

# 27. Head trauma

## CONCUSSION

AKA mild traumatic brain injury (MTBI).

**Definition:** Alteration of consciousness without structural damage as a result of nonpenetrating traumatic brain injury (TBI).

Alterations in consciousness may include: confusion, amnesia (the hallmarks of concussion), or loss of consciousness (LOC). Loss of consciousness is not a requirement<sup>1-4</sup> (see Table 27-2 for grading scales). Frequent neurobehavioral features of concussion are shown in Table 27-1. Patients themselves may be unaware whether or not they experienced LOC<sup>5</sup>.

The alteration should be "brief", but there is no consensus on the length of time considered to be "brief".

There are no gross or microscopic parenchymal abnormalities. CT is normal or significant only for mild swelling which may represent hyperemia<sup>6</sup>. MRI will demonstrate abnormalities in up to 25% of cases where CT is normal<sup>7</sup>. The term *contusion* should be used when there are structural abnormalities (as may be seen on CT).

Confusion may be evident immediately following the blow, or may take several minutes to develop<sup>8</sup>. When there is LOC, the fact that it is often virtually instantaneous (there may be a latency of a few seconds), and the usually rapid return of function with no evidence of microscopic changes suggests that the LOC is due to a transient disturbance in neuronal function. Levels of glutamate (an excitatory neurotransmitter) rise after concussion and the brain enters a hyperglycolytic and hypermetabolic state which may persist up to 7-10 days after the injury. During this period the brain may be more susceptible to a second insult (second impact syndrome, see below) which, due in part to impairment of cerebral autoregulation, may produce much more severe sequelae (including possible malignant cerebral edema, see page 852) than it would have acting alone.

Also, see below for sports-related concussion.

Concussion may be followed by post-concussive syndrome (see page 910).

## Management and admission criteria

See Categories 1 & 2 on page 856.

## SPORTS RELATED CONCUSSION

~ 10% of head and spinal injuries are a result of a sports-related event<sup>9</sup>. Concussion, AKA mild traumatic brain injury (MTBI), is very different from the severe types of head injuries commonly seen by neurosurgeons in the E/R and office. The widest experience in studying this entity derives from athletics, and generalization to other types of trauma must be done circumspectly.

## Concussion grading

The Glasgow coma scale is too insensitive for use with mild TBI. Many concussion grading systems have been proposed, the two most widely used are those of Cantu<sup>10, 11</sup>, and that of the American Academy of Neurology (AAN)<sup>4</sup> (based on the guidelines of the Colorado Medical Society<sup>12</sup>) both of which are shown in Table 27-2. LOC by itself may not be a significant discriminant (e.g. confusion > 30 minutes may be worse than LOC for a few seconds). Most systems consider a concussion to be mild if there is a change in sensorium without loss of consciousness, however they differ mostly in the definition of "change in sensorium".

There is no scientific basis to recommend one system over another. Recommendation: select one system and use it consistently. Do not place undue emphasis on grading.

Table 27-1 Possible findings in concussion<sup>1</sup>

- vacant stare or befuddled expression
- delayed verbal & motor responses: slow to answer questions or follow instructions
- easy distractibility, difficulty focusing attention, inability to perform normal activities
- disorientation: walking in the wrong direction, unaware of date, time or place
- speech alterations: slurred or incoherent, disjointed or incomprehensible statements
- incoordination: stumbling, inability to tandem walk
- exaggerated emotionality: inappropriate crying, distraught appearance
- memory deficits: repeatedly asking same question that has been answered, cannot name 3 out of 3 objects after 5 minutes
- any period of LOC: paralytic coma, unresponsiveness to stimuli

## Second impact syndrome (SIS)

A rare condition described primarily in athletes who sustain a second head injury while still symptomatic from an earlier one. Classically, the athlete walks off the field under their own power after the second injury, only to deteriorate to coma within 1-5 minutes and then, due to vascular engorgement, develops malignant cerebral edema that is refractory to all treatment and progresses to herniation. Mortality: 50-100%.

A syndrome compatible with SIS was first described by Schneider<sup>13</sup> in 1973, and was later dubbed the "second impact syndrome of catastrophic head injury" in 1984<sup>14</sup>. Although it is contended that SIS is rare (if it exists at all) and may be overdiagnosed<sup>15</sup>, its apparent predilection for teens and children still warrants extra precaution following concussion.

## Return to play guidelines

No system of return to play (RTP) guidelines has been rigorously tested and proven to be scientifically sound. Regardless of the system used, one universal recommendation of experts is:

★ a symptomatic player should not return to competition.

**PRACTICE GUIDELINE 27-1** shows AAN guidelines for RTP (if no contraindications, see Table 27-3<sup>16</sup>). For spine-related RTP guidelines, see page 980.

**Table 27-2 Concussion grading**

Grade	Cantu system*	★ AAN system*
1 (mild)	1. PTA < 30 mins 2. no LOC	1. transient confusion 2. no LOC 3. symptoms resolve in < 15 mins
2 (moderate)	1. LOC < 5 mins, or 2. PTA > 30 mins	as above, but symptoms last > 15 mins (still <u>no</u> LOC) (PTA is common)
3 (severe)	1. LOC ≥ 5 mins, or 2. PTA ≥ 24 hrs	<u>any</u> LOC

\* abbreviations: LOC = loss of consciousness; PTA = posttraumatic amnesia

**Table 27-3 Cerebral contraindications for return to contact sports**

1. persistent postconcussion symptoms
2. permanent CNS sequelae from head injury (e.g. organic dementia, hemiplegia, homonymous hemianopsia)
3. hydrocephalus
4. spontaneous SAH from any cause
5. symptomatic (neurologic or pain producing) abnormalities about the foramen magnum (e.g. Chiari malformation)

