Subdural Hematoma

EPIDEMIOLOGY
- **men**: women = 3:1
- most patients > 70 yrs.
- more common than EDH.

PATHOLOGY, PATHOGENESIS
- **Location**:
  - subdural space – run from cortical surface to dural sinus; commonly found along sagittal sinus and around anterior tip of temporal lobe.
  - rarely, bleeding source may be **cortical arteries** or **oozing brain laceration**.

ETIOLOGY
1. Vigorous head motion (acceleration-deceleration injury?) – may be trivial!
2. Spontaneous (30% of chronic SDH, i.e. intrinsic susceptibility – prone to recur)
   - 1) congophilathes
   - 2) (cerebral) CCF (laceration causing ventricular decompression (→ stretching of bridging veins) and low ICP (intracranial venous congestion – seen on MRI as meningeal enhancement)
   - 3) intracerebral hemorrhage, ruptured intracranial aneurysm (blood may dissect into subdural space, esp. PComA → convexial SDH; distal ACA → parafrontal SDH)
   - 4) intermittent bleeding from **dural AVF** (recurrent subdural hematomas)
   - 5) bleeding from intracranial tumors
   - *e.g. falls & assaults (~72% SDH cases!); vehicular trauma (only ≥ 24% - automobile absorbs some of energy - so deceleration rate is less)*.

   **SDH is not usually associated with skull fractures!!**, direct impact is not necessary!

   *If skull fracture is present, it is commonly contralateral to SDH

PATIENT CLASSIFICATION
- movement of brain relative to skull = trauma (via shearing mechanism) of subarachnoid vessels (CSA);
  - subdural space – run from cortical surface to dural sinus; commonly found along sagittal sinus and around anterior tip of temporal lobe.
  - rarely, bleeding source may be **cortical arteries** or **oozing brain laceration**.

   **N.B. bleeding is most commonly venous (e.g. EDH – arterial)**

   - as hematoma expands in subdural space, it raises ICP (↔ global ischemia) and compresses brain (↔ regional ischemia ↔ herniation).
   - brain atrophy (e.g. elderly, chronic alcoholism, dementia) predisposes to SDH even after minor trauma;
   - brain has additional space for movement, bridging veins are stretched, atrophic brain cannot tamponade beginning hematoma; SDH may reach > 100 mL before becoming symptomatic!

   **LOCATION**
   - 1) along cerebral convexities - most common! (most often frontotemporal).
   - 2) along interhemispheric fissure and tentorium (often associated with shaken baby syndrome).
   - *if between occipital lobe and tentorium
   - 3) posterior fossa (< 1%) - cerebellum undergoes little movement; most SDHs here are result of parenchymal cerebellar injury – posterior fossa SDHs have highest mortality!

   - subdural space (unlike epidural space) is not confined by cranial sutures and has no adhesions – SDH rapidly spreads along entire hemisphere and into hemispheric fissure, limited only by dural reflections at midline / tentorium.

   - bilateral SDHs (~10%) are more common in infants - adhesions in subdural space are absent at birth and develop with aging.

   **CLASSIFICATION**
   - **Acute SDH** manifests during first 72 hours:
     - most common type of traumatic intracranial hematoma (5-30% of severe head injuries; ~1% of mild head injuries)
     - commonly (~50%) associated with **extensive primary brain trauma**!!! (vs. EDH - diffuse parenchymal injury, contusions, lacerations, intracerebral hematomas - play major role in outcome!!!)
     - more common in elderly and in infants (both have larger subarachnoid space - allows for more movement between brain and dura).

     - **mortality**: simple SDH (if no other brain injury) = 20%; complicated SDH (e.g. with contusions) = 60%.
     - GCS 12-15 = 8%; GCS 3-5 = 76%.

     - **Subacute SDH** manifests within 1-20 days (surgical literature favors > 3 days; radiological literature favors > 7).
     - **Chronic SDH** manifests within > 20 days old.
       - most common after age 50 with apparently insignificant head trauma.
• most are derived from subdural hygroma; minority develop from untreated acute SDH.
• commonly associated with cerebral atrophy.
• risk factors for chronic SDH - elderly with cerebral atrophy, chronic alcoholism, epilepsy, bleeding disorders, arachnoid cysts, cardiovascular disease (hypertension, arteriosclerosis).
• 8.7-32% are bilateral.
• mortality ≈ 5-10%.
• small SDHs often spontaneously resorb; larger SDHs liquify (in ≈ 1 week) and form encapsulating vascular membranes (fibroblasts grow from dural surface and form thicker outer membrane by about 7 days, and thinner inner membrane after 2-3 weeks), rarely calcifies.
  - blood in chronic SDH has liquid consistency, typically resembling crank case oil (can be drained through burr holes!).
  - membrane consists of many fragile capillaries, intact and lysed RBCs, hemosiderin-laden macrophages, and granulation tissue.
  - organized hematoma is firmly attached by fibrous tissue to dura and is not at all adherent to arachnoid (arachnoid does not contribute to membrane formation).
  - at some point, critical mass is reached (hematoma assumes biconvex shape and becomes symptomatic):
    a) bleeding into chronic SDH (small recurrent hemorrhages from thin-walled vessels within membrane due to repeated minor trauma); up to 45% chronic SDHs rebleed; risk of rebleeding is greatest in first few months.
    b) osmotic swelling – due to blood break down and increased protein content (draws water osmotically across subdural membrane → clot enlargement).

