85 Aneurysms – Introduction, Grading, Special Conditions

85.1 Introduction and overview

85.1.1 Definitions

Subarachnoid hemorrhage: blood in the subarachnoid space (i.e., between the arachnoid membrane and the pia mater).

Aneurysm: (from the Greek aneurusma "dilatation"). An outpouching in the wall of an artery. May be focal (as in saccular AKA berry aneurysm) or fusiform. May be congenital or developmental. Etiologies:

1. congenital
2. developmental
   a) flow-related: generally at branch points of arteries, usually due to shear forces at these locations. Obsolete theory that this is due to underlying weakness of the media layer of the arterial wall at that location. Risk is increased in high flow states (e.g., chronic hypertension, drug-related e.g., cocaine, feeding vessels of AVMs…)
   b) mycotic (p. 1492): due to infection
   c) posttraumatic (p. 1491)
   d) conditions with abnormalities of blood vessels, including: autosomal dominant polycystic kidney disease (ADPKD) (p. 1455), vasculopathy (e.g., fibromuscular dysplasia (p. 209)), connective tissue disorders (Marfan syndrome (p. 1576), Ehlers-Danlos…)

Dissecting aneurysm (p. 1576): results from a tear in the arterial lining which allows blood to enter the arterial wall. Usually traumatically induced.
Pseudoaneurysm (p. 1576) (false aneurysm): a blood clot adjacent to a rent in the arterial wall.

85.1.2 Miscellaneous facts about SAH

1. may be posttraumatic or spontaneous. Trauma is the most common cause
2. most cases of spontaneous SAH are due to aneurysmal rupture
3. peak age for aneurysmal SAH (aSAH) is 55–60 yrs, ≈ 20% of cases occur between ages 15–45 yrs[1]
4. 30% of aSAHs occur during sleep
5. sentinel headaches that precede the aSAH-associated ictus have been reported by 10–50% of patients and most commonly occur within 2–8 weeks before overt SAH.[2,3,4]
6. headache is lateralized in 30%, most to the side of the aneurysm
7. SAH is complicated by:
   a) intracerebral hemorrhage in 20–40%
   b) intraventricular hemorrhage (p. 1454) in 13–28%
   c) subdural blood in 2–5%.
      When the subdural blood is over the convexity, it is usually due to PComA aneurysm.
      With an interhemispheric subdural hematoma, it is usually due to a distal anterior intracerebral artery (DACA) aneurysm (p. 1475)
8. soft evidence suggests that rupture incidence is higher in spring and autumn
9. patients ≥ 70 yrs of age have a higher proportion with a severe neurologic grade[5]
10. seizures may occur in up to 20% of patients after SAH, most commonly in the first 24 hours, and are associated with ICH, HTN, and aneurysm location (MCA & acomm)[6,7]

85.1.3 Outcome of aneurysmal SAH

1. 10–15% of patients die before reaching medical care
2. mortality is 10% within first few days
3. 30-day mortality rate was 46% in one series,[8] and in others over half the patients died within 2 weeks of their SAH[9]
4. median mortality rate in epidemiological studies from U.S. has been 32% vs. 44% in Europe and 27% in Japan (may be an underestimate based on underreported prehospital death)[10]
5. causes of mortality
   a) 25% die as a result of medical complications of SAH[11]

- neurogenic pulmonary edema (p. 1439)
- neurogenic stress cardiomyopathy (p. 1438) (AKA neurogenic stunned myocardium)

b) about 8% die from progressive deterioration from the initial hemorrhage[12 (p 27)]

6. among patients surviving the initial hemorrhage treated without surgery, rebleeding (p. 1437) is the major cause of morbidity and mortality. The risk is ≈ 15–20% within 2 weeks. The goal of early surgery (p. 1462) is to reduce this risk
7. of those reaching neurosurgical care, vasospasm (p. 1439) kills 7%, and causes severe deficit in another 7%[13]
8. about ≈ 30% of survivors have moderate to severe disability,[14] with rates of persistent dependence estimated between 8–20% in population-based studies[10]
9. ≈ 66% of those who have successful aneurysm clipping never return to the same quality of life as before the SAH[14,15]
10. patients ≥ 70 yrs of age fare worse for each neurologic grade.[5] A multivariate analysis revealed age and WFNS grade to be most predictive of long-term outcome, regardless of treatment modality[16]
11. the severity of clinical presentation is the strongest prognostic indicator

85.2 Etiologies of SAH

Etiologies of subarachnoid hemorrhage (SAH) include[17]:

1. trauma: the most common cause of SAH.[18,19] In all of the following discussion, only non-traumatic (i.e., "spontaneous") SAH will be considered
2. "spontaneous SAH"
   a) ruptured intracranial aneurysms (p. 1454): **75–80%** of spontaneous SAHs
   b) cerebral arteriovenous malformation (AVM): 4–5% of cases. AVMs more commonly cause ICH & IVH than SAH (p. 1505)
   c) certain vasculitides that involve the CNS, see Vasculitis and vasculopathy (p. 203)
   d) rarely due to tumor (many case reports[20,21,22,23,24,25,26,27,28,29,30,31])
   e) cerebral artery dissection (may also be posttraumatic)
      - carotid artery (p. 1578)
      - vertebral artery (p. 1579): may cause intraventricular blood (especially 4th and 3rd ventricle)
   f) rupture of a small superficial artery
   g) rupture of an infundibulum (p. 1423)
   h) coagulation disorders:
      - iatrogenic or bleeding dyscrasias
      - thrombocytopenia
   i) dural sinus thrombosis
   j) spinal AVM (p. 1395): usually cervical or upper thoracic
   k) cortical subarachnoid hemorrhage
   l) pretruncal nonaneurysmal SAH (p. 1496) (perimesencephalic hemorrhage)
   m) rarely reported with some drugs: e.g., cocaine (p. 215)
   n) sickle cell disease
   o) pituitary apoplexy (p. 865)
   p) no cause can be determined in 14–22% (p. 1494)

85.3 Incidence of aneurysmal SAH (aSAH)

Estimated annual rate of aSAH in the United States: 9.7–14.5 per 100,000 population.[32,33] Reported rates are lower in South and Central America,[34] and higher in Japan and Finland.[35] Incidence of SAH increases with age (avg. age of onset > 50[33,36,37,38]); tends to be higher in women (1.24 times higher than men),[34] and appears to be higher in African Americans and Hispanics (compared to Caucasians).[32,39,40]

85.4 Risk factors for aSAH

See references.[17,41]

1. behavioral
   - hypertension
   - cigarette smoking[42]
   - alcohol abuse

- sympathomimetic drugs such as cocaine (p. 215), amphetamines (including "crystal meth")
- exercise/sports: weight training,[43] especially when performed with valsalva maneuver, carries a low risk of precipitating bleeding from a pre-existing aneurysm

2. gender and race (see above)
3. history of cerebral aneurysm
   - ruptured aneurysm
   - unruptured aneurysm (esp. those that are symptomatic, larger in size, and located in posterior circulation)
   - morphology: bottleneck shape[44] and increased ratio of size of aneurysm to parent vessel have been associated with increased risk of rupture[45,46]
4. family history of aneurysms (at least 1 first-degree family member and especially if ≥ 2 are affected)
5. genetic syndromes
   - autosomal dominant polycystic kidney disease (p. 1455)
   - type IV Ehlers-Danlos syndrome
6. pregnancy: controversial. Studies have found evidence for increased risk while others have not (p. 1425)

85.5 Clinical features

85.5.1 Symptoms of SAH

Sudden onset of severe H/A (see below), usually with vomiting, syncope (apoplexy), neck pain (meningismus), and photophobia. If there is LOC, patient may subsequently recover consciousness.[47] Focal cranial nerve deficits may occur (e.g., third nerve palsy from aneurysmal compression of the third cranial nerve, causing diplopia and/or ptosis). Low back pain may develop due to irritation of lumbar nerve roots by dependent blood.

85.5.2 Headache

The most common symptom, present in up to 97% of cases. Usually severe (classic description: "the worst headache of my life") and sudden in onset (paroxysmal). The H/A may clear and the patient may not seek medical attention (referred to as a **sentinel hemorrhage** or headache, or warning headache; they occur in 30–60% of patients presenting with SAH). If severe or accompanied by reduced level of consciousness, most patients present for medical evaluation. Patients with H/A due to minor hemorrhages will have blood on CT or LP. However, warning headaches may also occur without SAH and may be due to aneurysmal enlargement or to hemorrhage confined within the aneurysmal wall.[48] Warning H/A are usually sudden in onset, milder than that associated with a major rupture, and may last a few days.

Differential diagnosis of severe, acute, paroxysmal headache (25% will have SAH[49]):

1. subarachnoid hemorrhage: including "warning headache" or sentinel H/A (see above)
2. benign "thunderclap headaches" (BTH) or crash migraine.[50] Severe global headaches of abrupt onset that reach maximal intensity in < 1 minute, accompanied by vomiting in ≈ 50%. They may recur, and are presumably a form of vascular headache. Some may have transient focal symptoms. There are no clinical criteria that can reliably differentiate these from SAH[51] (although seizures and diplopia, when they occurred, were always associated with SAH). There is no subarachnoid blood on CT or LP (CT and/or CTA should probably be performed on at least the first presentation to R/O SAH). Earlier recommendations to angiogram these individuals[52] have since been tempered by experience[53,54]
3. reversible cerebral vasoconstrictive syndrome (RCVS)[55] (AKA benign cerebral angiopathy or vasculitis[56]): severe H/A with paroxysmal onset, ± neurologic deficit, and string-of-beads appearance on angiography of cerebral vessels that usually clears in 1–3 months. More than 50% report prior use of vasoconstrictive substances (cocaine, marijuana, nasal decongestants, ergot derivatives, SSRIs, interferon, nicotine patches) sometimes combined with binge drinking. May also occur post-partum. Complications occurred in 24% including:
   a) usually during the 1st week: SAH, ICH, seizures, RPLS
   b) usually during the 2nd week: ischemic events (TIA, stroke)
4. airplane headache: usually sudden, often (but not exclusively) with onset during take-off (less common) or landing of aircraft. Short-lasting (by definition: ≤ 30 minutes after completion of ascent or descent[57]; however, in one series 76% of H/A otherwise typical for airplane H/A lasted > 30 minutes[58]), usually unilateral, primarily orbitofrontal (occasionally with spread to parietal

