This quiz is published on behalf of the education committee of the SNACC.
1.58 Y/M with past medical history of hypertension is admitted with headache and loss of consciousness. CT scan indicates that he has a subarachnoid hemorrhage. All the following are complications of SAH EXCEPT?

A. Hydrocephalus  
B. Seizures  
C. Cerebral vasodilation  
D. Rebleeding
A. Hydrocephalus

- Hydrocephalus following SAH is a common complication with an incidence varying from 6% to 67%. It can progress acutely (0-3 days), sub-acutely (4-13 days) or chronically (after 13 days). Patients present with worsening neurological status and diagnosis is confirmed by CT scan. Placement of VP shunt is the treatment of choice.
B. Seizures

- In SAH, blood can irritate the cerebral cortex resulting in seizures. The incidence of seizures after SAH ranges from 3% to 26%. Risk factors for onset of seizures after SAH include presence of intracerebral hemorrhage, anterior circulation aneurysms, hypertension, ischemic infarcts revealed on late CT scans, initial loss of consciousness lasting longer than 1 hour, hemiparesis, Hunt and Hess grade greater than III, and younger age (< 40 years). The incidence of late or recurrent epilepsy following SAH is 7% to 12%.

• Cerebral vasospasm and not cerebral vasodilation develops 3-12 days after SAH and is an important cause of morbidity and mortality. The amount and location of blood in the basal cisterns correlates with the incidence and severity of delayed cerebral vasospasm. The formation of oxyhemoglobin, free radicals, lipid peroxidation products, calcium, and prostaglandins by the subarachnoid blood causes changes such as contraction of the smooth muscles to the thickening of the vessel wall, leading to cerebral vasospasm.

D. Rebleeding

• The incidence of re-bleeding in patients with SAH is 11%. The prognosis for patients with rebleeding is poor with mortality rates ranging between 64% and 90%. The patient is at the greatest risk of re-bleeding within the first 24 h. Early clipping of ruptured aneurysms allows to eliminate risk of rebleeding. In patients where definitive treatment of aneurysm is delayed, short term use of antifibrinolytics can be considered to reduce risk of rebleeding.

2. He underwent surgery and on postoperative day 7 a transcranial Doppler in the ICU is indicative of vasospasm and she exhibits some neurological changes. All of the following strategies can be used to treat cerebral vasospasm EXCEPT

A. Administration of nimodipine
B. Maintaining SBP 20% below baseline
C. Intraarterial papaverine injection
D. Transluminal angioplasty

Go to Q3
A. Administration of nimodipine

- Nimodipine is a calcium antagonist that is thought to reduce the rate of cerebral vasospasm by reducing the influx of calcium into the vascular smooth muscle cells. According to the American Stroke Association, oral nimodipine should be administered to all patients with SAH (class I, level A). The Neurocritical care society advocates that oral nimodipine (60 mg every 4 h) should be administered after SAH for a period of 21 d (high quality of evidence, strong recommendation).


B. Maintaining SBP 20% below baseline

- Recent studies have shown that the maintenance of euvolemia and induced hypertension is of more benefit compared to the triple-H therapy. The American Stroke Association and Neurocritical Care Society recommend maintaining SBP less than 160 mm Hg and mean arterial pressure less than 110 mm Hg before the ruptured aneurysm is secured to reduce the risk of rebleeding unless the blood pressure is increased at baseline or cardiac status precludes it.
C. Intraarterial vasodilator injection

- Intraarterial vasodilator therapy is used in patients where distal vessels are affected. The use of intraarterial vasodilator therapy is recommended to treat symptomatic cerebral vasospasm by the American Stroke association and the Neurocritical care Society especially in patients who are not responding to hypertensive therapy.
D. Transluminal angioplasty

- There is a Class IIb, Level B evidence for the use of endovascular treatment such as transluminal angioplasty in the management of patients with cerebral vasospasm refractory to treatment.
3. All of the following statements about delayed cerebral ischemia (DCI) in patients with SAH are TRUE EXCEPT

A. Uncontrolled intracellular shift of calcium causes cerebral ischemia
B. Cortical spreading depolarization (CSD) is the pathophysiologic basis of DCI
C. Reduced nitric oxide levels and increased fibrin and platelet aggregation contributes to DCI
D. Cerebral ischemia can only occur in areas affected by angiographic vasoconstriction
A. Uncontrolled intracellular shift of calcium causes cerebral ischemia

- Brain cells are highly dependent on oxygen and hence during ischemia or hypoxia, ATP stores rapidly drop, causing the failure of sodium/potassium ATPase pumps leading to membrane depolarization and opening of voltage-sensitive calcium channels and reversal of the sodium–calcium exchanger. This leads to increased intracellular calcium either directly or indirectly which activates proteases, endonucleases and other factors involved in apoptosis causing cerebral ischemia.

B. Cortical spreading depolarization (CSD) is the pathophysiologic basis of DCI.

- CSD is described as self-propagating tissue depolarization waves that lead to ischemia and are associated with increase in extracellular potassium levels, compromise of the blood-brain barrier, formation of vasogenic and cytotoxic edema, and change in regional cerebral blood flow. Neurovascular uncoupling occurs as the energy expenditure of neurons is reaching its peak, paradoxical vasoconstriction occurs, resulting in cortical hypoperfusion and energy failure.