### PRACTICE GUIDELINE 27-1 SPORTS RELATED CONCUSSION

#### Level III<sup>16</sup> AAN guidelines:

AAN grade	Management recommendations for a single sports-related concussion (level III recommendations)
1 mild	<ol style="list-style-type: none"> <li>1. remove from contest</li> <li>2. examine q 5 mins for amnesia or postconcussive symptoms*</li> <li>3. may return to contest if symptoms clear within 15 mins</li> </ol>
2 moderate	<ol style="list-style-type: none"> <li>1. remove from contest</li> <li>2. disallow return that day</li> <li>3. examine on-site frequently for signs of evolving intracranial pathology</li> <li>4. reexamination the next day by a trained individual</li> <li>5. CT or MRI if H/A or other symptoms worsen or last &gt; 1 week†</li> <li>6. return to practice after 1 full week without symptoms*</li> </ol>
3 severe	<ol style="list-style-type: none"> <li>1. ambulance transport from field to E/R if still unconscious or for concerning signs (C-spine precautions if indicated)</li> <li>2. emergent neuro exam. Neuroimaging as appropriate</li> <li>3. may go home with head-injury instructions (see Table 27-10, page 857) if normal findings at time of initial neuro exam</li> <li>4. admit to hospital for any signs of pathology or for continued abnormal mental status</li> <li>5. assess neuro status daily until all symptoms have stabilized or resolved</li> <li>6. prolonged unconsciousness, persistent mental status alterations, worsening post-concussion symptoms, or abnormalities on neurologic exam → urgent neurosurgical evaluation or transfer to a trauma center</li> <li>7. after brief (&lt; 1 minute) grade 3 concussion, do not return to practice until asymptomatic for 1 full week*</li> <li>8. after prolonged (&gt; 1 minute) grade 3 concussion, return to practice only after 2 full weeks without symptoms*‡</li> <li>9. CT or MRI if H/A or other symptoms worsen or last &gt; 1 week†</li> </ol>



- \* evaluation at rest and with exertion (see text)
- † season is terminated for that player if CT/MRI shows edema, contusion, or other acute intracranial pathology. Return to play in any contact sports in the future should be seriously discouraged
- ‡ some experts also require a normal CT scan

The rationale for the waiting periods following grade 2 or 3 concussions is due to the potentially increased vulnerability of the brain to injury following a concussion (see *Second impact syndrome*, page 851). Almost all players with mild concussions should be able to return to the contest. Some also allow players with moderate concussions to return if they become symptom-free at rest and with exertion using provocative tests. **Exertion:** to evaluate under exertion, commonly utilized provocative tests include the 40-yard run, sit-ups, push-ups, and/or deep knee-bends<sup>9</sup>. In the E/R, exertion may be administered by having the patient lie on the exam table and tip the head backward slightly off the edge. The development of any symptoms during exertion is considered abnormal and precludes return to the present contest.

**Multiple concussions:** Multiple concussions in a short period of time are potentially dangerous (see *above*). Recommendations<sup>9</sup> for multiple concussions in the same season are shown in Table 27-4. Also, see *Chronic traumatic encephalopathy*, page 911 for long-term effects of multiple concussions.

### Neuroimaging

The need for neuroimaging (e.g. CT scan) in the athlete with resolved or improving symptoms is controversial, and is felt to be best left to the judgement of the treating physician. Suggested indications:

1. a severe concussion
2. symptoms persisting > 1 week, even if mild
3. before returning to competition after a 2nd or 3rd concussion in the same season

**Table 27-4 Recommendations for multiple sports-related concussions in the same season**

Concussion		Guidelines to be met before return to competition
No.	Severity	
2	mild	1 week*
	moderate or severe	1 month* + normal CT or MRI†
3	mild	most consider this a season ending injury and recommend CT or MRI†
	moderate	season ending injury, consideration for ending all participation in contact sports
2	severe	

\* without symptoms at rest and with exertion (see text)

† if any acute abnormalities on CT/MRI: terminate season. Consider ending all participation in contact sports

### CONTUSION

TBI with CT findings that may include:

- low attenuation areas: representing associated edema
- high attenuation areas (AKA "hemorrhagic contusions"): usually produce less mass effect than their apparent size. Most common in areas where sudden deceleration of the head causes the brain to impact on bony prominences (e.g. temporal, frontal and occipital poles). These areas may progress (or "blossom" in neuroradiological jargon) to frank parenchymal hemorrhages. Surgical decompression may sometimes be considered if herniation threatens (see page 893)

### Contrecoup injury

(French: "counter blow") in addition to the potential injury to the brain directly under the point of impact, the force imparted to the head may cause the brain to be thrust against the skull directly opposite the blow. May result in contusions typically in locations described above.

### OTHER DEFINITIONS

#### Posttraumatic brain swelling

This term encompasses two distinct processes:

1. increased cerebral blood volume: may result from loss of cerebral vascular autoregulation (see page 866). This hyperemia may sometimes occur with extreme rapidity, in which case it has sometimes been referred to as diffuse or "malignant cerebral edema"<sup>17</sup> which carries close to 100% mortality and may be more common in children. Management consists of aggressive measures to maintain ICP < 20 mm Hg and CPP > 60 mm Hg<sup>18A</sup>

2. true cerebral edema: classically at autopsy these brains “weep fluid”<sup>19</sup>. Both vasogenic and cytotoxic cerebral edema (see page 109) can occur within hours of head injury<sup>19, 20</sup> and occasionally may be treated with decompressive craniectomy (see *PRACTICE GUIDELINE 27-21*, page 893)

## Diffuse axonal injury (DAI) (AKA diffuse axonal shearing)

A **primary** lesion of rotational acceleration/deceleration head injury<sup>21</sup>. In its severe form, hemorrhagic foci occur in the corpus callosum and dorsolateral rostral brain stem with microscopic evidence of diffuse injury to axons (axonal retraction balls, microglial stars, and degeneration of white matter fiber tracts). Often cited as the cause of loss of consciousness in patients rendered immediately comatose following head injury in the absence of a space occupying lesion on CT<sup>22</sup> (although DAI may also be present with subdural<sup>23</sup> or epidural hematomas<sup>24</sup>).