region). Typically jabbing or stabbing in quality. Ipsilateral nasal congestion, a stuffy feeling of the face, or tearing may occur in < 5%.[57] Pathogenesis may be related to obstructed drainage of sinuses ("aerosinusitis" or barosinusitis); however, a vascular mechanism may be possible. H/A may respond to triptans (19%) or acetaminophen (5%).[58]

5. **benign orgasmic cephalgia**: a severe, throbbing, sometimes "explosive" H/A with onset just before or at the time of orgasm (distinct from pre-orgasmic headaches which intensify with sexual arousal[59]). In a series of 21 patients[60] neurologic exam was normal in all, and angiography done in 9 was normal. 9 had a history of migraine in the patient or a family member. No other symptoms developed in 18 patients followed for 2–7 yrs. Recommendations for evaluation are similar to that for thunderclap headaches above

85.5.3 Signs

Meningismus (see below), hypertension, focal neurologic deficit (e.g., oculomotor palsy, hemiparesis), obtundation or coma (see below), ocular hemorrhage (see below).

Meningismus

Nuchal rigidity (especially to flexion) often ensues in 6 to 24 hrs. Patients may have a positive Kernig sign (flex thigh to 90° with knee bent, then straighten knee, positive sign if this causes pain in hamstrings) or Brudzinski sign (flex the supine patient's neck, involuntary hip flexion is a positive sign).

Coma following SAH

Coma may follow SAH because of any one or a combination of the following[61]:

1. increased ICP
2. damage to brain tissue from intraparenchymal hemorrhage (may also contribute to increased ICP)
3. hydrocephalus
4. diffuse ischemia (may be secondary to increased ICP)
5. seizure
6. reduced CBF (p. 1438) low blood flow (e.g., due to reduced cardiac output)

Ocular hemorrhage

Three types of ocular hemorrhage (OH) may be associated with SAH. They occur alone or in various combinations in 20–40% of patients with SAH.[62]

1. subhyaloid (preretinal) hemorrhage: seen funduscopically in 11–33% of cases as bright red blood near the optic disc that obscures the underlying retinal vessels. May be associated with a higher mortality rate[63]
2. (intra)retinal hemorrhage: may surround the fovea
3. hemorrhage within the vitreous humor (Terson syndrome). First described by the French ophthalmologist Albert Terson. Occurs in 4–27% of cases of aneurysmal SAH,[64,65,66] usually bilateral. May occur with other causes of increased ICP including ruptured AVMs. Funduscopy reveals vitreous opacity. The location of the origin of the vitreous hemorrhage differs in various reports (subhyaloid, epiretinal, subinternal limiting membrane).[67] May be more common with anterior circulation aneurysms (especially ACoA), although 1 study found no correlation with location.[65] Also rarely reported with SDH and traumatic SAH. Often missed on initial examination. When sought, usually present on initial exam; however, it may develop as late as 12 days post SAH, and may be associated with rebleeding.[65] The mortality rate may be higher in SAH patients with vitreous hemorrhage than in those without. Patients should be followed for complications of OH (elevated intraocular pressure, retinal membrane formation → retinal detachment, retinal folds[68]). Most cases clear spontaneously in 6–12 mos. Vitrectomy should be considered in patients whose vision fails to improve[66] or if more rapid improvement is desired.[69] The long-term prognosis for vision is good in ≈ 80% of cases with or without vitrectomy[69]

The pathomechanics of OH are controversial. OH was originally attributed to extension of the blood from the subarachnoid space into the vitreous, but no communication exists between these two spaces. In actuality may be due to compression of the central retinal vein and the retinochoroidal anastomoses by elevated CSF pressure,[66] causing venous hypertension and disruption of retinal veins.

