

EANS/UEMS European examination in neurosurgery

Variants of questions with answers (compilation - Vyacheslav S. Botev,
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CASE HISTORIES IN NEUROVASCULAR

Case 1

It is five o'clock in the morning, and you are a Medical Officer providing medical care to the deployed International Security Assistance Force (ISAF).

Word was received that an improvised explosive device (IED) had detonated next to an army vehicle and that there were two casualties on route with unknown injuries. You have five people on your forward surgical team (four residents and one attending), and two are in Emergency Department already.

You saw first casualty - a young soldier with more wounds than you could count. You can't believe it but this 21-year-old soldier is fully conscious. He has multiple facial fractures, left-sided hemiparesis, a right small pupil and ptosis. After primary and secondary survey full trauma series were performed including the angiography:



1. What does the angiogram show?
2. How often are vascular injuries associated with head injuries?
3. Where do intracranial dissections most commonly occur?
4. What is the most common site for vertebral artery dissection?
5. What is the most common site for carotid artery dissection?
6. Describe the protocol for conservative management of arterial dissections.
7. When is intervention indicated?

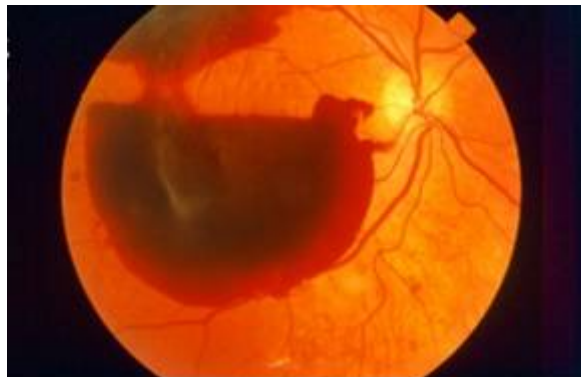
Case 2

You are working with your attending on a busy day. He tells you to go set up the angiography and fundoscopy for a 41-year-old female with HH grade II SAH.

There is aneurysm on cerebral angiography:



1. Where is it?
2. What special surgical procedure helps approach this?
3. What deficit might you expect post-operatively and how would you manage it?
4. What can you see on her fundoscopy?



5. What is the ISAT trial?
6. What is Hijdra method of grading subarachnoid hemorrhage after SAH?
7. What is Columbia University Medical Center management protocol for acute SAH?

Case 3

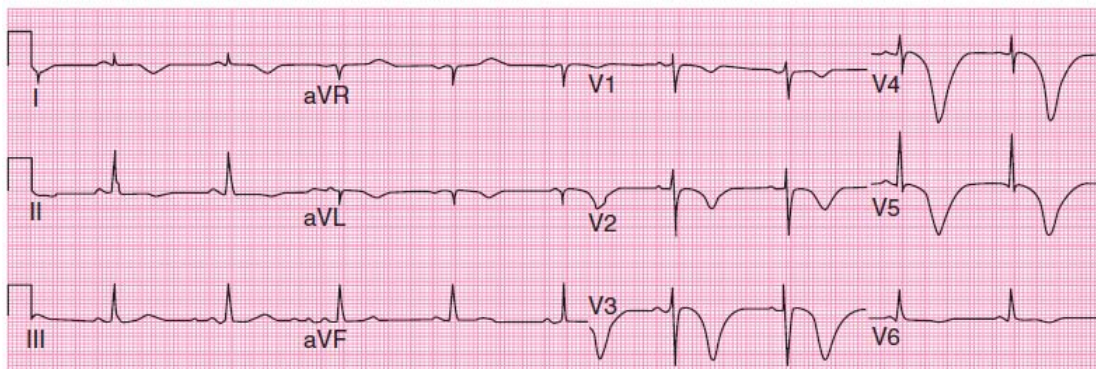
My first shift on my own with no training wheels (aka Preceptor) was in the Neuro ICU on Christmas Eve and it was the overnight shift. Sounding depressing yet? Ok. I can deal with that. Working on your own can be very liberating.

One of your patients is a high-grade (HH IV, Fisher 3, mFS 4) SAH s/p (status post) A-comm aneurysm coiling who was able to localize to painful stimulation bilaterally suddenly becomes less aroused and has right hemiparesis on bleed day 7 (the very first day of SAH is day zero).

1. What is the diagnosis?
2. How do you approach symptomatic vasospasm?
3. What are currently available therapy options for symptomatic cerebral vasospasm?

Case 4

A 35-year-old woman presents after collapsing at home briefly after complaining of headache. Patient is admitted with HH III, F 3, mFS 4, and receives EVD for IVH and acute obstructive hydrocephalus. The patient gets endotracheal intubation and is admitted to the Neuro ICU. In the unit, the BP is 60/40 mm Hg, sinus tachycardia at 110 bpm, SaO₂ drops to 70%, bilateral rhonchi and crackles on auscultation, on mechanical ventilator mode of assist control–volume control, set rate of 14, FIO₂ (fraction of inspired air) of 60%, PEEP (positive end-expiratory pressure) of 8. The ECG shows nonspecific T-wave and ST changes, troponin = 2.5.

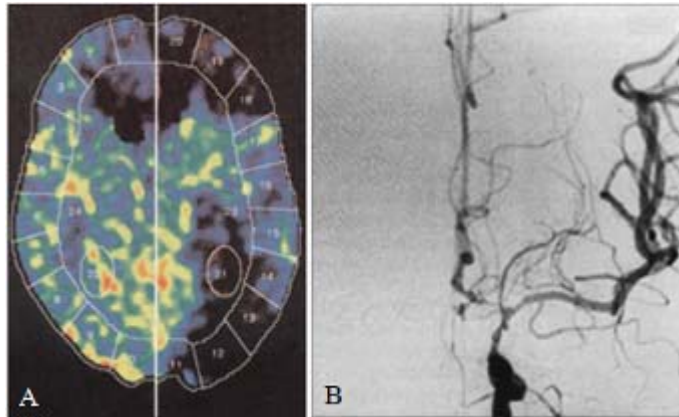


Chest x-ray is consistent with acute pulmonary edema.

1. What is the diagnosis?
2. How would you manage this?
3. How do you differentiate neurogenic stunned myocardium from acute myocardial infarction?