May be diagnosed clinically when loss of consciousness (coma) lasts > 6 hours in absence of evidence of intracranial mass or ischemia. May be graded as shown in *Table 27-5*.

**Table 27-5 Grading DAI**

DAI grade	Description
mild	coma > 6-24 hrs, followed by mild-to-moderate memory impairment, mild-to-moderate disabilities
moderate	coma > 24 hrs, followed by confusion & long-lasting amnesia. Mild-to-severe memory, behavioral and cognitive deficits
severe	coma lasting months with flexor and extensor posturing. Cognitive, memory, speech, sensorimotor and personality deficits. Dysautonomia may occur

## GRADING HEAD INJURIES

Despite many (valid) criticisms, the initial post-resuscitation Glasgow Coma Scale (GCS) score (see *Table 12-1*, page 279) remains the most widely used and perhaps best replicated scale employed in the assessment of head trauma.

**Stratification:** There are a number schemes to stratify the severity of head injury. Any such categorization is arbitrary. A simple system based only on GCS score is as follows: GCS 14–15 = mild, GCS 9–13 = moderate, and GCS ≤ 8 = severe.

A more involved system<sup>25</sup> incorporates other factors in addition to the GCS score as shown in *Table 27-6*.

A classification system based on CT scan<sup>26</sup> is shown in *Table 27-7*.

**Table 27-6 Categorization of head injury severity**

Category	Criteria*
Minimal	GCS† = 15 No loss of consciousness (LOC) No amnesia
Mild	GCS = 14 OR GCS = 15 plus EITHER Brief LOC (< 5 min) OR Impaired alertness or memory
Moderate	GCS = 9 – 13 OR LOC ≥ 5 min OR Focal neurologic deficit
Severe	GCS = 5 – 8
Critical	GCS = 3 – 4

\* ALL criteria in any oval must be met to qualify

† GCS = Glasgow coma scale (see *Table 12-1*, page 279)

## GENERAL

56-60% of patients with GCS score ≤ 8 have 1 or more other organ system injured<sup>27</sup>. 25% have “surgical” lesions. There is a 4-5% incidence of associated spine fractures with significant head injury (mostly C1 to C3).

A. CPP ≥ 70 mm Hg is generally recommended (see *ICP treatment threshold*, page 876)

When a detailed history is unavailable, remember: the loss of consciousness may have preceded (and possibly have caused) the trauma. Therefore, maintain an index of suspicion for e.g. aneurysmal SAH, hypoglycemia, etc. in the differential diagnosis of the causes of trauma and associated coma.

Brain injury from trauma results from two distinct processes:

1. primary brain injury: occurs at time of trauma (cortical contusions, lacerations, bone fragmentation, diffuse axonal injury, and brainstem contusion)
2. secondary injury: develops subsequent to the initial injury. Includes injuries from intracranial hematomas, edema, hypoxemia, ischemia (primarily due to elevated intracranial pressure (ICP) and/or shock), vasospasm

**Hypotension:** Hypotension<sup>A</sup> (shock) is rarely attributable to head injury except:

1. in terminal stages (i.e. with dysfunction of medulla and cardiovascular collapse)
2. in infancy, where enough blood can be lost intracranially or into the subgaleal space to cause shock
3. where enough blood has been lost from scalp wounds to cause hypovolemia

## DELAYED DETERIORATION

~ 15% of patients who do not initially exhibit signs of significant brain injury may deteriorate in a delayed fashion, sometimes referred to as patients who "talk and deteriorate" or when more lethal, patient who "talk and die"<sup>28</sup>. Etiologies:

1. ~ 75% will exhibit an intracranial hematoma
  - A. may be present on initial evaluation and can then worsen
  - B. may develop in a delayed fashion
    1. delayed epidural hematoma (EDH): see page 896
    2. delayed subdural (SDH): see page 899
    3. delayed traumatic contusions: see page 893
2. posttraumatic diffuse cerebral edema: see page 853
3. hydrocephalus
4. tension pneumocephalus
5. seizures
6. metabolic abnormalities, includes:
  - A. hyponatremia
  - B. hypoxia: etiologies include pneumothorax, MI, CHF...
  - C. hepatic encephalopathy
  - D. hypoglycemia: including insulin reaction
  - E. adrenal insufficiency
  - F. drug or alcohol withdrawal
7. vascular events
  - A. dural sinus thrombosis: see page 1166
  - B. carotid (or rarely, vertebral) artery dissection: see page 1163
  - C. SAH: due to rupture of aneurysm (spontaneous or posttraumatic) or carotid-cavernous fistula (CCF) (see page 1113)
  - D. cerebral embolism: including fat embolism syndrome
8. meningitis
9. hypotension (shock)

Table 27-7 CT classification of head injury

Category	Definition	Mortality
Diffuse Injury I	no visible intracranial abnormality	10%
Diffuse Injury II	basal cisterns* present, 0-5 mm midline shift and/or lesion densities present	14%
Diffuse Injury III	basal cisterns* compressed or absent, 0-5 mm midline shift, no high or mixed density lesion > 25 cc	34%
Diffuse Injury IV	midline shift > 5 mm, no high or mixed density lesion > 25 cc	56%

\* see page 909 for information on basal cisterns

## 27.1. Transfer of trauma patients

It is sometimes necessary for a neurosurgeon to accept a trauma patient in transfer

A. SBP < 90 mm Hg may impair CBF and exacerbate brain injury and should be avoided see page 878

from another institution that is not equipped to handle major neurologic injuries, or to transfer patients to other facilities for a variety of reasons. *Table 27-8* lists factors that should be assessed and stabilized (if possible) prior to transfer. These items should also be evaluated in trauma patients on whom a neurosurgeon is consulted in his or her own E/R as well as in patients with other CNS abnormalities besides trauma (e.g. SAH).