85.6 Work-up of suspected SAH

85.6.1 Overview

1. tests to diagnose SAH
   a) non-contrast high-resolution CT scan: very sensitive and specific (see below)
   b) if CT is negative: LP in suspicious cases. Very sensitive, but only 65–80% specific (see below)
   c) with CT, the concern is a false negative test (missing a SAH), and with LP the concern is a false positive (a bloody tap mimicking a SAH). However, the combination of a negative CT and a negative LP is extremely strong in ruling out SAH[70]
2. tests to identify *source* of SAH. Options: CTA, MRA, or catheter angiography. The choice needs to take into account the patient's age, renal function, and even best guess of where an aneurysm might be located
   a) MRA (p. 243). Pros: no radiation, and 2D-TOF MRA does not use contrast. Cons: poor sensitivity for aneurysm detection early after SAH (see below)
   b) CTA vs. digital subtraction catheter angiogram (DSA): one needs to balance the risk of the procedure and ease of obtaining it against the information expected to be obtained
      - total iodine load in a healthy adult should be < 90 gm in 24 hours. In older patients and/or possible compromised renal function, this volume should be less. CTA typically uses 65–75 cc of contrast with ≈ 300 mg iodine/ml, or ≈ 21 gm iodine. The amount of contrast with a cerebral arteriogram varies. However, if an angiogram is needed after a CTA, in most cases you do not have to wait 24 hours
      - if there is concern about renal function (e.g., serum creatinine > 100 mcmol/L) hydrate the patient and optionally give Mucomyst® (p. 232)
      - catheter angiography (DSA) may be necessary after a positive CTA to better delineate the anatomy, or to determine dominant filling and cross flow, or in highly suspicious cases with a negative CTA (see below). While CTA permits reliable assessment of feasibility of endovascular treatment in most cases,[71] DSA is still necessary in some
3. if CTA/angiogram is negative: see SAH of unknown etiology (p. 1494)

85.6.2 Laboratory/radiographic findings

CT scan

► **Sensitivity and specificity.** High-quality non-contrast CT (no motion artifact, 3rd generation or newer high-resolution CT scanner) is very sensitive to intracranial SAH. Sensitivity decreases with time as the blood dissipates.

- within 6 hrs of SAH: sensitivity is 98–100%, specificity 100%, negative predictive value 99.4%, positive predictive value 100% (in 240 adults with new acute H/A peaking in ≤ 1 hr[72])
- sensitivity of CT < 12 hrs after SAH: ≈ 98%
- after 12 hrs, the sensitivity is too low to rely on noncontrast CT alone to exclude the potentially lethal diagnosis of ruptured aneurysm. But it can be helpful if positive
  - sensitivity of CT < 24 hrs of SAH: ≈ 93%
  - CT < 72 hrs of SAH: ≈ 80%
  - CT 1 week after SAH: ≈ 50%

► **Findings.** Blood appears as high density (white) within subarachnoid spaces (► Fig. 85.1). Subtle hints for SAH: look for blood in the occipital horns of the lateral ventricles and the dependent portions of the Sylvian fissures.

CT also assesses:

1. ventricular size: acute hydrocephalus after aneurysmal rupture (p. 1426) occurs in 21%[73]
2. hematoma: intracerebral hemorrhage or large amount of subdural blood with mass effect may need emergent evacuation (most common with MCA aneurysms)
3. amount of blood in cisterns and fissures: important prognosticator for vasospasm (p. 1441) and can identify pretruncal nonaneurysmal hemorrhage (p. 1496)
4. CT can predict aneurysm location based on the pattern of blood in ≈ 78% of cases (but mostly for MCA and AComA aneurysms)[74]
   a) blood predominantly in anterior interhemispheric fissure (± blood in lateral ventricles) (► Fig. 88.1) or within the gyrus rectus suggests AComA aneurysm
   b) blood predominantly in 1 Sylvian fissure is compatible with PComA or MCA aneurysm on that side (► Fig. 85.1)
   c) blood predominantly in the prepontine or peduncular cistern suggests a basilar apex or SCA aneurysm

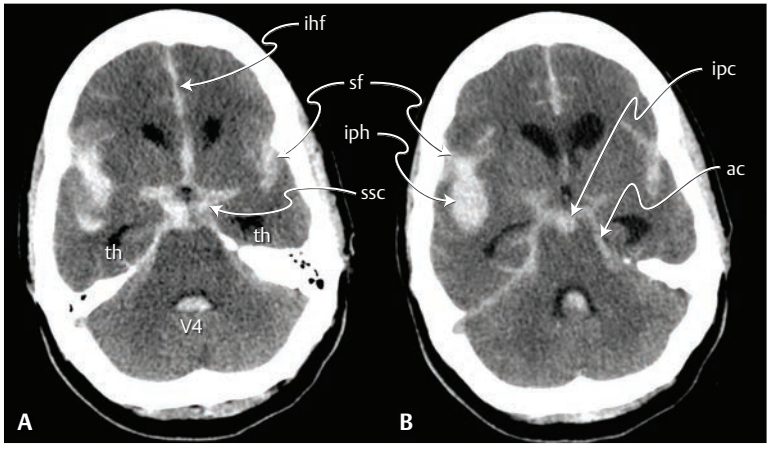

Fig. 85.1 SAH on CT. Image: axial CT scan in patient with SAH (and intraparenchymal hematoma) from a ruptured right middle cerebral artery aneurysm.
Image: A: CT slice through the suprasellar cistern (ssc) showing a classic SAH pattern, with blood in the ssc, interhemispheric fissure (ihf) & Sylvian fissures (sf).
B: CT slice slightly above the ssc showing SAH in the interpeduncular cistern (ipc), the ambient cisterns (ac) as well as intraparenchymal hematoma (iph) from the aneurysmal rupture.
Note the dilated temporal horns (th) suggesting early hydrocephalus and blood in the 4th ventricle (V4).