Case 5

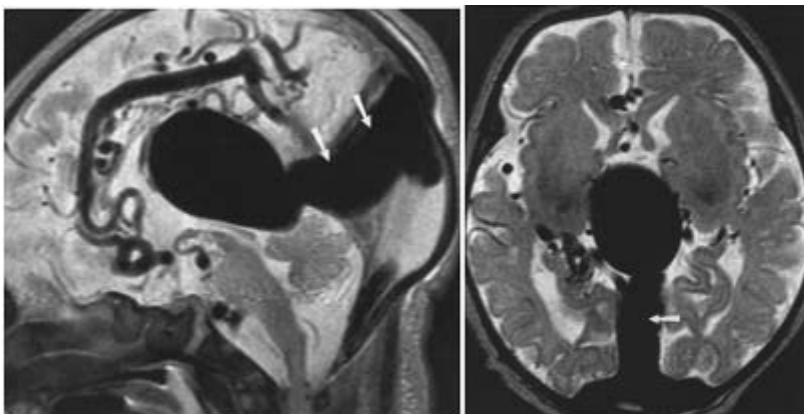
A 61-year-old female presents with grade I SAH. She underwent successful clipping of an Acom aneurysm and was intact post-operatively. Then on day 4 in the middle of a night, the ICU resident calls to notify you that she developed increasing lethargy. Her CT scan was normal but her xenon CT (A) and her angiogram (B) were not:



1. What is the diagnosis?
2. What is the medical treatment?
3. If despite this the patient continues to deteriorate what procedure might you consider and when?
4. What is delayed cerebral ischemia?
5. What are most common complications of subarachnoid hemorrhage?

Case 6

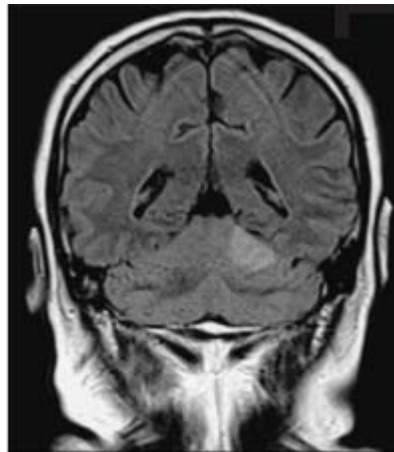
It is 3 o'clock in the morning on July 2 during your postgraduate year 2 (PGY 2). The intern finished medical school about an hour ago and is looking to your expert leadership with a child admitted to the hospital a week ago for hydrocephalus. The intern wants to know everything about this case.



1. What is the diagnosis?
2. What are the classic clinical presentations for this entity?
3. What are the two major types of vascular abnormalities associated with this entity?
4. What anatomic structure or variant is denoted by the arrows?

Case 7

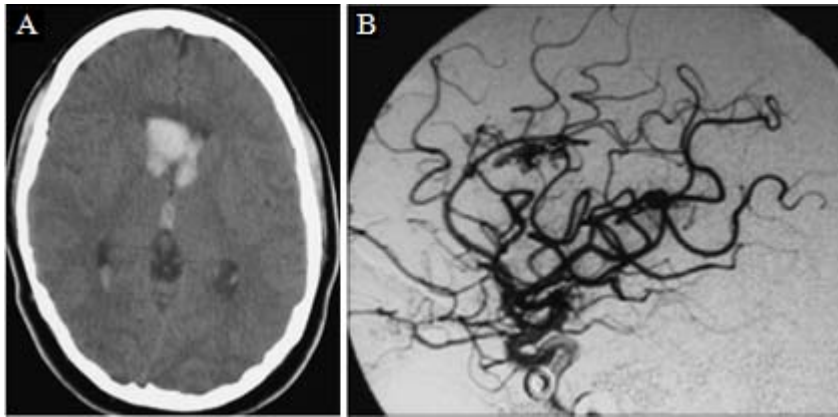
A 67-year-old male developed acute headache with severe vertigo, along with recurrent vomiting. His neurologic examination revealed vertical nystagmus, but no focal motor or sensory deficits. His brain CT was normal. MRI was carried out:



1. What does this MR image suggest?
2. How should this be managed?

Case 8

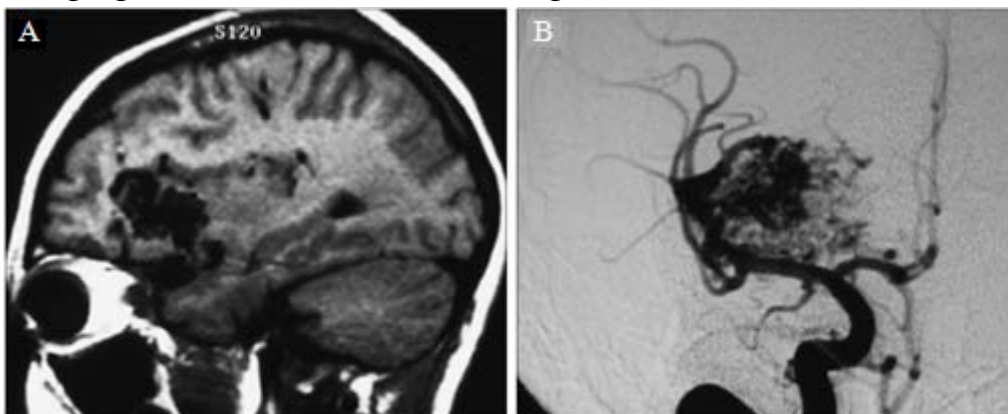
This 27-year-old female suffered a sudden onset of severe headache and passed out. Her husband took her to the local emergency department. She regained consciousness in the car and was neurologically intact by the time she was evaluated. A CT scan was performed (A). Based on the CT scan an angiogram was obtained (B):



1. What does the CT scan demonstrate, what is the leading differential diagnosis, and why was the angiogram performed acutely?
2. What does the angiogram show, how would you classify this lesion, and what is its natural history?
3. What are the various treatment options and what are the relative pros and cons to each?
4. How would you grade intraventricular hemorrhage (IVH)?
5. How would you grade hydrocephalus associated with IVH after SAH?
6. What is the bicaudate index?

Case 9

This 26-year-old female presented with 10 years of right-sided proptosis and increasing headaches. MRI revealed a lesion (A). As a result she was sent for an angiogram (B) which shows this right frontal AVM.