C. Reduced nitric oxide levels and increased fibrin and platelet aggregation contributes to DCI

- Endothelial and platelet dysfunction, coagulation cascade activation, and impaired fibrinolysis can occur after SAH leading to a hypercoaguable state and formation of microthrombi contributing to DCI.

Dority JS, Oldham JS. Subarachnoid Hemorrhage. Anesthesiology Clinics, 2016, Volume 34, Issue 3, Pages 577-600
D. Cerebral ischemia can only occur in areas affected by angiographic vasoconstriction

- DCI occurs due to a number of underlying pathophysiologic mechanisms. Vasospasm is one of the mechanisms contributing to DCI. Regions of hypoperfusion are observed in patients without concomitant angiographic vasospasm and hence the statement is incorrect.

Vergouwen MD et al. Definition of delayed cerebral ischemia after aneurysmal subarachnoid hemorrhage as an outcome event in clinical trials and observational studies: proposal of a multidisciplinary research group. Stroke 2010; 41: pp. 2391-2395
4. The following statements are true regarding vasospasm EXCEPT

A. Increased flow velocity through an artery can be measured before signs of ischemia are apparent
B. Evidence of vasospasm can be detected within 3 hours of subarachnoid hemorrhage
C. Endovascular therapy has no role in the reversal of vascular narrowing due to vasospasm
D. Calcium channel blockers are useful in decreasing morbidity and mortality after subarachnoid hemorrhage
A. Increased flow velocity through an artery can be measured before signs of ischemia are apparent

- Cerebral vasospasm can be detected with a transcranial doppler prior to the presentation of neurologic deficits, prompting earlier intervention. TCD is a noninvasive test that allows indirect detection of large-vessel narrowing based on quantification of acceleration of flow. Intracerebral vasospasm is associated with increased flow velocity measurements in arteries.

B. Evidence of vasospasm can be detected within 3 hours of subarachnoid hemorrhage.

- Studies using TCD ultrasonography have confirmed the delayed onset and time course of vasospasm. Evidence of vasospasm initially was seen 3 days after subarachnoid hemorrhage, appeared to be maximal between days 6 and 8, and was much reduced by day 12. It is unlikely for vasospasm to be evident within 3 hours.

C. Endovascular therapy has a role in the reversal of vascular narrowing due to vasospasm

- Interventional endovascular therapies include transluminal angioplasty and infusion of intra-arterial vasodilators. They are used to treat patients with symptomatic vasospasm and those who do not respond to medical measures or cannot tolerate them owing to comorbidities. The effects of balloon angioplasty is sustained but cannot reach distal vessels. Intra-arterial nimodipine, verapamil, nicardipine, and milrinone have all been shown to dilate blood vessels and augment cerebral blood flow.

D. Calcium channel blockers are useful in decreasing morbidity and mortality after subarachnoid hemorrhage

- Nimodipine, a calcium channel blocker, has been the standard of care for SAH for almost 3 decades. Nimodipine seems to exert its effect owing to its action on neuronal calcium channels. Although it does not reduce the rate of vasospasm, it improves outcomes.


Incorrect
5. The above patient had a brain tissue oxygen monitor inserted. All the following statements about brain oxygen tension monitoring are TRUE EXCEPT

A. SjvO2 and NIRS are non invasive and PbiO2 is invasive
B. Treatment should be initiated when there is a 20% or more reduction from baseline of rScO2 (regional cerebral oxygen saturation) values
C. PbiO2 is a focal monitor while SjvO2 is global
D. The PbiO2 probe should be placed in the penumbral region
A. SvjO2 and NIRS are non invasive and PtiO2 is invasive

• SvjO2 and PtiO2 are invasive methods used for brain oxygen tension monitoring. Near-infrared spectroscopy (NIRS) derived cerebral oximetry is currently the only noninvasive, bedside monitor of cerebral oxygenation.

Kirkman MA, Smith M. Brain Oxygenation Monitoring. Anesthesiology Clinics, 2016, Volume 34, Issue 3, Pages 537-556
B. Treatment should be initiated when there is a 20% or more reduction from baseline of rScO2 values

- NIRS measures regional cerebral oxygen saturation (rScO2) with high temporal and spatial resolution and permit simultaneous measurement over multiple regions of interest. The normal range of rScO2 is between 60% and 75%. rScO2 values of less than or equal to 50% or a greater than or equal to 20% reduction from baseline as a trigger for initiating measures to improve cerebral oxygenation.

Kirkman MA, Smith M. Brain Oxygenation Monitoring. Anesthesiology Clinics, 2016, Volume 34, Issue 3, Pages 537-556
C. PtiO2 is a focal monitor while SvO2 is global

- PtiO2 monitoring allows direct measurement of focal tissue oxygen tension in a specific region of the brain whereas SvO2 is an indicator of the balance between global cerebral oxygen delivery and metabolic demand.

D. The PtiO2 probe should be placed in the penumbral region

- PtiO2 gives a focal measure of the brain tissue oxygen tension. The region of interest interrogated by a PtiO2 probe is approximately 17 millimeter square. Probe placement is therefore crucial. In patients with focal lesions, such as intracerebral hemorrhage (ICH) or traumatic contusions, a perilesional location is favored, whereas in aneurysmal SAH, probe placement in appropriate vascular territories is advised.

Kirkman MA, Smith M. Brain Oxygenation Monitoring. Anesthesiology Clinics, 2016, Volume 34, Issue 3, Pages 537-556