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Data has been reflected above to reveal bridging veins:

![Subdural Hematoma](source)

Data has been reflected back (with small portion visible at lower right) to reveal subdural hematoma:

![Subdural Hematoma](source)

Here is bilateral chronic subdural hematoma; blood clots are brown to tan because of organization:

![Subdural Hematoma](source)
CLINICAL FEATURES

A. Clinically silent.

B. Brain compression (slow venous obstruction enabling large hematomas to form before clinical signs appear) — can progress rapidly or slowly.

Most acute SDHs manifest within 48 hours:
- = 50% patients are unconscious from time of injury; LUCID INTERVAL is observed in 30-70% cases.
- may have focal signs* (due to prolonged brain tissue compression under hematoma), but often clinical manifestations are nonlocalizing (due to ICP*) later, brain herniation may develop.
- deficits are soft (not as profound as in other hematomas)
- hemianesthesia, hemianopia are seldom observed (anatomic structures are deep and not easily compressed)

In pre-CT era, chronic SDHs earned label "great imitator" because of variable course and presentation (sometimes mistaken for dementia, stroke, or brain tumor!).
- in 25-50% cases, there is no clear history of head trauma.
- signs or symptoms fluctuate in ≤ 24% cases (mimic TIA's).
- headache (90%), mild hemiparesis (45-58%), confusion (50%), drowsiness (40-50%), personality changes, papilledema, gait dysfunction, seizures are most common presenting features.

N.B. after hematomas have exerted pressure on brain for long time (perhaps year or more), removing them does little to improve cognitive function.

DIAGNOSIS

Hematostasis (PT, aPTT, platelet count) L.P. (absolutely contraindicated) — xanthochromia, variable number of RBCs.

Noncontrast CT - crescentic collection over hemispherical convexity, without extension into depths of sulci; can cross suture lines and continue along falx and tentorium (do not cross midline!)

N.B. blood pressure is less and is less restricted (vs. EDH) → crescentic shape

Acute SDH - hyperdense* (40-90 HU); rarely, can appear isodense:
- low hematocrit (anemia)
- hyperacute clot (< 1 h old)
- active bleeding
- small hematomas may not be depicted because attenuation similar to adjacent inner table of skull (H: wider CT window and level, e.g. 240 and 80 HU).

Subacute SDH — isodense*, important signs - effacement of cortical sulci, displacement of gray matter — white matter junction ("bucking"); membranes are not vascularized.
- better visualization — MRI (high signal on T1) or contrast-enhanced CT (opacification of cortical vessels - definition of brain margins).

Chronic SDH - hypodense* (15-30 HU, i.e. isodense to CSF); vascularized membranes enhance with contrast.
*compared to brain

Rule of thumb: blood remains denser than brain for 1 week, and is less dense after 3 weeks.

underlying brain is flattened (maus effect), and subarachnoid space is often clear.

absent midline shift - suspect contralateral mass (e.g. 20% chronic SDHs are bilateral); useful sign — ventricular frontal horns lie closer together ('rabbit's ear' configuration).

excessive midline shift - suspect underlying cerebral edema.

in intrhemispheric SDHs, lungs cerebral appears thickened and irregular.
- intrhemispheric SDH may mimic SAH (subarachnoid blood clears after several days; SDH remains wedge-shaped, smooth-bordered, hyperattenuating lesion).
- intrhemispheric SDH may spread onto tentorium → characteristic 'comma shape' on axial CT.

posterior fossa SDH does not cross midline or extend above tentorium (vs. EDH).

temporal and tentorial SDHs are better detected on coronal MRI (than on axial CT).

rebleeding into subacute / chronic SDH makes hematoma biconvex and heterogeneous density (mixed old and fresh blood, sedimentation levels) — in general, looks like EDH with heterogeneous density.

Size of extra-axial hematoma is more important factor than whether blood is epidural or subdurals in location!

Suspect child abuse* (esp. if posterior intrhemispheric & tentorial SDHs)

cerebral swelling of chronic SDH makes hematoma biconvex and water density — in general, looks like EDH with water density.