**Table 27-8 Factors to assess in head injured patients**

Clinical concern	Items to check	Steps to remedy
hypoxia or hypoventilation	ABG, respiratory rate	intubate any patient who has hypercarbia, hypoxemia, or is not localizing
hypotension or hypertension	BP, Hgb/Hct	transfuse patients with significant loss of blood volume
anemia	Hgb/Hct	transfuse patients with significant anemia
seizures	electrolytes, AED levels	correct hyponatremia or hypoglycemia; administer AEDs when appropriate*
infection or hyperthermia	WBC, temperature	LP if meningitis is possible and no contraindications ( <i>see page 201</i> )
spinal stability	spine x-rays	spine immobilization (spine board, cervical collar & sandbags...); patients with locked facets should be reduced if possible before transfer

\* see *Seizures*, page 394, as well as *Posttraumatic seizures* on page 398

## 27.2. Management in E/R

### 27.2.1. Neurosurgical exam in trauma

The following describes some features that should be assessed under certain circumstances with the understanding that this must be individualized. This addresses only craniocervical injuries.

#### General physical condition (oriented towards neuro assessment)

1. visual inspection of cranium:
  - A. evidence of basal skull fracture (*see Basal skull fractures*, page 887):
    1. raccoon's eyes: periorbital ecchymoses
    2. Battle's sign: postauricular ecchymoses (around mastoid air sinuses)
    3. CSF rhinorrhea/otorrhea: *see page 301*
    4. hemotympanum or laceration of external auditory canal
  - B. check for facial fractures
    1. LeFort fractures (*see page 890*): palpate for instability of facial bones, including zygomatic arch
    2. orbital rim fracture: palpable step-off
  - C. periorbital edema, proptosis
2. cranio-cervical auscultation
  - A. auscultate over carotid arteries: bruit may be associated with carotid dissection
  - B. auscultate over globe of eye: bruit may indicate traumatic carotid-cavernous fistula (*see Carotid-cavernous fistula*, page 1113)
3. physical signs of trauma to spine: bruising, deformity
4. evidence of seizure: single, multiple, or continuing (status epilepticus)

#### Neurologic exam

1. cranial nerve exam
  - A. optic nerve function
    1. if conscious: serial quantitation of vision in each eye is important<sup>29</sup> (*see page 863*). A Rosenbaum near vision card is ideal (*see inside back cover*), otherwise use any printed material. If patient cannot see this, check if they can count fingers. Failing this, check for hand motion vision and lastly light perception. Children may develop transient cortical blindness lasting 1-2 days, usually after a blow to the back of the head

2. if unconscious: check for afferent pupillary defect (see page 831), best demonstrated with swinging flashlight test (see page 830). Indicates possible optic nerve injury
3. funduscopic exam: check for papilledema, pre-retinal hemorrhages, retinal detachment, or retinal abnormalities suggestive of anterior optic nerve injury. If a detailed exam is required, pharmacologic dilatation with mydriatics may be employed, however, this precludes pupillary exam for a variable period of time, and should be undertaken advisedly (see page 832)
- B. pupil: size in ambient light; reaction to light (direct & consensual)
- C. VII: check for peripheral VII palsy (facial asymmetry of unilateral upper and lower facial muscles): see *Posttraumatic facial palsy*, page 889
- D. VI: abducens palsy following trauma may occur as a result of ↑ ICP (see page 836) or with clival fractures (see page 888)
2. level of consciousness/mental status
  - A. Glasgow coma scale for quantitating level of consciousness in poorly responsive patient (see Table 12-1, page 279)
  - B. check orientation in patient able to communicate
3. motor exam (assesses motor tracts from motor cortex through spinal cord)
  - A. if patient is cooperative: check motor strength in all 4 extremities
  - B. if uncooperative: check for movement of all 4 extremities to noxious stimulus (differentiate voluntary movement from posturing or stereotypical spinal cord reflex). This also assesses sensation in an unresponsive patient
  - C. if any doubt about integrity of spinal cord: also check "resting" tone of anal sphincter on rectal exam, evaluate voluntary sphincter contraction if patient can cooperate, check anal wink with pinprick, and assess bulbocavernosus reflex (see *Neurological assessment*, page 944 for details)
4. sensory exam
  - A. cooperative patient:
    1. check pinprick on trunk and in all 4 extremities, touch on major dermatomes (C4, C6, C7, C8, T4, T6, T10, L2, L4, L5, S1, sacrococcygeal)
    2. check posterior column function: joint position sense of LEs
  - B. uncooperative patient: check for central response to noxious stimulus (e.g. grimace, vocalization..., as opposed to flexion-withdrawal which could be a spinal cord mediated reflex)
5. reflexes
  - A. muscle stretch ("deep tendon") reflexes if patient is not thrashing: e.g. preserved reflex indicates that a flaccid limb is due to CNS injury and not nerve root injury (and vice versa)
  - B. check plantar reflex for upgoing toes (Babinski sign)
  - C. in suspected spinal cord injury: the anal wink and bulbocavernosus reflex are checked on the rectal exam (see above)

## INDICATIONS FOR CT AND ADMISSION CRITERIA FOR TBI

A multidisciplinary panel<sup>30</sup> prospectively followed 7,035 patients with head trauma to determine the probability of an intracranial injury (ICI) (and to evaluate the utility of skull x-rays (SXR) in head trauma, discussed only briefly here, see page 859 for more). The panel stratified patients into one of three groups based on the likelihood of intracranial injury as outlined in the following sections. The breakdown is fairly similar to a 4 tier system based on an analysis of 10,000 patients in Italy<sup>31</sup>.

### CATEGORY 1. LOW RISK FOR INTRACRANIAL INJURY

Possible findings are shown in Table 27-9.

In this group, there is an extremely low likelihood of intracranial injury (ICI), even if a skull fracture is present on SXR (incidence of ICI:  $\leq 8.5$  in 10,000 cases with 95% confidence level<sup>30</sup>). NB: this category excludes patients with a history of loss of consciousness.

**Table 27-9 Findings with low risk of ICI**

- asymptomatic
- H/A
- dizziness
- scalp hematoma, laceration, contusion, or abrasion
- no moderate nor high risk criteria (see Table 27-11 and Table 27-13) (no loss of consciousness, etc.)

