of the nose, skewed, or “wrong way eyes” toward the weak side. Aphasia may occur if bleed affects the dominant hemisphere or neglect in non dominant hemisphere.</td>
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<tr>
<td><strong>Lobar</strong></td>
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<td>Most often affect parietal and occipital lobes. Higher incidence of seizures. Occipital hemorrhages frequently present with very dense contralateral homonymous hemianopsia. Haemorrhage in frontal region will produce contralateral plegia or paresis of leg with relative sparing of arm.</td>
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<tr>
<td><strong>Pontine</strong></td>
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<td>Medial hematoma that extends into the bass of pons. Deep coma over first few mins following hemorrhage. Total paralysis. Pupils pinpoint.</td>
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### Prognosis

- 30 day mortality from ICH 35 – 52%.
- Half of these deaths occur within the 1st few days.
- Only small no. of patients function independently after event.
  - Initial ICH vol and level of consciousness
    - ICH vol of 60cm or > on initial CT and a GCS of 8 or < predicted a 30 day mortality of 91%.
    - ICH vol. of < 30cm and GCS of 9 or > predicted a 30 d mortality of 19%.
  
2. Hematoma growth
   - Independent predictor of mortality and poor outcome
   - Each 10% increase in haematoma vol, pt were 5% more likely to die and 16% more likely to increase one point on the mRS (modified Rankin scale).

3. Early neurologic deterioration
4. Preceding anticoagulant or antiplatelet use

5. ICH score (predict mortality after ICH)
   - Components:
     - GCS
     - ICH vol
     - Intraventricular extension of hemorrhage
     - Infratentorial origin
     - Age

**Treatment**

1 Recommendations for Initial medical Therapy

**Class I**

1. Monitoring and Mx of patients with ICH should take place in an ICU setting
2. Appropriate antiepileptic therapy should always be used for treatment of clinical seizures in pts with ICH
3. Sources of fever should be treated and antipyretic med should be administered to lower temp in febrile patients with stroke

**Class II**

1. Treatment of elevated ICP should include a balanced and graded approach that begins with simple measures such as elevation of head of bed and analgesia and sedation. More aggressive therapies to decrease elevated ICP, such as osmotic diuretics (mannitol and hypertonic saline solution), drainage of CSF via ventricular catheter, neuromuscular blockage, and hyperventilation, generally require ICP monitoring, bp to maintain CPP > 70mmHg
2. Hyperglycemia should be treated
3. Target bp in various situations and potential meds are: labetolo, nicardipine,.. esmolol, enalapril, hydralazine, nipride, nitroglycerin.
4. Brief period of prophylactic antiepileptic therapy soon after ICH onset may reduce risk of early seizures in patient with lobar hemorrhage

**Intracranial pressure control**

- ICP monitoring
- Cerebral perfusion pressure
- Maintained above > 60mmHg
- CPP = MAP – ICP
- Medical therapies: Mannitol, hyperventilation, barbiturate coma, temperature management

**Mannitol**

- To achieve plasma hyperosmolality (300 -310 mosmol/kg)
- Hypotonic fluids are contraindicated
- Mild hypernatremia should be tolerated

**Hyperventilation**

- PaCO2 of 25 – 30 mmHg
- Effect only lasts for mins to a few hours

Steroids should not be used to lower ICP. Dexamethasone did not improve outcome and did increase complication rates, primarily infection

**Barbiturate Coma**

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1 Guidelines for management of Spontaneous ICH in adults June 2007 www.stroke.ahajournals.org
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- Depresses cerebral metabolic activity
- Effective in lowering refractory hypertension but ineffective or potentially harmful as 1st line or prophylactic treatment in pts with brain injuries
- Common complication: hypotension

Temperature management
- Cooling to 32-34°C can be effective in lowering refractory intracranial hypertension
- Longer term (24–4hrs) is ass with relatively high rate of complications

Hemostatic therapy
- Activated recombinant factor VIIa
- Promotes hemostasis at sites of vascular injury
- Evidence suggests that treatment within first 3-4 hr after onset to slow progression of bleeding has shown promise in one moderate sized phase II trial
- Efficacy and safety of this treatment must be confirmed in phase III trials before its use

