Subdural Hematoma

Last updated: January 7, 2022

EPIDEMIOLOGY

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

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) e.g. encephalopathies
   2) (saccular) CSF leak / CSF shunts causing ventricular decompression (≈ stretching of bridging veins) and low ICP (intracranial venous congestion - seen on MRI as abnormal meningeal enhancement)
   3) intracerebral hemorrhage, ruptured intracranial aneurysm (blood may dissect into subdural space, esp. PComA→ convexital SDH, distal ACA→ parafalcine 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!); shaken baby syndrome

N.B. bleeding is most commonly venous (vs. 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, 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)
* i.e. 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 homogenic 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 hour; most common type of traumatic intracranial hematoma (50-30% of severe head injuries; 1% of mild head injuries)

- commonly (≈ 50%) associated with "e.g. intracerebral brain injury" (!!! (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)

average age of trauma patient without acute SDH - 26 years; average age of patient with acute SDH - 41 years.

- mortality:
  - simple SDH (if no other brain injury) = 20%; complicated SDH (e.g. with contusions) = 60%
  - GCS 12-15 = 86%
  - GCS 3-5 = 76%

PATHOLOGY, PATHOGENESIS

- movement of brain relative to skull - "rupture" (via shearing mechanism) of MENINGEAL VENOUS SINUS (CEDES: 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 artery (hematoma looks more like EDH - less shaped) or oozing brain laceration.

SDH - (rapidly clotting) blood collection in plane between dura and arachnoid.

PATHOLOGY

- Spontaneous
  - Recurrent SDH workup
    - Location
      - SDH in infants
        - Newborns
        - Older infants
      - Subdural Tap
    - Treatment
      - Conservative Management
      - Hematoma Evacuation
  - Chronic SDH
    - Acute SDH
    - Chronic SDH
  - Pathogenesis
    - Location
      - Location
      - Classification
        - Acute SDH manifests during first 72 hour; most common type of traumatic intracranial hematoma (50-30% of severe head injuries; 1% of mild head injuries)
        - commonly (≈ 50%) associated with "e.g. intracerebral brain injury" (!!! (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)

SDH is not usually associated with skull fractures*
* if skull fracture is present, it is commonly contralateral to SDH
Subdural Hematoma

**Subacute SDH manifests when > 3-20 days old** (surgical literature favors > 3 days; radiological literature favors > 7 days).

**Chronic SDH manifests when > 14-20 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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**Dura has been reflected above to reveal bridging veins:**

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

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

A. Clinically silent.

B. Brain compression (slow venous bleeding enables 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; (SEMI LUCID INTERVAL is observed in 30-70% cases.
- There may be 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, hemianopsia 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 TIAs).
- headache (90%), mild hemiparesis (45-58%), confusion (56%), 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.

Nonepileptic, Stereotypical, and Intermittent Symptoms (NESSIS)

- NESSIS clinically manifests as symptoms lasting > 5 minutes, dysphasia, preserved awareness, lack of positive symptomatology (such as clonic movements), lack of response to AEDs.
- possible pathophysiology - cortical spreading depression or depolarization (CSD) (phenomenon similar to seen in SAH-associated vasospasm, stroke, TBI) – first described in animals in 1940 by Leão.
- EEG is negative for ictal or epileptiform discharges*; EEG most commonly shows focal or generalized slowing.

*N.B. scalp EEG has only 70% sensitivity to detect ictal or epileptiform discharges.

differentiation from seizures is difficult without EEG; some clinical features are helpful:

<table>
<thead>
<tr>
<th>Proposed Scoring System for the Diagnosis of NESSIS (before EEG)</th>
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<tbody>
<tr>
<td>Sensitivity of 96.6%, specificity of 100%</td>
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<tr>
<td><strong>Features supporting NESSIS</strong></td>
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<tr>
<td>Negative symptoms</td>
</tr>
<tr>
<td>Duration ≤ 2 minutes</td>
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<tr>
<td>Dysphasia</td>
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<tr>
<td>Migraine</td>
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<tr>
<td>≥ 5 episodes</td>
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<tr>
<td>Stereotypy</td>
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<tr>
<td>Features against NESSIS</td>
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<tr>
<td>Impaired awareness</td>
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<td>Chronic movements</td>
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<tr>
<th>Odds ratio (95% CI)</th>
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<tr>
<td>Favoring epilepsy</td>
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<tr>
<td>Favoring NESSIS</td>
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<table>
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<tr>
<th>Score</th>
<th>NESSIS diagnosis if total score ≥4 and no positive EEG</th>
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<tbody>
<tr>
<td>4</td>
<td>Yes</td>
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<tr>
<td>3</td>
<td>Yes</td>
</tr>
<tr>
<td>2</td>
<td>No</td>
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<td>1</td>
<td>No</td>
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outcomes and mortality are better compared to patients with confirmed epilepsy.

- treatment
  - CSF in human models have shown potential response to lamotrigine and topiramate;
  - for patients in whom epilepsy is still considered a possibility, topiramate could be an interesting first-line option, as it would address both seizures and CSF.

- GENESIS trial (Generating Evidence on N Engl J Med) is on the way.

### DIAGNOSIS

**Hematoma** (PT, dPTT, platelet count)

- LP (absolutely contraindicated) - xanthochromia, variable number of RBCs.

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

- MRI - most sensitive imaging test!

- N.B. blood pressure is less and space 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)
- 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 (“bucketing”); 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.

- underlining brain is flattened (mass effect), and subarachnoid space is often clear.
- absent midline shift - suspect contralateral mass (e.g. = 20% chronic SDHs are bilateral); useful sign - venricular frontal horns lie closer together (‘rabbit’s ear’ configuration).
- excessive midline shift - suspect underlying cerebral edema.
- in interhemispheric SDH, falx cerebri appears thickened and irregular.
  - interhemispheric SDH may mimic SAH (subarachnoid blood clearing after several days; SDH remains wedge-shaped, smooth-bordered, hyperattenuating lesion).
  - interhemispheric 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 frontal 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 subdural in location!

- Suspect child abuse? (esp. if posterior interhemispheric & tentorial SDHs)
- ommotic 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) but CT is usually enough.

- chronic SDH - high signal on T1; membranes have low signal intensities (on T1 and T2).

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.

**T1 & T2-MRI** - bilateral subacute SDHs (increased signal intensity; areas of intermediate intensity represent more acute hemorrhage into subacute collections)

- Shell fractures with adjacent small acute SDH (epiglott and level values are widened over standard values to aid detection).

**MRI** - subacute SDH with extension into anterior interhemispheric fissure (note that sutures do not contain space).
Subdural Hematoma

SDH with adjacent SAH due to ruptured MCA aneurysm:

SDH due to ruptured right PCoM 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:
**Subdural Hematoma**

**Subacute 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.

**Bilateral SDHs, right greater in size than left.**

**Acute SDH.**

MRI - bilateral SDHs (arrows) with subcortical extension.
Subdural Hematoma

Interhemispheric acute SDH:

Bilateral chronic SDHs; midline shift is absent:

Chronic SDH with no mass effect:

Bilateral acute-on-chronic SDHs:

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

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 (specific cortical veins).

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**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.

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**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.

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**Recurrent SDH Workup**

1) MRI + DWI, CTA – for vascular abnormalities

2) MRI of spine – for occult CSF leak
CONSERVATIVE MANAGEMENT

- surgical drainage is not required in many cases (acute SDH may disappear spontaneously, but may evolve into subacute or chronic lesion) - managed with serial CT and close neurological observation in a neurosurgeon and then as an outpatient
- seizure prophylaxis and other measures → see p. TiH13
- monitor coagulation parameters!
- platelet transfusion for patient on ASA/Plavix → probably no effect!


**CRONIC SDH**

- chronic SDH growth is due to the highly friable nature of the vascularized membrane that forms after initial injury.