## Management recommendations

CT scan is not usually indicated. Plain SXR's are not recommended: 99.6% of SXR's in this group are normal. Linear non-displaced skull fractures in this group require no treatment, although in-hospital observation (at least overnight) may be considered.

Patients in this group who meet *Criteria for observation at home* shown in Table 27-12 (disregarding CT scan item) may be managed with observation at home with written head-injury discharge instructions, e.g as illustrated in Table 27-10.

### CATEGORY 2.

#### MODERATE RISK FOR INTRACRANIAL INJURY

Possible findings are shown in Table 27-11.

**Table 27-11 Findings with moderate risk of ICI**

- history of change or loss of consciousness on or after injury
- progressive H/A
- EtOH or drug intoxication
- posttraumatic seizure
- unreliable or inadequate history
- age < 2 yr (unless trivial injury)
- vomiting
- posttraumatic amnesia
- signs of basilar skull fracture
- multiple trauma
- serious facial injury
- possible skull penetration or depressed fracture
- suspected child abuse.
- significant subgaleal swelling<sup>31</sup>

**Table 27-10 Sample discharge instructions for head injuries**

Seek medical attention for any of the following:

- a change in level of consciousness (including difficulty in awakening)
- abnormal behavior
- increased headache
- slurred speech
- weakness or loss of feeling in an arm or leg
- persistent vomiting
- enlargement of one or both pupils (the black round part in the middle of the eye) that does not get smaller when a bright light is shined on it
- seizures (convulsions or fits)
- significant increase in swelling at injury site

Do not take sedatives or pain medication stronger than Tylenol for 24 hours. Do not take aspirin or other anti-inflammatory medications.

**Table 27-12 Criteria for observation at home**

1. normal cranial CT<sup>32</sup>
2. initial GCS  $\geq 14$
3. no high risk criteria (see Table 27-13)
4. no moderate risk criteria (see Table 27-11) except loss of consciousness
5. patient is now neurologically intact (amnesia for the event is acceptable)
6. there is a responsible, sober adult that can observe the patient
7. patient has reasonable access to return to the hospital E/R if needed
8. no "complicating" circumstances (e.g. no suspicion of domestic violence, including child abuse)

## Management recommendations

1. brain CT scan (unenhanced): clinical grounds alone may miss important lesions in this group<sup>32</sup>. 8-46% of patients with minor head injury (MHI) have an intracranial lesion (the most frequent finding was hemorrhagic contusion)<sup>33</sup>
2. SXR: not recommended (see page 859) unless CT scan not available. Useless if normal. A SXR is helpful only if positive (a clinically unsuspected depressed skull fracture might be important)
3. observation
  - A. at home, if the patient meets the criteria outlined in Table 27-12. Provide caregiver with written head-injury discharge instructions (sometimes called "subdural precautions"), as shown in Table 27-10
  - B. in-hospital observation to rule-out neurologic deterioration if patient does not meet criteria in Table 27-12 (including cases where CT scan is not done).

Managing patients with in-hospital observation and only getting a CT scan in cases of deterioration (GCS score  $\leq 13$ ) is as sensitive as CT in detecting intracranial hematomas<sup>33,37</sup>, but is less cost effective than routinely performing an early CT scan and discharging patients who have a normal CT and no other indication for hospitalization<sup>33</sup>

## CATEGORY 3.

### HIGH RISK FOR INTRACRANIAL INJURY

Possible findings are shown in *Table 27-13*.

#### Management recommendations

1. STAT unenhanced brain CT scan
2. admit
3. if there are focal findings, notify operating room to be on standby. For rapid deterioration, consider emergency burr holes (see *Exploratory burr holes*, page 864)
4. determine if intracranial monitor is indicated (see page 868)
5. SXR usually not recommended: a fracture is rarely surprising, and a SXR is inadequate for assessing for intracranial injury. A SXR is possibly useful for localizing a radio-opaque penetrating foreign body (knife blade, bullet...) for the O.R. (omit if significant delay required).

**Table 27-13 Findings with high risk of ICI**

- depressed level of consciousness not clearly due to EtOH, drugs, metabolic abnormalities, postictal, etc.
- focal neurological findings
- decreasing level of consciousness
- penetrating skull injury or depressed fracture

### OTHER RISK FACTORS

#### Occipital vs. frontal fractures

Patients with occipital fractures may be at higher risk of significant intracranial injury (ICI). May be related to the fact that in forward trauma, one may protect oneself with the outstretched arms. Furthermore, the facial bones and air sinuses exert an impact absorbing effect.

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## 27.2.2. Radiographic evaluation

### CT SCANS IN TRAUMA

An unenhanced (i.e. non-contrast) CT scan of the brain usually suffices for patients seen in the emergency department presenting after trauma or with a new neurologic deficit. Enhanced CT or MRI may be appropriate after the unenhanced CT, but are not usually required emergently (exceptions include: significant brain edema due to suspected neoplasm that is not demonstrated without contrast).

The main emergent conditions to rule out (and brief descriptions):

1. blood (hemorrhages or hematomas):
  - A. extra-axial blood: surgical lesions are usually  $\geq 1$  cm maximal thickness
    1. epidural hematoma (EDH) (see page 894): usually biconvex and often due to arterial bleeding
    2. subdural hematoma (SDH) (see page 896): usually crescentic, usually due to venous bleeding. May cover larger surface area than EDH. Chronology of SDH: acute = high density, subacute = isodense, chronic = low density
  - B. subarachnoid blood (SAH): high density spread thinly over convexity and filling sulci or basal cisterns. Trauma is the most common cause of SAH. However, when the history of trauma is not clear, an arteriogram may be indicated to R/O a ruptured aneurysm (possibly precipitating the trauma)
  - C. intracerebral hemorrhage (ICH): increased density in brain parenchyma
  - D. hemorrhagic contusion (see page 893): often "fluffy" inhomogeneous high-density areas within brain parenchyma adjacent to bony prominences (frontal and occipital poles, sphenoid wing). Less well defined than ICH
  - E. intraventricular hemorrhage: present in  $\sim 10\%$  of severe head injuries<sup>38</sup>. Associated with poor outcome; may be a marker for severe injury rather than the cause of the poor outcome. Use of intraventricular rt-PA has been reported for treatment<sup>39</sup> (see page 1130)
2. hydrocephalus: enlarged ventricles may sometimes develop following trauma
3. cerebral swelling: obliteration of basal cisterns (see page 909), compression of ventricles and sulci...
4. evidence of cerebral anoxia: loss of gray-white interface, signs of swelling
5. skull fractures:
  - A. basal skull fractures (including temporal bone fracture)
  - B. orbital blow-out fracture
  - C. calvarial fracture (CT may miss some linear nondisplaced skull fractures)