   d) blood predominantly within ventricles (p. 1454)
      - blood primarily in 4th and 3rd ventricle (▶ Fig. 88.2): suggests lower posterior fossa source, such as PICA aneurysm (p. 1480) or VA dissection
      - blood primarily in the 3rd ventricle suggests a basilar apex aneurysm
5. with multiple aneurysms, CT may help identify which one bled by the location of blood (see above). See also other "clues" (p. 1490)
6. ✖ CT is not sensitive for infarct in the first 24 hours after infarct (see CT scan with acute ischemic infarct (p. 1559))

▶ **Differential diagnosis of SAH on CT.** Things that can mimic the appearance of SAH on CT include:
1. pus: as in meningitis
2. following contrast administration: sometimes IV, and especially intrathecal
3. occasionally the pachymeningeal thickening seen in spontaneous intracranial hypotension (p. 421)

CT angiography (CTA)

CTA (p. 238), a 64-slice CTA is 98% sensitive and 100% specific for detecting aneurysms > 3 mm diameter,[75] and in a prospective study detected 97% of aneurysms, and was deemed as safe and effective when used as the initial and sole imaging study for ruptured and unruptured cerebral aneurysms.[76] CTA shows a 3-dimensional image (as can modern catheter angiography), which can help differentiate adherent vessels from those arising from the aneurysm. CTA also demonstrates the relation to nearby bony structures which can be important in surgical planning. CTA may also be used for evaluation of vasospasm.[77]

Lumbar puncture

The most sensitive test for SAH, approaching 100%, with a negative predictive value of 100% for SAH. However, false positives—e.g., with traumatic taps (Differentiating SAH from traumatic tap (p. 1814))—occur with enough frequency that the specificity may be in the range of 80%[78] and possibly as low as 65%.[75] Skipping the LP and going right to CTA is controversial with arguments both for[79] and against.[80] The LP is more helpful to *rule-out* SAH if CSF has no blood.

✖ Caution: lowering the CSF pressure with an LP may possibly precipitate rebleeding by increasing the transmural pressure (p. 1427) (the pressure across the aneurysm wall). Therefore remove only a small amount of CSF (several ml) and use a small (≤ 20 Ga) spinal needle.

Findings (also, see ▶ Table 23.4):

1. opening pressure: usually elevated with SAH
2. appearance:
   a) non-clotting bloody fluid that does not clear with sequential collection tubes
   b) xanthochromia (XTC): yellow coloration (▶ Fig. 85.2) of CSF supernatant due to the lysis of RBCs which releases heme pigments that break down to bilirubin. XTC is the most reliable means of differentiating traumatic tap from SAH in patients with a negative head CT. The minimum amount of time required for bilirubin to become detectable in the CSF, as well as the minimum amount of blood that needs to enter the CSF to produce XTC remains unknown. XTC is usually not apparent until 2–4 hours after the SAH. It is present in almost 100% by 12 hours after the bleed, and remains in 70% at 3 weeks, and is still detectable in 40% at 4 weeks. False positives: XTC may occur with jaundice or high protein levels in the CSF.
   Very bloody specimens may need to be centrifuged in the lab to be able to look for XTC. Spectrophotometry is more sensitive than visual inspection, but may not be specific enough to warrant widespread use[81,82]
3. cell count: RBC count is usually > 100,000 RBCs/mm^3 in SAH
4. compare RBC count in first to last tube: a reduction of RBC count from the first tube to the last tube of 70% with < 500 RBC/mm^3 in the final tube has been suggested to be diagnostic of a traumatic tap[83] (controversial (p. 1814))
5. protein: elevated due to blood breakdown products
6. glucose: normal or reduced (RBCs may metabolize some glucose with time)

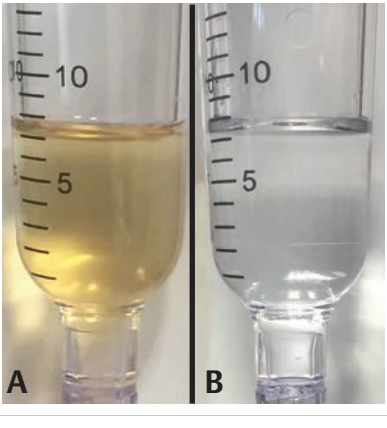

Fig. 85.2 Xanthochromic CSF in A, compared to normal CSF in B.