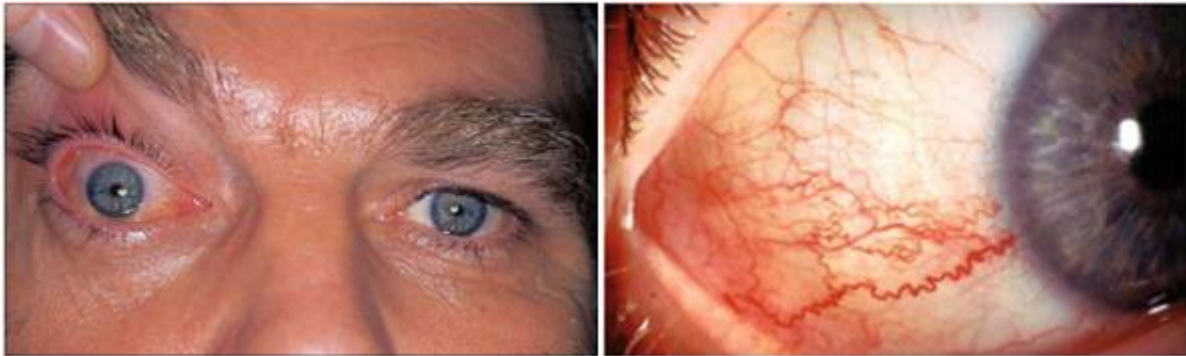


1. How likely is treatment of the AVM to cure or significantly improve her symptoms?
2. Would you consider this lesion for radiosurgery?
3. If you were to recommend surgery, would you embolize the AVM pre-operatively?

4. What surgical approach would you use?
5. How do AVMs appear on MRI?
6. What are the average annual risk and the lifetime risk of hemorrhage for an unruptured AVM?

Case 10

A 50-year-old male was knocked out by a fall down a flight of stairs. Upon waking he was found to have a complete right ptosis, and generalized limitation of movements of the right eye.



1. What other clinical features are shown and what is the diagnosis?
2. What other clinical features would confirm this diagnosis?
3. How might the vision of the right eye be affected?
4. What is the further management?

CASE HISTORIES IN NEUROVASCULAR

Answers

Case 1

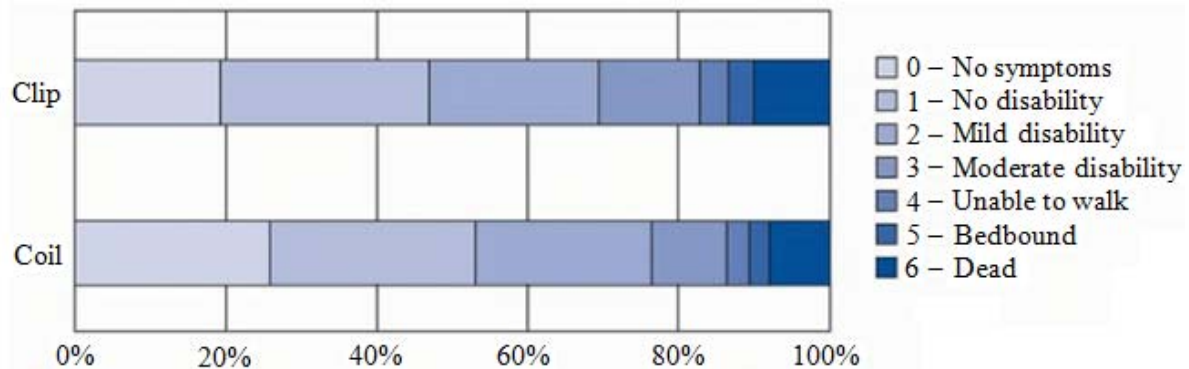
1. The angiogram demonstrates an ICA dissection.
2. Previously, blunt traumatic vascular injuries were thought to be extremely rare. With screening, however, the incidence may be as high as 1% of blunt injuries. Mandible fractures, facial fractures, spine fractures and DAI appear to be associated with an increased incidence of blunt vascular injury. A high index of suspicion must be maintained as sequelae are often delayed in presentation with disastrous, irreversible consequences.
3. Vertebral >> basilar > internal carotid > MCA > ACA, PCA, PICA.
4. Between C2 and occiput.
5. 2 cm distal to the ICA origin.
6. If no bleeding occurs, heparinize the patient for 1 to 2 weeks, then prescribe Coumadin for up to 6 months. Repeat the angiogram to assess healing prior to suspending treatment.
7. Dissections presenting with SAH, intradural dissections, persistent symptoms, angiographically progressive dissections, or if conservative management fails.

Case 2

1. PICA origin.
2. Far lateral suboccipital craniotomy gives you early control of the vertebral artery and a flatter view across the anterior brainstem.
3. Lower cranial nerve palsies are usually temporary but swallowing evaluation is necessary before allowing the patient to eat. GDC treated cases often have the same problems (therefore this is an effect of the bleed) and PEG is often necessary.
4. Macular preretinal hemorrhage in a patient with Terson syndrome.
Terson syndrome can present with dome-shaped hemorrhages in the macula. A macular “double ring” sign may be seen with the inner ring caused sub-ILM hemorrhage and the outer ring caused by sub-hyaloid hemorrhage.

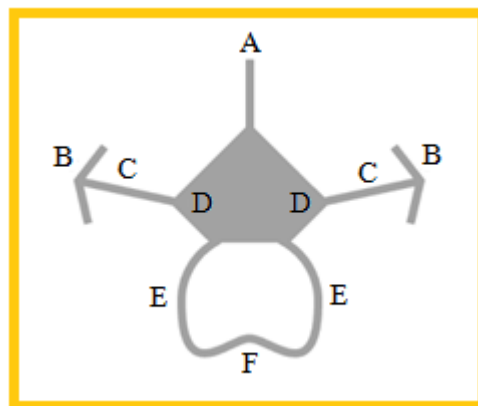
Although intraocular hemorrhages most frequently develop in the first hour after SAH, Terson syndrome can have a delayed onset, with reports of intraocular hemorrhage occurring up to 47 days after SAH.

5.



Distribution of modified Rankin Scale outcome scores in the ISAT trial, which compared surgical clipping to endovascular coiling as the primary treatment for SAH due to ruptured intracranial aneurysm. The results showed that 190 of 801 (23.7%) patients allocated to coil treatment were dependent or dead at 1 year, compared with 243 of 793 (30.6%) who were allocated neurosurgical clipping.