1. linear vs. stellate
2. open vs. closed
3. diastatic (separation of sutures)
4. depressed vs. nondepressed: CT helps assess need for surgery
6. ischemic infarction: findings are usually minimal or subtle if < 24 hrs since CVA
7. pneumocephalus: may indicate skull fracture (basal or open convexity)
8. shift of midline structures (due to extra- or intra-axial hematomas or asymmetric cerebral edema): shift can cause altered levels of consciousness (*see page 280*)

### Indications for initial brain CT

1. presence of any moderate<sup>40</sup> or high risk criteria (*see Table 27-11 and Table 27-13*) which include: GCS  $\leq$  14, unresponsiveness, focal deficit, amnesia for injury, altered mental status (including those that are significantly inebriated), deteriorating neuro status, signs of basal or calvarial skull fracture
2. assessment prior to general anesthesia for other procedures (during which neurologic exam cannot be followed in order to detect delayed deterioration)

### Follow-up CT

**Routine** follow-up CT (when there is no indication for *urgent* follow-up CT, *see below*):

1. for patients with severe head injuries:
  - A. for stable patients, follow-up CTs are usually obtained between day 3 to 5, (some recommend at 24 hrs also) and again between day 10 to 14
  - B. some recommend routine follow-up CT several hours after the "time zero" CT (i.e. initial CT done within hours of the trauma) to rule-out delayed EDH (*see page 896*), SDH (*see page 899*), or traumatic contusions<sup>41</sup> (*see page 893*)
2. for patients with mild to moderate head injuries:
  - A. with an abnormal initial CT, the CT scan is repeated prior to discharge
  - B. stable patients with mild head injury and normal initial CT do not require follow-up CT

**Urgent** follow-up CT: performed for neurological deterioration (loss of 2 or more points on the GCS, development of hemiparesis or new pupillary asymmetry), persistent vomiting, worsening H/A, seizures or unexplained rise in intracranial pressure (ICP).

### SPINE FILMS

1. cervical spine: must be cleared radiographically from the cranio-cervical junction down through and including the C7-T1 junction. Spinal injury precautions (cervical collar...) are continued until the C-spine is cleared. The steps in obtaining adequate films are outlined in *Spine injuries, Radiographic evaluation and initial C-spine immobilization* on page 938
2. thoracic and lumbosacral spine films should be obtained based on physical findings and on mechanism of injury (*see Spine injuries, Radiographic evaluation and initial C-spine immobilization* on page 938)

### SKULL X-RAYS

A skull fracture increases the probability of a surgical intracranial injury (ICI) (in a comatose patient it is a 20-fold increase, in a conscious patient it is a 400-fold increase<sup>42, 43</sup>). However, significant ICI can occur with a normal SXR (SXR was normal in 75% of minor head injury patients found to have intracranial lesions on CT, attesting to the insensitivity of SXRs<sup>33</sup>). SXRs affect management of only 0.4-2% of patients in most reports<sup>30</sup>.

A SXR may be helpful in the following:

1. in patients with moderate risk for intracranial injury (*see Table 27-11, page 857*) by detecting an unsuspected depressed skull fracture (however, most of these patients will get a CT scan, which obviates the need for SXR)
2. if a CT scan cannot be obtained, a SXR may identify significant findings such as pineal shift, pneumocephalus, air-fluid levels in the air sinuses, skull fracture (depressed or linear)... (however, sensitivity for detecting ICI is very low)
3. with penetrating injuries: helps in visualization of some metallic objects



## MRI SCANS IN TRAUMA

Usually not appropriate for acute head injuries. While MRI is more sensitive than CT, there were no surgical lesions demonstrated on MRI that were not evident on CT<sup>44</sup>.

MRI may be helpful later after the patient is stabilized, e.g. to evaluate brainstem injuries, small white matter changes<sup>45</sup> (e.g. punctate hemorrhages in the corpus callosum seen in diffuse axonal injury, *see page 853*)... Spinal MRI is indicated in patients with spinal cord injuries.

## ARTERIOGRAM IN TRAUMA

Cerebral arteriogram: useful with non missile penetrating trauma (*see page 916*).

## 27.2.3. E/R management specifics

### INITIAL RESUSCITATION

#### PRACTICE GUIDELINE 27-2 IBP AND OXYGENATION

**Level II**<sup>46</sup>: monitor BP and avoid hypotension (SBP < 90 mm Hg)

**Level III**<sup>46</sup>: monitor oxygenation and avoid hypoxia (PaO<sub>2</sub> < 60 mm Hg or O<sub>2</sub> saturation < 90%)

### ADMITTING ORDERS FOR MINOR OR MODERATE HEAD INJURY

#### Admitting orders for minor head injury (GCS ≥ 14)<sup>A</sup><sup>B</sup>

1. activity: BR with HOB elevated 30-45°
2. neuro checks q 2 hrs (q 1 hr if more concerned; consider ICU for these patients). Contact physician for neurologic deterioration
3. NPO until alert; then clear liquids, advance as tolerated
4. isotonic IVF (e.g. NS + 20 mEq KCl/L) run at maintenance: ~ 100 cc/hr for average size adult (peds: 2000 cc/m<sup>2</sup>/d)<sup>C</sup>
5. mild analgesics: acetaminophen (PO, or PR if NPO), codeine if necessary
6. anti-emetic: give infrequently to avoid excessive sedation, avoid phenothiazine anti-emetics (which lower the seizure threshold); e.g. use trimethobenzamide (Tigan®) 200 mg IM q 8 hrs PRN for adults