MRI is most sensitive imaging test! (esp. in subacute and chronic phase).

absence of clear history of trauma → angiography (search for ruptured aneurysm or dural AV fistula).

angiographic signs of SDH – avascular zone between skull and brain with away dislocation of major vessels.
Skull fracture with adjacent small acute SDH (window and level values are widened over standard values to aid detection):

MRI - Subacute SDH with extension into anterior interhemispheric cistern (note that sutures do not contain spread):

SDH with adjacent SAH due to ruptured MCA aneurysm:

SDH due to ruptured right PComA aneurysm:

Right frontal subacute SDH - note displaced gray matter–white matter junction, and midline shift:

Subacute-on-chronic SDH with blood fluid level (acute hemorrhage into chronic collection)
Acute-on-chronic SDH:

- Less dense than brain but denser than CSF; it is denser posteriorly; midline displacement is greater than would be expected from size of lesion - suggests extensive underlying swelling; contralateral (left) ventricle is dilated.

Subacute SDH - bilateral SDHs, right greater in size than left:

Source of picture: The Internet Pathology Laboratory for Medical Education (by Edward C. Klatt, MD) >>

Acute SDH:

MRI - bilateral SDHs (arrows) with suboccipital extension.
Interhemispheric acute SDH:

Bilateral chronic SDHs; midline shift is absent:

Chronic SDH with no mass effect:

Bilateral chronic SDHs with acute rebleeding:

Acute SDH extending over entire left hemisphere; sedimentation level is seen (patient has clotting dysfunction):
Subdural Hematomas

Subacute SDHs:
A) Noncontrast CT: inward displacement of gray-white matter junction of left cerebral hemisphere; small sedimentation level is apparent in posterior portion.
B) Contrast CT at same level as A: good visualization of lateral margin of left cerebral hemisphere (opacified cortical veins).

Acute and subacute SDHs:
A) T1-MRI: large fluid collections (white arrows) consistent with large subacute SDHs.
B) T2-MRI: hyperintense signal (white arrows) representing subacute blood and hypointense signal (black arrows) representing more recent hematoma.

Calcified SDHs:
A) In frontal view, bilateral shell-like calcifications form cast of cerebral hemispheres.
B) In lateral view, outer calcified membranes are near bony skull, and inner membranes are separated from outer by relatively clear zones.
SUBDURAL HEMATOMA

TREATMENT

In all cases, hematoma complete resolution should be documented (conservatively treated acute SDH can evolve into chronic SDH).

CONSERVATIVE MANAGEMENT

- surgical drainage is not required in many cases (acute SDH may disappear spontaneously, but may evolve into subacute or chronic lesion!).
- seizure prophylaxis and other measures → see p. TrH13
- platelet transfusion for patient on ASA/Plavix – probably no effect!

Chronic SDH

- chronic SDH can be treated with TRANEXAMIC ACID (TXA) 650-750 mg PO daily for 30 days without concomitant surgery; tranexamic acid might simultaneously inhibit fibrinolytic and inflammatory (kinin-kallikrein) systems, which might consequently resolve CSDH; some experts administer it postop (after burr hole washout).


Kageyama H, Toyooka T, Tsuzuki N, Oka K.

- EG-1964 (Edge Therapeutics, Inc.) - polymer-based filament that contains APROTININ - pancreatic trypsin inhibitor.
  - indicated to prevent rebleeding and reduce the need for blood transfusions following cardiac bypass surgery.
  - IV administration has serious thrombotic side effects.
  - EG-1964 delivers sustained dose of aprotinin over 21-28 days directly to site of SDH.

- chronic SDH growth is due to the highly friable nature of the vascularized membrane that forms after initial injury; proposed treatment for recurrent symptomatic SDH - middle meningeal artery (MMA) embolization with the goal of eliminating the arterial supply to this vascularized membrane.

HEMATOMA EVACUATION

ACUTE SDH

- indications for surgery → see p. TrH13
- technique → see p. Op320

Chronic SDH

- any coagulopathy must be reversed ASAP.
- must be surgically evacuated if symptomatic* (+ significant mass effect on imaging); *except mild headaches

A. Burr hole craniostomy ("burr hole washout") - procedure of choice. → see p. Op320

B. Twist drill craniostomy – at bedside for very sick patient. → see p. Op320

C. Craniotomy, indicated for:
  a) multilocular / calcified SDHs.
  b) burr hole / twist drill drainage failures

N.B. some experts advocate craniotomy as first choice for cSDH but studies show that all results (complications, dispo, recurrences requiring reoperation*, mortality) are worse than with BHWO.