MRI

Not sensitive for SAH acutely within the first 24–48 hrs[84] (too little met-Hb), especially with thin layers of blood. Better after ≈ 4–7 days (excellent for subacute to remote SAH, > 10–20 days). FLAIR MRI is the most sensitive imaging study for detecting blood in the subarachnoid space. May be helpful in determining which of multiple aneurysms bled (p. 1490).[85]

Magnetic resonance angiography (MRA)

Compared to catheter DSA, sensitivity is 87% and specificity is 92% for detecting intracranial aneurysms (IAs). Sensitivity is significantly worse for aneurysms < 3 mm diameter.[86,87,88]

MRA's ability to detect IAs depends on aneurysm size, rate and direction of blood flow in the aneurysm relative to the magnetic field, and aneurysmal thrombosis and calcification. MRA may be most useful as a screening test in high-risk patients, including patients with two first-degree relatives with IAs, especially those who are also smokers or hypertensive themselves.[89]

Catheter angiogram

General information

Injection of radio-opaque (iodinated) contrast ("dye") into selective vessels using a catheter typically inserted into the femoral artery at the upper thigh, while taking serial X-rays to obtain a "video-like" representation of the vasculature.

The gold standard for evaluation of cerebral aneurysms. Current state of the art uses digital subtraction angiography (DSA). Demonstrates source (usually aneurysm) in ≈ 80–85%; remainder are so-called "SAH of unknown etiology" (p. 1494). Shows if radiographic vasospasm is present— **clinical** vasospasm (p. 1439) almost never occurs < 3 days following SAH—and assesses primary feeding arteries, collateral flow in case of a need for arterial sacrifice.

General principles:

1. study the vessel of highest suspicion first (in case patient's condition should change, necessitating discontinuation of procedure)
2. continue to do complete 4-vessel angiogram (even if aneurysm(s) have been demonstrated) to rule out additional aneurysms and assess collateral circulation
3. if there is an aneurysm or suspicion of one, obtain additional views to help delineate the neck and orientation of the aneurysm (see index for specific aneurysm)
4. ★ if no aneurysm is seen, before an arteriogram can be considered negative, must:
   a) visualize *both PICA origins*: 1–2% of aneurysms occur at PICA origin. Both PICAs can usually be visualized with one VA injection if there is enough flow to reflux down the contralateral VA. Occasionally it is necessary to see more of the contralateral VA than what refluxes to PICA and selective catheterization may be required
   b) *flow contrast through the ACoA*: if both ACAs fill from one side, this is usually satisfactory. It may be necessary to perform a cross compression AP study with carotid injection (first, rule out plaque in the carotid to be compressed), or use a higher injection rate to facilitate flow through the ACoA
   c) if an infundibulum (see below) co-localizes to the SAH, it may be unwise to label the case as angiogram-negative, and exploration is recommended by some[90]

Infundibulum

A funnel-shaped initial segment of an artery, to be distinguished from an aneurysm. Found in 7–13% of otherwise normal arteriograms,[91,92] with a higher incidence in cases of multiple or familial aneurysms. Bilateral in 25%.[92] Most commonly found at the origin of the PComAs, but they rarely occur at other sites. Criteria for differentiating infundibula from aneurysms are shown in ▶ Table 85.1. Infundibula may represent incomplete remnants of previous fetal vessels.[93 (p 272)]

Although they may bleed,[90,95,96,97] there is less risk of rupture than with a saccular aneurysm (no infundibulum < 3 mm in size bled[98] in the cooperative study). However, infundibula have been documented to progress to an aneurysm (i.e., they are preaneurysmal) which may bleed (13 case reports in the literature as of 2009). Recommended treatment: at the time of surgery for another reason, consider treating an infundibulum with wrapping, or placing in an encircling clip, or sacrificing the artery if it can be done safely (infundibula lack a true neck).

Table 85.1 Criteria of an infundibulum

1. triangular in shape
2. mouth (widest portion) < 3 mm[a] [94]
3. vessel at apex

[a] a widely accepted but probably arbitrary dimension

Angiographic findings

1. general features to take note of when analyzing an aneurysm on angiogram (special considerations for specific aneurysms are covered in designated sections)
   a) size of aneurysm dome:
      - MRI or CT helps with this since the aneurysm may be partially thrombosed and the portion that is patent and fills with contrast and is therefore visualized on angiogram may be much smaller than the actual size
      - large aneurysms (≥ 15 mm dia.) are associated with lower rates of complete occlusion by endovascular coiling[99,100]
   b) neck size
      - narrow necks < 5 mm are ideal for coiling[101]

- broad necks ≥ 5 mm are associated with increased risk of incomplete occlusion and recanalization with coiling[100]
- stent or balloon-assisted coiling may be needed for wide-necked aneurysms. Stents should be avoided if possible (p. 1923)

c) dome:neck ratio ≥ 2 is associated with higher rate of successful coil occlusion[101]

2. for basilar bifurcation aneurysms (p. 1482)

85.7 Grading SAH

85.7.1 General information

Four grading scales are in common use. The two most widely quoted grading scales, the Hunt-and-Hess and the WFNS, are presented below.