6.



Hijdra scale.

Hijdra method of grading subarachnoid hemorrhage identifies 10 basal cisterns and fissures: (A) frontal interhemispheric fissure; (B) sylvian fissure, lateral parts; (C) sylvian fissure, basal parts; (D) suprasellar cistern; (E) ambient cistern; and (F) quadrigeminal cistern. The amount of blood in each cistern and fissure is graded 0, no blood; 1, small amount of blood; 2, moderately filled with blood; and 3, completely filled with blood. The sum score is 0 to 30 points.

7. Columbia University Medical Center management protocol for acute SAH

Blood Pressure	Control elevated BP during the preoperative phase (systolic BP <160 mm Hg) with IV labetalol or nicardipine to prevent rebleeding.
Rebleeding Prophylaxis	4 g ε-aminocaproic acid IV upon diagnosis followed by 1 g/h until aneurysm repair, for a maximum of up to 72 h after ictus.
IV Hydration	Preoperative: normal (0.9%) saline at 1.0-1.5 mL/kg/h Postoperative: normal (0.9%) saline at 1.0-1.5 mL/kg/h and 250 mL 5% albumin every 2 h if the CVP is ≤ 5 mm Hg.
Laboratory Testing	Periodically check complete complete blood count and electrolytes. Obtain serial ECGs and check admission cardiac troponin 1 (cT1) to evaluate for cardiac injury; perform echocardiography in patients with abnormal ECG findings or cT1 elevation.
Seizure Prophylaxis	Fosphenytoin or phenytoin IV load (15-20 mg/kg); discontinue on postoperative day 1 unless patient has seized, is poor grade, has focal cortical pathology, or is otherwise unstable.
Vasospasm Prophylaxis	Nimodipine 60 mg PO every 4 h until day SAH 21 or discharge
Physiologic Homeostasis	Cooling blankets to maintain temperature ≤ 37.5° C, Insulin drip to maintain glucose at 100-120 mg/dL, Transfuse to maintain hemoglobin > 7.0 g/dL (in the absence of active cerebral or cardiac ischemia).
Ventricular Drainage	Emergent external ventricular drain (EVD) placement in all comatose patients (Hunt-Hess IV/V), as well as lethargic patients with hydrocephalus, Begin trials of clamping EVD and monitoring ICP on day 3 after placement, Perform ventriculoperitoneal shunting during subacute phase of illness in patients with persistent cognitive dysfunction and ventriculomegaly.
Vasospasm Diagnosis	TCD sonography every 1-2 days until the 10 th day after SAH, CT or MR perfusion on day 4-8 after SAH if high risk.
Therapy for Symptomatic Vasospasm	Place patient in Trendelenberg (head down) position, Infuse 500 mL 5% albumin over 15 min. If the deficit persists, raise the systolic BP with phenylephrine or norepinephrine until the deficit resolves (target: 180-220 mm Hg). 250 mL 5% albumin solution every 2 h if the CVP is ≤ 8 mm Hg or the PADP is ≤ 14 mm Hg. If refractory, monitor cardiac output and add dobutamine or milrinone to maintain cardiac index ≥ 4.0 L/min/m ² . Transfuse to maintain hemoglobin > 10.0 g/dL. Emergency angiogram for intra-arterial verapamil or cerebral angioplasty unless the patient responds well to the above measures.

PADP, pulmonary artery diastolic pressure.

Case 3

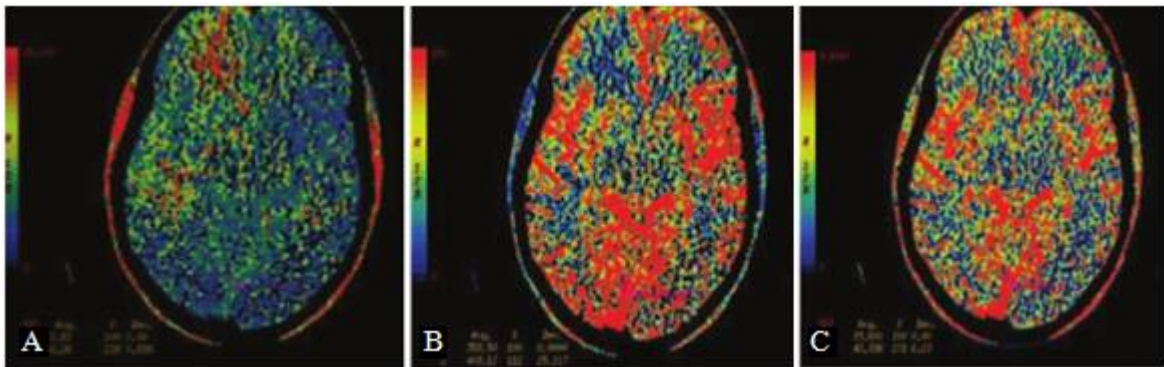
1. Symptomatic vasospasm.
2. A. Transcranial Doppler (TCD).

TCD has been used for many years for monitoring SAH patients who are at risk for developing vasospasm. The TCD velocities rise in the segments of brain vasculature where vasospasm occurs. A mean flow velocity greater than 120 cm/s is commonly used as a reference point for “TCD spasm.”

B. CT Angiography (CTA) and CT Perfusion (CTP).

CTA can provide a rapid, noninvasive method of visualizing the brain vasculature in patients suspected of having vasospasm. CTA can provide 2-D and 3-D images of the vessels and can be useful in detecting vasospasm and brain aneurysms without the risks that are associated with conventional angiography.

CTP provides three different perfusion maps.



Computed tomographic perfusion. A. Mean transit time (MTT). B. Cerebral blood flow (CBF). C. Cerebral blood volume.

- a. Mean transit time (MTT) map: Time (in seconds) measured for the contrast material to reach the cerebral hemispheres. In the event of vasospasm, the areas that are supplied by the spasm vessel will have prolonged MTT.
- b. Cerebral blood flow (CBF) map: The blood volume per given time. The areas that are supplied by the spasm vessel will have corresponding reduced CBF (usually the delayed MTT and reduced CBF occur concurrently).
- c. Cerebral blood volume (CBV) map: The blood volume in the event of vasospasm shows either normal or increased blood volume if the cerebral autoregulation is intact (by compensatory self-dilatation of the brain vessels). This is the window of opportunity for appropriate medical and endovascular interventions. When and if the CBV map shows reduced volume, this may indicate complete infarction and hence irreversible ischemic injury.