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


2. Dr. Okulnowo (UPMC) gives MEHRD, DOREPAK, Dr. Day (UAMS) gives DEXAMETHASONE 2 mg qhs for 2 week, then taper to placebo without repeat CT findings.

Desc-SDH Trial


— multicenter (23 sites), randomized, placebo-controlled trial in the United Kingdom

— adults with symptomatic chronic SDH

  o mean age 74 years
  o symptoms: headache, gait disturbance, confusion or cognitive decline, limb weakness, speech disturbance, depressed consciousness, and seizures.
  o exclusions: glucocorticoids are contraindicated, (or had been receiving within 1 month before screening) glucocorticoids on a regular basis, severe lactose intolerance, psychiatric disorders, acute SDH.

  o 94% underwent surgery during index admission (decision to operate was made by the treating clinician) — trial was unable to statistically explore dexamethasone role as an alternative to surgery

  o groups were similar (e.g. 60% in both groups had a score of 1-3 on the modified Rankin scale at admission. 94% in both groups had GCS 13-15).

  o 1:1 ratio dexamethasone (375 mg to 341 patients*) vs. placebo (373 to 339 patients*).

  o Oral dexamethasone 2 mg tablet regimen started within 72 hrs of admission: 8 mg bid on days 1 to 7 and then as outpatient.

  o 4 mg bid on days 7 to 9 → 3 mg bid on days 7 to 9 → 2 mg bid on days 10 to 12 → 2 mg once daily on days 13 and 14.

  + some withdrew consent, some were lost to FU.

Outcomes

— primary outcome - score of 0.3 (favorable outcome) on the modified Rankin scale at 6 months after randomization, favorable outcome was reported: 83.9% patients in dexamethasone group

  90.3% patients in placebo group

  difference, -6.4 percentage points [95% confidence interval, -11.4 to -1.4] in favor of the placebo group (p = 0.01).

  odds ratio for a favorable outcome with dexamethasone was 0.55 (95% CI, 0.33 to 0.91) in favor of the placebo group (P = 0.02).

  — adverse events: more in the dexamethasone group than in the placebo group.

  Adverse events of special interest: hyperglycemia leading to treatment or discontinuation of the trial regimen, new-onset diabetes, hyperinsulinaemic state, new-onset psychosis, peptic ulceration / GI bleeding, upper GI side effects.

  — repeat surgery for recurrence was performed: 17.9% in dexamethasone group

  7.1% patients in placebo group.

  — follow-up imaging was not mandated (trial outcomes did not include imaging results to address the possible effect of dexamethasone on the size of chronic subdural hematoma).

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<tr>
<th>Outcome</th>
<th>Dexamethasone</th>
<th>Placebo</th>
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<tr>
<td>Favorable</td>
<td>83.9%</td>
<td>90.3%</td>
</tr>
<tr>
<td>Favorable</td>
<td>82%</td>
<td>100%</td>
</tr>
<tr>
<td>Adverse</td>
<td>10.9%</td>
<td>3.2%</td>
</tr>
<tr>
<td>Any infection</td>
<td>9.4%</td>
<td>11.1%</td>
</tr>
<tr>
<td>Repeat surgery</td>
<td>7.5%</td>
<td>7.1%</td>
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TREATMENT

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

In all cases, hematoma complete resolution should be documented (conservatively treated acute SDH can evolve into chronic SDH).
3. Proposed treatment for recurrent (or new in high risk patients – elderly on anticoagulation) symptomatic SDH: middle meningeal artery (MMA) embolization with the goal of eliminating the arterial supply to this vascularized membrane:
   - use PVA particles - able to travel distally within the MMA vasculature and cover a broad anatomic area, limiting the chance of new collaterals forming distally (vs. liquid embolics such as Onyx or nBCA glue provide more of a “stump” embolization proximally).