#### Admitting orders for moderate head injury (GCS 9-13)<sup>B</sup>

1. orders as for minor head injury (*see above*) except patient is kept NPO in case surgical intervention is needed (including ICP monitor)
2. for GCS = 9-12 admit to ICU. For GCS = 13, admit to ICU if CT shows any significant abnormality (hemorrhagic contusions unless very small, rim subdural...)
3. patients with normal or near-normal CTs should improve within hours. Any patient who fails to reach a GCS of 14-15 within 12 hrs should have a repeat CT at that time<sup>40</sup>

### EARLY USE OF PARALYTICS AND SEDATION (PRIOR TO ICP MONITORING)

#### PRACTICE GUIDELINE 27-3 EARLY SEDATION AND PARALYSIS

**Level III**<sup>47</sup>: sedation and neuromuscular blockade (NMB) can be helpful for transporting the head-injured patient, but they interfere with the neuro exam

**Level III**<sup>47</sup>: NMB should be used when sedation alone is inadequate

The routine use of sedatives and paralytics in neurotrauma patients may lead to a

- A. traditionally, mild head injury has been defined as GCS ≥ 13. However, the increased frequency of both surgical lesions and CT scan abnormalities in patients with GCS = 13 suggests that they would be better classified with the *moderate* rather than mild head injuries<sup>32</sup>
- B. *see Indications for CT and admission criteria for TBI on page 856 for admitting criteria*
- C. the concept of "running the patient dry" is considered obsolete (*see page 878*)

higher incidence of pneumonia, longer ICU stays, and possibly sepsis<sup>48</sup>. These agents also impair neurologic assessment<sup>47, 49</sup>. Use should therefore be reserved for cases with clinical evidence of intracranial hypertension (see Table 27-14), or where use is necessary for transport or to permit evaluation of the patient<sup>50</sup>.

**Table 27-14 Clinical signs of IC-HTN\***

1. pupillary dilatation (unilateral or bilateral)
2. asymmetric pupillary reaction to light
3. decerebrate or decorticate posturing (usually contralateral to blown† pupil)
4. progressive deterioration of the neurologic exam not attributable to extracranial factors

\* Items A-C represent clinical signs of herniation. The most convincing clinical evidence of IC-HTN is the witnessed evolution of 1 or more of these signs. IC-HTN may produce a bulging fontanelle in an infant.

† "blown pupil": fixed & dilated pupil

## INTUBATION AND HYPERVENTILATION

**Indications for intubation** in trauma (also see PRACTICE GUIDELINE 27-4):

1. depressed level of consciousness (patient cannot protect airway): usually GCS  $\leq 7$
2. need for hyperventilation (HPV): see below
3. severe maxillofacial trauma: patency of airway tenuous
4. need for pharmacologic paralysis for evaluation or management

### PRACTICE GUIDELINE 27-4 INTUBATION - INDICATIONS

**Level III<sup>51</sup>:** secure the airway (usually by endotracheal intubation) in patients with GCS  $\leq 8$  who are unable to maintain their airway or who remain hypoxic despite supplemental O<sub>2</sub>

**Cautions regarding intubation:**

1. if basal skull fracture through cribriform plate is possible, avoid nasotracheal intubation (to avoid intracranial entry of tube). Use orotracheal intubation
2. prevents assessment of patient's ability to verbalize<sup>49</sup> e.g. for determining Glasgow Coma Scale score
3. risk of pneumonia: see PRACTICE GUIDELINE 27-5 regarding antibiotics

### PRACTICE GUIDELINE 27-5 ANTIBIOTICS FOR INTUBATION

**Level II<sup>52</sup>:** periprocedural antibiotics for endotracheal intubation reduce the risk of pneumonia, but do not alter length of stay or mortality

## Hyperventilation (HPV)

### PRACTICE GUIDELINE 27-6 EARLY/PROPHYLACTIC HYPERVENTILATION

**Level II<sup>53</sup>:** prophylactic hyperventilation (PaCO<sub>2</sub>  $\leq 25$  mm Hg) is not recommended

#### Level III

- hyperventilation (HPV) before ICP monitoring is established should be reserved as a temporizing measure<sup>53</sup> for patients with signs of transtentorial herniation (see Table 27-14) or progressive neurologic deterioration not attributable to extracranial causes<sup>47</sup>
  - HPV should be avoided during the first 24 hrs after TBI (when CBF is often dangerously decreased)<sup>53</sup>
1. since HPV may exacerbate cerebral ischemia, HPV should not be used prophylactically (see page 880)
  2. prior to ICP monitoring, HPV should only be used briefly when CT or clinical signs of IC-HTN are present<sup>50</sup> (see Table 27-14 for clinical signs)
    - A. when appropriate indications are met: HPV to PaCO<sub>2</sub> = 30-35 mm Hg
    - B. HPV should not be used to the point that PaCO<sub>2</sub> < 30 mm Hg (this further reduces CBF but does not necessarily reduce ICP)
  3. acute alkalosis increases protein binding of calcium (decreases ionized Ca<sup>++</sup>). Patients being hyperventilated may develop ionized hypocalcemia with tetany (despite normal total [Ca])

## PRACTICE GUIDELINE 27-7 EARLY USE OF MANNITOL

**Level III**<sup>47,54</sup>: the use of mannitol before ICP monitoring is established should be reserved for patients who are adequately volume-resuscitated with signs of transtentorial herniation (*see Table 27-14*) or progressive neurologic deterioration not attributable to extracranial causes

Indications in E/R (also *see page 882* for more details):