Recommendations for Prevention of Deep Vein Thrombosis and PE
Class I
Patients with acute primary ICH and hemiparesis/hemiplegia should have intermittent pneumatic compression for prevention of CTE

Class II
1. After documentation of cessation of bleeding, low dose S/C LMWH or enoxaparin may be considered in pts with hemiplegia after 3-4 days from onset
2. Pts with an ICH who develop an acute proximal VTE or subclinical PE should be considered for acute placement of vena cava filter
3. Decision to add long-term antithrombotic therapy several weeks or more after placement of vena cava filter must take into consideration the likely cause of hemorrhage (amyloid has higher risk of recurrent ICH vs HT), as well as conditions with increased arterial thrombotic risk (eg AF) and the overall health and mobility of the patient

Recommendations for Surgical Approaches
Class I
1. Patients with cerebellar hemorrhage >3cm who are deteriorating neurologically or who have brainstem compression and/or hydrocephalus from ventricular obstruction should have surgical removal of hemorrhage asap.

Class II
2. Although stereotactic infusion of urokinase into the clot cavity within 72 hrs of ictus apparently reduces the clot burden and risk of death, rebleeding is more common and functional outcome is not improved; therefore, its usefulness is unknown
3. For patients presenting with lobar clots within 1cm of the surface, evacuation of supratentorial ICH by standard craniotomy might be considered.

Class III
4. The routine evacuation of supratentorial ICH by standard craniotomy within 96 hrs of ictus is not recommended
Recommendations for Timing of Surgery

Class II

1. No evidence at present indicates that ultra-early craniotomy improves functional outcomes or mortality rate. Operative removal within 12 hrs, particularly when performed by less invasive methods, has most supportive evidence, number of subjects treated within this window is very small. Very early craniotomy may be associated with an increased risk of recurrent bleeding.

Class III

1. Delayed evacuation by craniotomy appears to offer little if any benefit with a fairly high degree of certainty. In those patients presenting in coma with deep hemorrhages, removal of ICH by craniotomy may actually worsen outcome and is not recommended.

Recommendations for Decompressive Craniectomy

Class II

1. Too few data currently exist to comment on the potential of decompressive craniectomy to improve outcome in ICH

Resumption of antiplatelet therapy

Not much data
Asprin reduces ischemic stroke by 25%.
AHA/ASA guidelines state that antiplatelets should be discontinued for at least 1 – 2 weeks

Back to the question:

Key information given:
Pt's symptoms started 2 days ago with insidious onset of symptoms. He is alert with mild facial weakness and hemiparesis. His SBP is quite high at 170. CT brain shows quite a large bleed probably more than 30mm.

Options given:

a) Surgery
   No clear indication that this patient needs surgery at this point in time as the patient is not neurologically bleeding and it would be a delayed evacuation with has little or no benefit

b) Dexamethasone
   No benefit in fact increases complication rates eg infection

c) GTN infusion
   Bp is at 170 Cut off from the guidelines is > 180mmHg. Therefore no need to lower bp.

Suggested Recommended Guidelines for Treating Elevated Blood Pressure in Spontaneous ICH

1. If SBP is > 200 mm Hg or MAP is > 150 mm Hg, then consider aggressive reduction of blood pressure with continuous intravenous infusion, with frequent blood pressure monitoring every 5 minutes.
2. If SBP is > 180 mm Hg or MAP is > 130 mm Hg and there is evidence of or suspicion of elevated ICP, then consider monitoring ICP and reducing blood pressure using intermittent or continuous intravenous medications to keep cerebral perfusion pressure _60 to 80 mm Hg.
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3. If SBP is _180 mm Hg or MAP is _130 mm Hg and there is not evidence of or suspicion of elevated ICP, then consider a modest reduction of blood pressure (eg, MAP of 110 mm Hg or target blood pressure of 160/90 mm Hg) using intermittent or continuous intravenous medications to control blood pressure, and clinically reexamine the patient every 15 minutes.

d) Mannitol infusion
   No indication that he has a high ICP so you would not give mannitol unless we know what his ICP is.

   Therefore the answer is (believe it or not)
   e) Observation.