4. 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

**HEMATOMA EVACUATION**

**ACUTE SDH**

**Guidelines**: “Surgical management of acute subdural hematomas” in Neurosurgery. 2006 Mar;58(3 Suppl):S1-S24

- indications for ASAP* surgery: also see p. ThH 1 >>
  a) acute SDH with a thickness > 10 mm, regardless of the GCS score
  b) midline shift > 5 mm, regardless of the GCS score.
  c) GCS < 9 and GCS decreased between the time of injury and hospital admission by ≥ 2 points
  d) GCS < 9 and asymmetric or fixed and dilated pupils
  e) GCS < 9 and ICP > 20 mm Hg

- acute SDH in coma (GCS < 9) → ICP monitoring.

- surgical evacuation of an acute SDH should be performed using a craniotomy ± bone flap removal and duraplasty. see p. Op320 >>

*delay for 4 hours is a benchmark of neurosurgical care:

Wilberger et al. (1991) study also revealed that the appropriate strategies to prevent secondary brain injury may be more important than the timing of surgery in determining outcome (i.e. an increased focus on how to manage ICP during the interval between injury and surgery).

- after evacuation of traumatic SDH, place EVD, esp. if preop GCS was ≤ 8.
**SUBDURAL HEMATOMA**

**CHRONIC SDH**
- any comatose patient must be reversed ASAP.
- must be surgically evacuated if symptomatic* + significant mass effect on imaging)

A Burr hole craniostomy (“burr hole washout”) - procedure of choice: see p. Op320 >>

<table>
<thead>
<tr>
<th>Burr hole drainage</th>
<th>Craniotomy</th>
<th>Statistical significance</th>
</tr>
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<tbody>
<tr>
<td>Recurrence</td>
<td></td>
<td></td>
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<tr>
<td>10/10 (20%)</td>
<td>7/19(37%)</td>
<td>p = 0.15%</td>
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Meta-analyses have confirmed findings of the paper by Swien and Geley (Weigel et al., 2003; Lega et al., 2009; Mondorf et al., 2009)

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

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

N.B. some experts advocate craniostomy 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**

**ACUTE SDH**

Most important prognostic factors
1. Concomitant primary brain injury
2. GCS score
3. Age (esp. > 40 yrs).
4. Time from trauma to surgical evacuation of hematoma (esp. > 4 hours)

**CHRONIC SDH**

After surgical evacuation, subdural hematoma recurs in 10-20% patients. Chronic SDH increases mortality 17-fold at 1 year.

**SDH IN INFANTS**

**NEWBORN**
- SDH was once considered most common intracranial birth injury (but now ↓ with improved obstetric care).
- in majority of cases, bleeding is bilateral and located over dorsolateral 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, funduscopy (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 a
  - calcification streaks plaques, often parallel to vault, may be visible in capsule of chronic SDH.
- treatment – evacuation through craniostomy.

Ultrason - left echoic acute subdural hematoma (H), associated with right subarachnoidal anechoic effusion (E), falx cerebri (arrow) remains straight.

**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)
  - complication of shunt procedures
  - bleeding disorders.
  - clinically: macrocrania (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 craniostomy – to let fluid drain into pterygoid fossa).
    - removal of brain tissue was once thought to be important to avoid brain growth restriction, but this no longer seems necessary.
  - 25% have some psychomotor retardation.
**SUBDURAL HEMATOMA**

- anterior fontanelle becomes effectively closed between 9 and 18 months.
- equipment - sterile prep and drape, gloves, razor, blunt short-beveled spinal needle with stylet (20 G, 1½ or 2½ inch), manometer, CSF collection tubes, and local anesthetics (if desired).
- scalp is shaved anterior to ears (over lateral margins of anterior fontanelle).
- restrain infant by bundling him in sheet; supine, head firmly held by attendant (avoid excessive neck flexion).
- raise skin wheal of local anesthetic at puncture site.
- insert needle through skin at extreme lateral limit of anterior fontanelle where it meets coronal suture (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).
  - dura 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.
- continued leakage from puncture site → apply collodion-impregnated cotton fluff over puncture wound + elevate head 20-30°.

**BIBLIOGRAPHY** for ch. “Head Trauma” → follow this LINK >>