C. Continuous Electroencephalography (cEEG).

cEEG may be helpful in detecting vasospasm in high-grade SAH patients in whom clinical examinations may be of limited use.

3.

A. Triple-H therapy.

B. Invasive therapy options for symptomatic vasospasm.

There are a number of different vasodilators available for IA therapy of vasospasm in patients with aneurysmal SAH. These include papaverine, nicardipine, verapamil, and more recently milrinone. These vasodilators often produce an immediate result in increasing the vessel calibers, but there is a limitation: the positive effect may not last long.

C. Intrathecal (IT) infusion and basal cistern implants of calcium channel blockers.

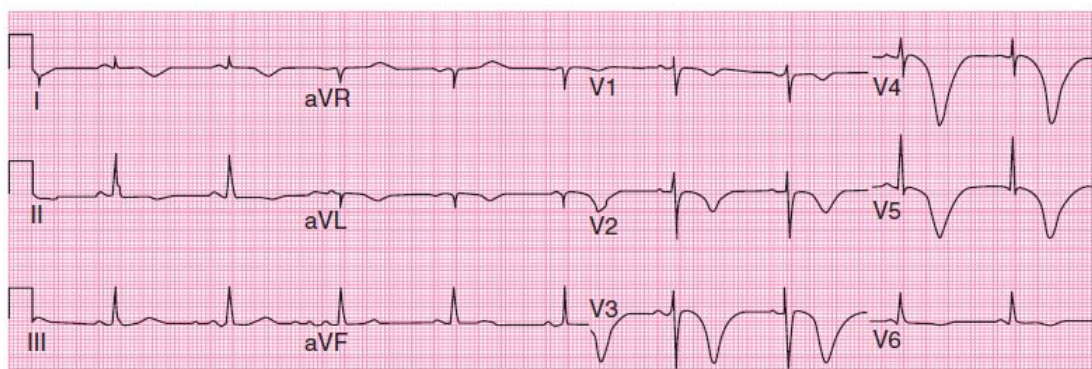
D. Intra-aortic balloon counterpulsation therapy.

E. NeuroFlo Device.

Case 4

1. Neurogenic stunned myocardium.

Neurogenic stunned myocardium is a physiologically interesting phenomenon that is associated with a number of different disease states. For neurointensivists, a good example of this syndrome is in the setting of high-grade aneurysmal SAH. There are a number of terminologies that are considered synonymous with this syndrome and that share a similar proposed pathophysiology: neurogenic stunned myocardium, takotsubo cardiomyopathy, broken-heart syndrome, contraction band necrosis syndrome, and Gebrochenes-Herz syndrome among others. Despite different terms and variations between each of these syndromes, there is a common denominator that links all of these phenomena: mental stress.



Cerebral T wave in neurogenic stunned myocardium syndrome (deep, inverted T waves with prolonged QTc intervals, so-called “cerebral” T waves are seen in this patient with aneurysmal SAH).

2. Typical stunned myocardium patients should be treated with securing of the ruptured aneurysm in order to avoid rebleed and then providing appropriate hemodynamic support (avoid the use of pure α_1 -adrenergic receptor agonist). The use of inotropic agents in order to support the reduced contractility and low Ejection Fraction (EF) while paying close attention to keep the patient euvolemic is essential (neglecting to address intravascular volume depletion in the setting of stunned myocardium would require a higher than necessary amount of vasopressors and may cause worsening of the cardiac injuries). Initiating prophylactic triple-H therapy in a patient with neurogenic stunned myocardium during the first few days of SAH may be more harmful than beneficial.

3. While the ECG alone cannot differentiate myocardial infarction (MI) from stunned myocardium, there are a few helpful tips: severely depressed (EF) with nonspecific, cerebral T waves 10-fold higher troponin values for MI and usually only mildly elevated troponin for stunned myocardium and quick reversibility of stunned myocardium with adequate hemodynamic support by judicious use of pressors and inotropic agents (eg, avoiding increased use of phenylephrine while the patient is severely volume depleted and having an EF of 20%: simply using IV Neo-Synephrine [phenylephrine] prophylactically to increase the blood pressure in the absence of symptomatic vasospasm is only going to make cardiac injury worse).

Case 5

1. Symptomatic vasospasm.

2. Hypertensive, hypervolemic therapy with hemodilution to a hematocrit of 30. Nimodipine is also given.

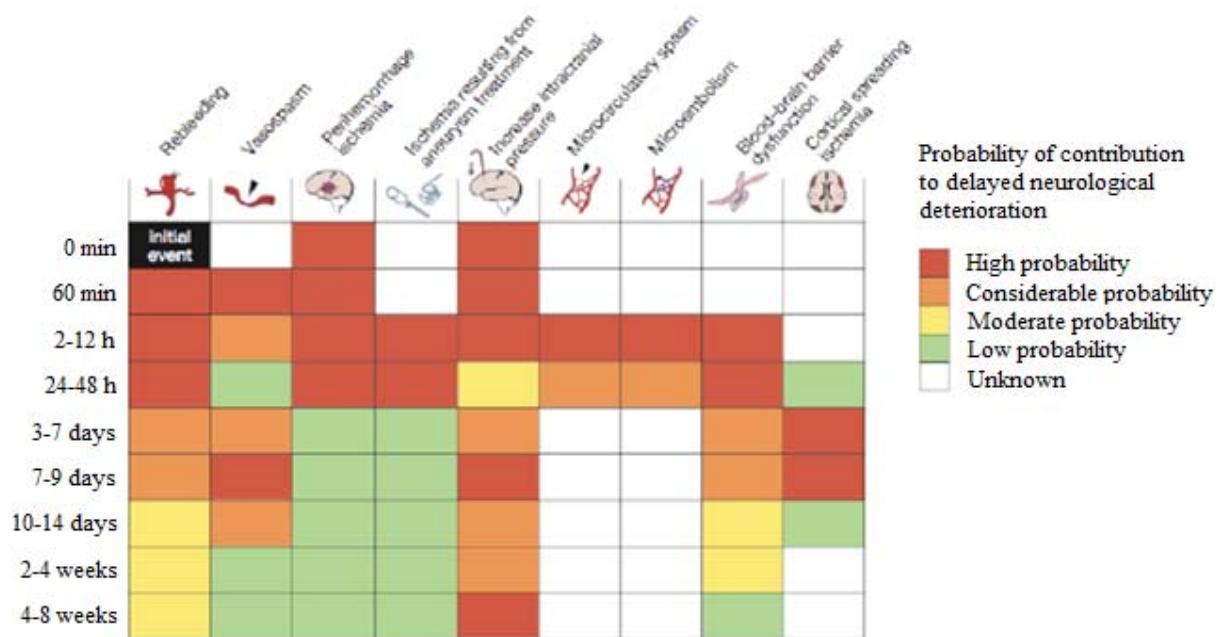
3. Balloon angioplasty should be considered for spasm of the proximal A1, M1, vertebral, basilar and ICA. For distal spasm intra-arterial papaverine is an option. Either should be instituted as soon as it is determined that the patient is medically refractory.