85.7.2 Hunt and Hess grade

See ▶ Table 85.2 and ▶ Table 85.3 for grading system. Grades 1 and 2 were operated upon as soon as an aneurysm was diagnosed. Grade ≥ 3 managed until the condition improved to Grade 2 or 1. Exception: life-threatening hematoma or multiple bleeds (which were operated on regardless of grade).

Analysis of data from the International Cooperative Aneurysm Study revealed that with normal consciousness, Hunt and Hess (H&H) grades 1 and 2 had identical outcome, and that hemiparesis and/or aphasia had no effect on mortality.

Mortality:
Admission Hunt and Hess Grade 1 or 2: 20%.
Patients taken to O.R. (for any procedure) at H&H Grade 1 or 2: 14%.
Major cause of death in Grade 1 or 2 is rebleed.
Signs of meningeal irritation increases surgical risk.

Table 85.2 Hunt and Hess classification[a] of SAH[102]

Grade	Description
1	asymptomatic, or mild H/A and slight nuchal rigidity
2	Cr. N. palsy (e.g., III, VI), moderate to severe H/A, nuchal rigidity
3	mild focal deficit, lethargy, or confusion
4	stupor, moderate to severe hemiparesis, early decerebrate rigidity
5	deep coma, decerebrate rigidity, moribund appearance
Add one grade for serious systemic disease (e.g., HTN, DM, severe atherosclerosis, COPD) or severe vasospasm on arteriography.	
[a]original paper did not consider patient's age, site of aneurysm, or time since bleed; patients were graded on admission and pre-op	

Table 85.3 Modified classification[103] adds the following

Grade	Description
0	unruptured aneurysm
1 a	no acute meningeal/brain reaction, but with fixed neuro deficit

85.7.3 World Federation of Neurosurgical Societies / World Federation of Neurological Surgeons (WFNS) grading of SAH

Due to lack of data on the significance of features such as headache, nuchal rigidity, and major focal neurologic deficit, the WFNS Committee on a Universal SAH Grading Scale[104,105] developed the grading system shown in ▶ Table 85.4. It employs the Glasgow Coma Scale (▶ Table 18.1) (GCS) to grade the level of consciousness, and uses the presence or absence of major focal neurologic deficit to distinguish grade 2 from grade 3.

Table 85.4 WFNS SAH grade[104]

WFNS grade	GCS score[a]	Major focal deficit[b]
0[c]		
1	15	–
2	13–14	–
3	13–14	+
4	7–12	+ or –
5	3–6	+ or –

[a]GCS = Glasgow Coma Scale, see ▶ Table 18.1
[b]aphasia, hemiparesis or hemiplegia (+ = present, – = absent)
[c]intact (unruptured) aneurysm

85.8 Pregnancy and intracranial hemorrhage

85.8.1 General information

Intracranial hemorrhage (subarachnoid or intraparenchymal) is a rare occurrence during pregnancy (estimated range of incidence: 0.01–0.05% of all pregnancies[106]) and yet is responsible for 5–12% of maternal deaths during pregnancy.

Intracranial hemorrhage of pregnancy (ICHOP) commonly occurs in the setting of eclampsia, and is more commonly intraparenchymal[107] and may be associated with loss of cerebrovascular autoregulation PRES (p. 202).[108] HELLP syndrome (hemolysis, elevated liver enzymes, and low platelet count) is a severe variant of pre-eclampsia.[109] Symptoms of eclampsia with or without ICHOP include H/A, mental status changes, and seizures.

It has been asserted that risk of aneurysmal SAH does not appear to be increased in pregnancy, delivery, and puerperium.[110,111] A literature review of 154 reported cases of ICHOP-related SAH revealed that 77% were aneurysmal and 23% were from ruptured AVM (other series show the percentage of AVMs range from 21–48%). Mortality is ≈ 35% for aneurysmal and ≈ 28% for AVM hemorrhage (the latter being higher than in non-gravid patients). There is an increasing tendency for bleeding with advancing gestational age for both aneurysms and AVMs (earlier it had been asserted that this held true for aneurysms only[112]).