*24% (vs. 6-7% for BHWO)

Postoperatively → see p. Op320

PROGNOSIS

Most important prognostic factors:
1. Concomitant primary brain injury
2. GCS score
3. Age (esp. > 60 yrs.)
Chronic SDH increases mortality 17-fold at 1 year.

After surgical evacuation, subdural hematoma recurs in 5-30% patients.

**SDH IN INFANTS**

**NEWBORNS**
- SDH was once considered the most common intracranial birth injury (but now ↓ with improved obstetric care).
- in majority of cases, bleeding is bilateral and located over dorumetal surfaces of frontal and parietal lobes.
- clinically: seizures, pallor, tense anterior fontanel, rapidly enlarging head, hypotonia, poor Moro reflex.
  - may be abnormal at birth (no spontaneous respiration, severe hypotension, seizures, retinal hemorrhage).
  - initial manifestation may be generalized seizures within first 6 months of life.
  - acute cases may progress to herniation.
- diagnosis: ultrasound, CT, funduscoppy (50% show retinal or subhydraloid hemorrhages).
  - if SDH interfered with brain growth, skull vault may be thick, with paucity of convolutional impressions, and hypertrophy of air cells and parasellar sinuses.
  - calcification streaks/plaques, often parallel to vault, may be visible in capsule of chronic SDH.
- treatment – evacuation through craniotomy.

Ultrasound - left echogenic acute subdural hematoma (H), associated with right subarachnoid anechogenic effusion (E). Falx sinus (arrow) – subtemporal craniectomy – to let fluid drain into pterygoid fossa.

**OLDER INFANTS**
- SDH is most common intracranial lesion in children < 2 years:
  a) Shaken baby syndrome!! (chronic SDH in infants who do not yet walk).
  see p. TH2D >>
  b) complication of shunt procedures
  c) bleeding disorders.
- clinically: macronasma (symmetrical or asymmetrical), tense anterior fontanel, lethargy / irritability, seizures, poor feeding, vomiting, failure to thrive.
- diagnosis: CT, diagnostic subdural tap (should be done bilaterally, even if positive on one side).
- treatment – repeated daily subdural taps monitored by CT, head circumference measurements.
  - resorption of hematoma must occur over weeks ± months.
  - if > 10 taps are done (symptoms persist after 2 wk of daily drainage) → surgical treatment (e.g. subdural-peritoneal shunting or subdural-subgaleal shunting for 6 months; historically – subtemporal craniectomy – to let fluid drain into pterygoid fossa).
  - removal of membranes was once thought to be important to avoid brain growth restriction, but this no longer seems necessary.
- 25% have some psychomotor retardation.

**SUBDURAL TAP**
- anterior fontanelle becomes effectively closed between 9 and 18 months.
- equipment - sterile prep and drapes, gloves, razor, blunt short-beveled spinal needle with stylet (20 G, 1½ or 2½ inch), manometer, CSF collection tubes, and local anesthesia (if desired).
- scalp is shaved (i.e. at least 2-3 cm from midline) - to prevent sagittal sinus injury.
  - use ZIGZAG PUNCTURE to prevent later fluid leakage (puncture dislocated skin at right angle, then aim needle laterally).
  - data is entered with sudden “popping” sensation.
  - cerebral cortex is ≈ 1.5 cm from skin surface (attachment of hemostat 5-7 mm from beveled end of needle should provide adequate safeguard).
- remove stylet - subdural fluid is allowed to drain spontaneously (fluid is never aspirated - risk of drawing pial vessels into point of needle).
  - only 10-20 mL of subdural fluid should be removed from each side at one time (removing larger amounts may precipitate rebleeding or shock).
  - if pial vessel is punctured, bleeding will usually cease spontaneously.
  - pressure is measured with manometer.
  - specimens are collected for Gram staining, culture, cells, glucose, and protein.
- remove needle → apply pressure → firm sterile dressing → place in sitting position (to prevent leakage).
- repeat on opposite side.

4. Time from trauma to surgical evacuation of hematoma (esp. > 4 hours).

Macrocrania (symmetrical or asymmetrical), tense anterior fontanel, rapid enlarging head, hypotonia, poor Moro reflex. May be abnormal at birth (no spontaneous respiration, severe hypotension, seizures, retinal hemorrhage). Initial manifestation may be generalized seizures within first 6 months of life. Acute cases may progress to herniation.

Diagnosis: ultrasound, CT, funduscoppy (50% show retinal or subhyaloid hemorrhages). If SDH interfered with brain growth, skull vault may be thick, with paucity of convolutional impressions, and hypertrophy of air cells and parasellar sinuses. Calcification streaks/plaques, often parallel to vault, may be visible in capsule of chronic SDH.

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- Repeat on opposite side.
- continued leakage from puncture site → apply collodion-impregnated cotton fluff over puncture wound + elevate head 20-30°.