4. Delayed cerebral ischemia from vasospasm accounts for a large proportion of morbidity and mortality after SAH. Progressive arterial narrowing develops after SAH in approximately 70% of patients, but delayed ischemic deficits develop in only 20% to 30%. The process begins 3 to 5 days after hemorrhage, becomes maximal at 5 to 14 days, and gradually resolves over 2 to 4 weeks. Accordingly, deterioration attributable to vasospasm never occurs before the third day after SAH, and occurs with peak frequency between 5 and 7 days. There is a strong relationship between the amount of cisternal blood seen on the initial CT and the risk for development of symptomatic ischemia; for uncertain

reasons, the presence of large amounts of blood in the lateral ventricles adds to this risk.

Symptomatic vasospasm usually involves a decrease in the level of consciousness, hemiparesis, or both, and the process is usually most severe in the immediate vicinity of the aneurysm. In more severe cases, the symptoms develop earlier after aneurysm rupture, and multiple vascular territories are involved.

5. The initial hemorrhage is a key prognostic factor, although its contribution to delayed neurological deterioration remains to be established. Rebleeding in a delayed fashion is a very serious cause of delayed neurological deterioration, and vasospasm is a classic cause of deterioration. Ischemia proximal to intracranial hematomas (if it occurs) and ischemia secondary to aneurysm treatment tend to occur early on, as it is recommended that most ruptured aneurysms should be treated acutely. Increased intracranial pressure can occur at any time. The more recently proposed and less explored causes of delayed neurological deterioration include microcirculatory spasm and dysfunction, microembolism, blood–brain barrier dysfunction, and cortical spreading ischemia.

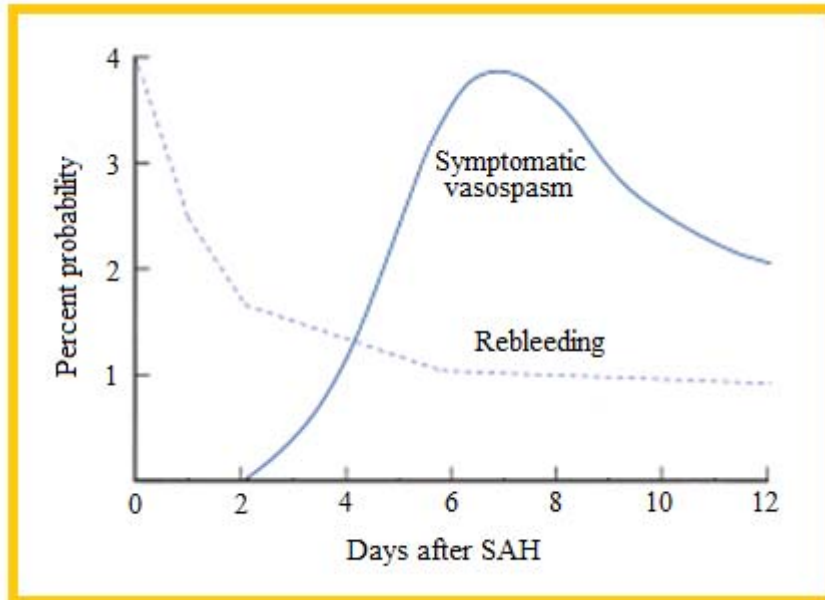


Complications of subarachnoid hemorrhage, their time of occurrence and their possible contribution to delayed neurological deterioration.

Aneurysmal rebleeding is dreaded complication of SAH. The risk of rebleeding is highest within the first 24 hours after the initial aneurysmal rupture (4%) and remains elevated (approximately 1% to 2%/day) for the next 4 weeks. The cumulative risk of rebleeding in untreated patients is 20% at 2 weeks, 30% at 1 month, and 40% at 6 months. After the first 6 months, the risk of rebleeding is 2% to 4% annually.

Poor clinical grade and larger aneurysm size are the strongest risk factor for in-hospital rebleeding. The prognosis of patients who rebleed is poor; approximately 50% die immediately, and another 30% die from subsequent complications.

Although rebleeding is often attributed to uncontrolled hypertension, elevated BP has not been convincingly linked to an increased risk of aneurysm rebleeding. Endogenous fibrinolysis of the clot around the rupture point of the aneurysm may be a more important causative mechanism.



The daily percentage probability for the development of symptomatic vasospasm (solid line) or rebleeding (dashed line) after SAH. Day 0 denotes day of onset of SAH.

Case 6

1. Vein of Galen aneurysm.
2. High-output congestive heart failure, hydrocephalus, or macrocephaly.
3. The two major types of vascular abnormalities are true arteriovenous malformations (AVMs) and direct arteriovenous fistulas (between choroidal arteries and the vein of Galen).
4. The falcine sinus. In the setting of a persistent median prosencephalic vein, the straight sinus may not develop because the median prosencephalic vein provides diencephalic venous drainage. Instead, a falcine sinus is frequently noted, as in this case (arrows).

Comment

The CT appearance of a vein of Galen aneurysm in an infant is characteristic. On unenhanced images, the vein of Galen appears as a hyperdense, demarcated mass at the level of the posterior third ventricle and diencephalon. After intravenous contrast administration, there is marked homogeneous enhancement of the malformation. MR imaging not only confirms the presence of flow within this abnormality, but also better delineates both the arterial and venous anatomy. In combination with MR angiography, MR imaging may show large choroidal arteriovenous fistulas or the presence of a parenchymal AVM. In this case, a vein of Galen aneurysm is associated with a large AVM confirmed by MR angiography. Because many women undergo obstetric ultrasound as part of their prenatal care, many of these malformations are detected in utero with color flow Doppler sonography.