Patients with ICHOP having AVMs tend to be younger than those with aneurysm, paralleling the occurrence in the general population. One major oft-quoted study showed an increased risk of hemorrhage from AVMs during pregnancy[113] (citing an 87% hemorrhage rate); however, another investigation disputes this assertion,[114] and found the risk of hemorrhage to be 3.5% during the pregnancy in patients with no history of hemorrhage, or 5.8% in those with previous hemorrhage. Another study evaluated risk of aneurysm rupture during pregnancy and delivery from the Nationwide Inpatient data and calculated the rupture risk during pregnancy and delivery to be 1.4% and 0.05%, respectively.[115] Literature review[106] found that the risk of recurrent hemorrhage following ICHOP from aneurysm or AVM during the remainder of the pregnancy was 33–50%.

85.8.2 Management modifications for pregnant patients

Modifications of evaluation and treatment techniques may be necessary for the pregnant patient.

1. neuroradiologic studies
   a) CAT scan: with shielding of the fetus, CAT scanning of the brain produces minimal radiation exposure to the child
   b) MRI:
      - generally felt to have low potential for complications; however, many centers will not do MRI during first trimester.
      - gadolinium-based contrast agents (GBCAs) are teratogenic in animals in high repeated doses. It has not been studied in human pregnancy. A cohort of 26 women who received GBCAs during the first trimester showed no evidence of teratogenicity or mutagenicity.[116] There have also been no reported issues related to nephrogenic systemic fibrosis (p. 243). GBCAs are FDA Class C drugs—not recommended for use during pregnancy, but may be used if benefits outweigh potential risks.
   c) angiography: with shielding of the fetus, radiation exposure is minimal. Iodinated contrast agents pose little risk to the fetus. The mother should be well hydrated during and after the study[106]

2. antiseizure medications: see Pregnancy and antiseizure medications (p. 500)
3. diuretics: the use of mannitol in pregnancy should be avoided to prevent fetal dehydration and maternal hypovolemia with uterine hypoperfusion
4. antihypertensives: nitroprusside should not be used in pregnancy
5. nimodipine is potentially teratogenic in animals, the effect on humans is unknown. It should be used only when the potential benefit justifies the risk

85.8.3 Neurosurgical management

The currently recommended treatment of a ruptured aneurysm in the pregnant patient is immediate surgical treatment to avoid rebleeding and ischemic complications due to vasospasm. A meta-analysis has demonstrated that mother and fetus both benefit from surgical treatment—with maternal mortality decreasing from 63% to 11% and fetal mortality decreasing from 27% to 5%.[106,117] Successful endovascular treatment for aSAH has been reported, but fetal exposure to radiation is a concern. The absorbed fetal dose has been estimated to range from 0.17 to 2.8 mGy, corresponding to a fetal risk of a hereditary disease at birth and a cumulative risk for a fatal cancer by age 15, which are both substantially lower than those which naturally occur.[118] Because endovascular treatment requires heparin for systemic anticoagulation, it carries the risk of hemorrhagic implications when labor spontaneously begins during or around the time of embolization.

85.8.4 Obstetric management following ICHOP

Several reports have indicated that the fetal and maternal outcome is no different for vaginal delivery vs. C-section, and is probably more dependent on whether the offending lesion has been treated. However, there are no formal studies to help guide the optimal treatment of pregnant women with aSAH. One strategy[117] is to perform an emergent C-section, followed by aneurysm treatment, if the fetus is mature enough for survival outside the uterus. If the fetus is < 24 weeks, treat the aneurysm and maintain the pregnancy. If the fetus is between 24–28 weeks, a strategy should be tailored according to the maternal and fetal status. C-section may be used for fetal salvage for a moribund mother in the third trimester. During vaginal delivery, the risk of rebleeding may be reduced by the use of caudal or epidural anesthesia, shortening the second stage of labor, and low forceps delivery if necessary.

85.9 Hydrocephalus after SAH

85.9.1 Hydrocephalus after traumatic SAH

See also posttraumatic hydrocephalus (p. 1108).

85.9.2 Acute hydrocephalus

General information

The frequency of hydrocephalus (HCP) on the initial CT after SAH depends on the defining criteria used, with a reported range of 9–67%.[119] A realistic range is ≈ 15–20% of SAH patients, with 30–60% of these showing no impairment of consciousness.[119,120] 3% of those *without* HCP on initial CT develop HCP within 1 week.[119]

Factors felt to contribute to acute HCP include blood interfering with CSF flow through the Sylvian aqueduct, 4th ventricle outlet, or subarachnoid space, and/or with reabsorption at the arachnoid granulations.

Findings associated with acute HCP include[120]

1. increasing age
2. admission CT findings: intraventricular blood, diffuse subarachnoid blood, and thick focal accumulation of subarachnoid blood (intraparenchymal blood did *not* correlate with chronic HCP, and patients with a normal CT had a low incidence)
3. hypertension: on admission, prior to admission (by history), or post-op
4. by location:
   a) posterior circulation aneurysms have a higher incidence of HCP
   b) MCA aneurysms correlate with low incidence of HCP
5. miscellaneous: hyponatremia, patients who were not alert on admission, use of preoperative antifibrinolytic agents, and low Glasgow outcome score