Early in embryologic development, the deep brain structures and diencephalon are drained by the median prosencephalic vein. As the internal cerebral veins begin to develop, this vein slowly regresses. A caudal remnant of the median prosencephalic vein will become the normal vein of Galen. In patients with vein of Galen aneurysms related to either a parenchymal AVM or a direct arteriovenous fistula between the choroidal vessels, a persistent median prosencephalic vein may occur. Because this provides diencephalic venous drainage, the straight sinus may not form. Instead, a falcine sinus is frequently noted, as in this case (arrows). Vein of Galen malformations resulting from direct arteriovenous fistulas are frequently associated with venous obstruction and venous hypertension. High-output heart failure in newborns and hydrocephalus due to obstruction of the aqueduct of Sylvius by the enlarged vein of Galen with macrocephaly are common clinical scenarios in infants. Treatment of these malformations is catheter angiography and glue embolization of the shunt. Several embolization procedures may be necessary to completely obliterate the vascular shunts and communications. This is often done over a several-month period before the child is a toddler.

Case 7

1. This left cerebellar hyperintensity represents acute stroke. Hypertension is the main predisposing etiology, although cerebellar stroke also occurs with tumors, arteriovenous malformations, trauma, blood dyscrasias, amyloid

angiopathy, sympathomimetic use, and as a complication of intracranial or spinal surgery.

Cerebellar ischemic stroke may have a delayed hemorrhagic transformation. Central and larger strokes have more life-threatening complications, such as obstructing hydrocephalus and brainstem compression. Cerebellar stroke usually presents with headache of abrupt onset, associated nausea and/or vomiting, ataxia, and vertigo. There may be depressed consciousness, neck stiffness, dysarthria, nystagmus, gaze palsy, and facial weakness. Brainstem compression leads to rapid death. Brain MRI is the imaging modality of choice, with attention to location, size, and degree of compression of fourth and quadrigeminal cisterns. CT is less effective at posterior fossa imaging.

2. Surgery remains the main therapy for large cerebellar strokes, particularly if hemorrhagic. (We might not miss some of our cerebellar hemisphere!) Ventriculostomy is appropriate for significant hydrocephalus. Craniotomy with clot evacuation is indicated for cerebellar hemorrhages >3–4 cm in diameter with altered consciousness. However, awake patients with near-normal neurologic function and a hemorrhage <3 cm in diameter, particularly if lateral and without hydrocephalus, may be candidates for careful nonoperative management by a neurosurgeon. Large, central cerebellar strokes with loss of brainstem reflexes have poor prognosis and may be managed with supportive care only. Catheter evacuation of cerebellar hemorrhages in selected patients has been described.

Case 8

1. The CT scan shows an intraventricular hemorrhage with associated hemorrhage into the corpus callosum, without interhemispheric SAH. In a 27-year-old patient the most likely cause is either an AVM or a pericallosal artery aneurysm. Urgent angiography is required to differentiate these two pathologies (the latter will require urgent surgical or endovascular management to prevent rehemorrhage).

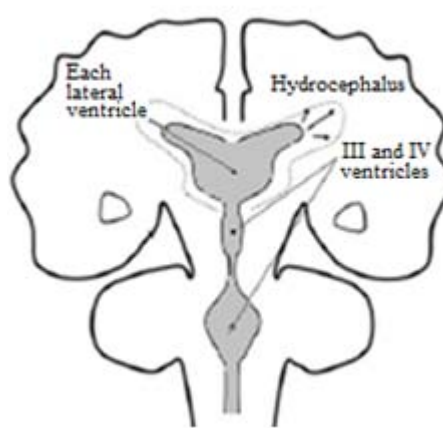
2. The angiogram demonstrates a small pericallosal AVM fed by several small branches and drained by both a cortical vein into the superior as well as the inferior sagittal sinus. By the Spetzler–Martin grading system this malformation is grade I (<3 cm = 1, 3–6 cm = 2, >6 cm = 3; deep venous drainage = 1, eloquent location = 1), which is important in terms of the

operative risk of resection (combined mortality and morbidity: 3% = grade I, 10% = grade II, 20% = grade III, 40% = grade IV, 75% = grade V). Generally speaking, the natural history of an AVM is that it carries risk of bleeding and seizures. Occasionally, they can cause neurologic deficit either through steal (controversial) or direct compression of vital structures usually by a large venous varix. They can also cause intractable headaches, especially when situated in the occipital lobe and thalamus. The risk of bleeding is often quoted at about 3% per year, with half of all bleeds leading to some residual deficit. The long-term risk of seizures is dependent on location and is difficult to estimate.

3. Treatment options include embolization followed by surgery or radiosurgery, surgery alone, or radiosurgery alone. Embolization alone is unlikely to cure safely this AVM as total opacification of the nidus including the venous drainage is unlikely to be possible without precipitating further hemorrhage or embolization of particulate matter (namely NBCA glue) into the distal pericallosal. The lesion is appropriate in size for radiosurgery (<3 cm in diameter).

4. There are three grading scales based on gross hemorrhage size and the presence of dilation within each ventricle.

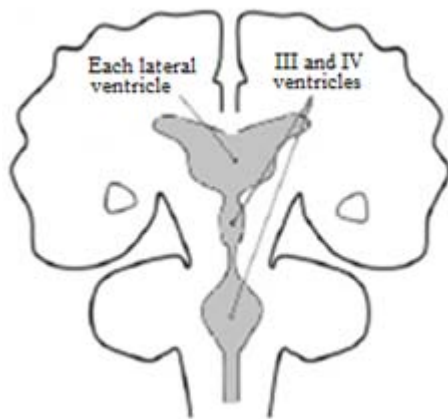
1. The IVH score (maximum score is 23).



Score	Each lateral ventricle
0	No blood
1	Less than 1g full
2	Between 1g and 2g full
3	More than 3g full
	III and IV ventricles
0	No blood
1	Any blood
	Hydrocephalus
0	No
1	Yes

The IVH score (range 0–23). The IVH score is calculated using the following equation: $3 \times (\text{right lateral ventricle score} + \text{left lateral ventricle score} + \text{hydrocephalus score}) + \text{third ventricle score} + \text{fourth ventricle score}$.

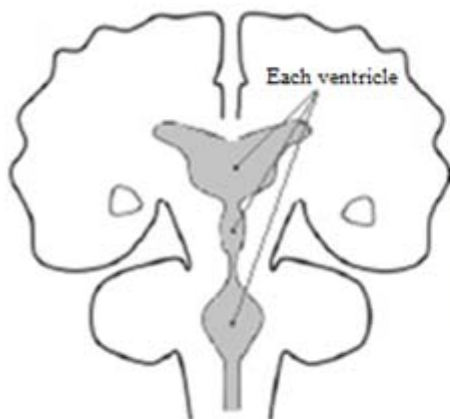
2. Graeb devised an IVH scale (maximum score is 12).



Score	Each lateral ventricle
0	No blood
1	Trace of blood
2	Less than 50% filled
3	More than 50% filled
4	Completely filled and expanded
III and IV ventricles	
0	No blood
1	Blood present, size normal
2	Filled with blood and expanded

Graeb score is calculated by grading the lateral ventricles separately and adding that sum to scores for the third and fourth ventricles, which also are summed separately. The Graeb scale is most commonly reported scale in adults and correlates significantly with short-term outcome (Glasgow Outcome Score at 1 month).

3. LeRoux described another IVH scale (maximum score is 16).



Score	Each ventricle
0	No blood
1	Trace of blood
2	Less than half of the ventricle is filled with blood
3	More than half of the ventricle is filled with blood
4	The ventricle is filled with blood and expanded

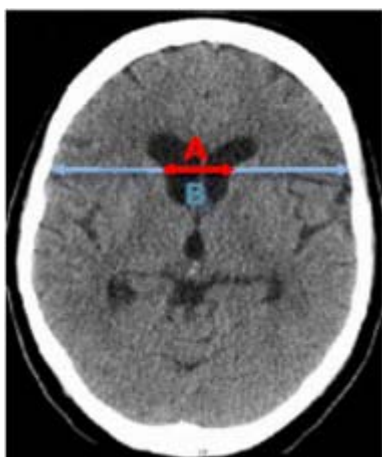
The total LeRoux score is a composite score grading each of the four ventricles separately.

5. Dinger described a hydrocephalus scale (maximum score is 24).

The score depends on the amount of ventricular dilatation. The more the ventricle is dilated, the higher the score. All individual scores for each ventricle are added up, providing a final score.

Score	0	1	2	3
Lateral Frontal horn	No dilatation	Mild	Moderate	Rounding, increasing radius, decreasing ventricular angle, and sulcal effacement of the lobe.
Atrium	No dilatation	Mild	Moderate	Rounding and enlargement with sulcal effacement of the parietooccipital lobe.
Temporal horn	No dilatation	Mild	Moderate	Increased width
3 rd ventricle	No dilatation	Mild	Moderate	Increased width and ballooning of the anterior recess.
IV th ventricle	No dilatation	Mild	Moderate	Ballooning

6. Determination of hydrocephalus after SAH.



Van Gijn presented measurements of hydrocephalus as the width of the frontal horns at the level of the caudate nuclei divided by the diameter of the brain at the same level, and called it the bicaudate index.

Bicaudate index (A/B), a simple and linear method for measuring the size of the ventricular system. A is the width of the frontal horns between the parallel walls of the caudate nuclei, at the level of the foramina of Monro, B is the diameter of the brain at the same level. The 95th percentile for the bicaudate index is 0.16 at age 30 years or under, 0.18 at 50 years, 0.19 at 60 years, 0.21 at 80 years, and 0.25 at 100 years.

Case 9

1. The proptosis is related to venous engorgement in the orbit (A). As the shunt is taken down either endovascularly or via surgery this is likely to resolve. The headaches are probably due to stretching of the retro-orbital dura by the markedly enlarged venous varices and should also improve.
2. This lesion has not bled and is in non-eloquent cortex. A very aggressive radiosurgical dose could be given and if the lesion did not thrombose by 3 years the lesion could certainly be re-treated. However, the true volume of

this AVM is really just over 10 ml making cure somewhat unlikely. Embolization and surgery would be reasonable alternative options.

3. Embolization has the advantage of decreasing the high flow through this malformation, thereby reducing the risk of post-resection perfusion pressure break-through. This phenomenon, felt to be due to shunting of blood into surrounding brain with disturbed autoregulatory capacity, is less common since the advent of staged NBCA embolization. Pre-operative embolization also renders the malformation more manipulable which will be helpful in mobilizing the malformation from its attachments along the sylvian fissure.

4.



A pterional craniotomy would suffice here. The sylvian fissure would then be split and the posterior lateral aspect of the malformation followed to the frontal horn of the ventricle. The malformation would then be mobilized from medial to lateral and the subfrontal feeders taken off the ACA. This would leave the malformation pedicled on the large lateral draining vein as it enters the sylvian fissure. This would be taken last. Usually, it is not necessary to divide the sylvian feeders directly off the MCA branches; instead these can be taken subpially in the frontal lobe, lessening the heat transfer onto the important en-passage vessels.

5. “Bag of black worms” (due to flow voids).

6. The average annual risk of rupture is 2 to 4%. One can use The equation $\text{Lifetime Risk} = 1 - (0.97)^{(\text{years left of life})}$ to calculate lifetime risk of AVM rupture. When the AVM ruptures, the risk of rerupture is ~ 6–7% that first year, then normalizes to 2–4% per subsequent year.

Case 10

1. The man has chemosis (conjunctival edema) and axial proptosis. The diagnosis is traumatic carotico-cavernous fistula.

2. There may be arterialized ('corkscrew') episcleral vessels, an orbital bruit, and sensory loss in the area of distribution of the ophthalmic division of the trigeminal nerve.
3. The risks to vision are from retinal ischemia due to reduced arterio-venous perfusion gradient, glaucoma due to raised episcleral venous pressure, and corneal exposure due to proptosis.
4. CT scanning or MRI will demonstrate the proptosis, enlarged extra-ocular muscles and superior ophthalmic vein, and possibly enlargement of the cavernous sinus. Other effects of the trauma may also be apparent, a basal skull fracture being particularly likely. The definitive investigation is carotid angiography. It is important to note that the clinical signs do not necessarily indicate the side of the fistula and four vessel angiography should be undertaken. In traumatic carotico-cavernous fistula, there is usually a large, direct fistula, i.e. communicating directly between the ICA and the cavernous sinus.