1. evidence of intracranial hypertension (*see Table 27-14*)
2. evidence of mass effect (focal deficit, e.g. hemiparesis)
3. sudden deterioration prior to CT (including pupillary dilatation)
4. after CT, if a lesion that is associated with increased ICP is identified
5. after CT, if going to O.R.
6. to assess "salvageability": in patient with no evidence of brainstem function, look for return of brainstem reflexes

Contraindications:

1. prophylactic administration is not recommended due to its volume-depleting effect. Use only for appropriate indications (*see above*)
2. hypotension or hypovolemia: hypotension can negatively influence outcome<sup>50</sup>. Therefore, when intracranial hypertension (IC-HTN) is present, first utilize sedation and/or paralysis, and CSF drainage. If further measures are needed, fluid resuscitate the patient before administering mannitol. Use hyperventilation in hypovolemic patients until mannitol can be given
3. relative contraindication: mannitol may slightly impede normal coagulation
4. CHF: before causing diuresis, mannitol transiently increases intravascular volume. Use with caution in CHF, may need to pre-treat with furosemide (Lasix®)

**Rx:** bolus with 0.25-1 gm/kg over < 20 min (for average adult: ~ 350 ml of 20% solution). Peak effect occurs in ~ 20 minutes (*see page 882* for follow-up dosing).

## PROPHYLACTIC ANTIEPILEPTIC DRUGS (AEDs)

## PRACTICE GUIDELINE 27-8 PROPHYLACTIC ANTICONVULSANTS AFTER TBI

**Level II**<sup>55-57</sup>: prophylactic phenytoin, carbamazepine, phenobarbital or valproate<sup>58</sup> do not prevent late PTS

**Level II:** AEDs<sup>57</sup> (e.g. phenytoin, valproate, or carbamazepine<sup>55, 56, 58</sup>) may be used to decrease the incidence of early PTS (within 7 days of TBI) in patients at high risk of seizures after TBI (*see Table 27-15*), however, this does not improve outcome

Routine use of prophylactic antiepileptic drugs (AEDs) in traumatic brain injury (TBI) is ineffective in preventing the late development of posttraumatic seizures (PTS) i.e. epilepsy, and has been shown to not be useful except in certain circumstances<sup>55, 56</sup>.

*See page 399* for details on using and discontinuing prophylactic AEDs following TBI. (*Table 27-15* reiterates the markers for patients at increased risk of early PTSs).

## Table 27-15 Conditions with increased risk of posttraumatic seizures

1. acute subdural, epidural, or intracerebral hematoma
2. open-depressed skull fracture with parenchymal injury
3. seizure within the first 24 hrs after injury
4. Glasgow Coma Scale score < 10
5. penetrating brain injury
6. history of significant alcohol abuse
7. ± cortical (hemorrhagic) contusion on CT

## POSTTRAUMATIC SUBARACHNOID HEMORRHAGE

AKA traumatic SAH (tSAH). Trauma is the most common cause of SAH. There is some evidence that nimodipine (Nimotop®) may improve outcome in head-injured patients with subarachnoid blood detected on CT<sup>59</sup>. **Rx:** 60 mg PO or per NG q 4 hrs, hold for hypotension (*see page 1053*). For hydrocephalus after tSAH, *see page 906*.

## PATIENTS WITH ASSOCIATED SEVERE SYSTEMIC INJURIES

Hypotension (defined as a single SBP < 90 mm Hg) doubles mortality, hypoxia (apnea or cyanosis, or  $\text{PaO}_2 < 60$  mm Hg on ABG) also increases mortality<sup>60</sup>, and the combination of both triples mortality and increases the risk bad outcome.

In centers where diagnostic peritoneal lavage (DPL) is used to assess for intra-abdominal hemorrhage, if the initial fluid is not grossly bloody and the patient is hemodynamically stable, the patient should be taken for cranial CT while the remainder of the lavage fluid is collecting for quantitative analysis.

Patients with grossly positive DPL and/or hemodynamic instability may need to be rushed to the O.R. for emergent laparotomy by trauma surgeons without benefit of cerebral CT. These guidelines are offered:

- \* **CAUTION:** many patients with severe trauma may be in DIC (either due to systemic injuries, or directly related to severe head injury possibly because the brain is rich in thromboplastin<sup>61</sup>). Operating on patients in DIC is usually disastrous. At the least, check the PT/PTT
- 1. if neuro-exam is relatively good (i.e. GCS > 8, which implies at least localizing)
  - A. operative neurosurgical intervention is probably not required
  - B. utilize good neuroanesthesia techniques (elevate head of bed, judicious administration of IV fluids, avoiding prophylactic hyperventilation...)
  - C. obtain a head CT scan immediately post-op
- 2. if patient has focal neurologic deficit, an exploratory burr-hole should be placed in the O.R. simultaneously with the treatment of other injuries. Placement is guided by the pre-op deficit (see *Exploratory burr holes*, page 864)
- 3. if there is severe head injury (GCS  $\leq$  8) without localizing signs, or if initial burr hole is negative, or if there is no pre-op neuro exam, then
  - A. measure the ICP: insert a ventriculostomy catheter (if the lateral ventricle cannot be entered after 3 passes, an intraparenchymal fiber-optic monitor or subarachnoid bolt should be used)
    - 1. normal ICP: unlikely that a surgical lesion exists. Manage ICP medically and, if a IVC was inserted, with CSF drainage
    - 2. elevated ICP ( $\geq$  20 mm Hg): inject 3-4 cc of air into ventricles through IVC, then obtain portable intraoperative AP skull x-ray (intra-operative pneumoencephalogram) to determine if there is any midline shift
      - a. mass effect with  $\geq$  5 mm of midline shift is explored<sup>62</sup> with burr-hole(s) on the side opposite the direction of shift
      - b. if no mass effect, intracranial hypertension is managed medically and with CSF drainage
  - B. routine use of exploratory burr holes for children with GCS = 3 has been found not to be justified<sup>63</sup>

### INDIRECT OPTIC NERVE INJURY

~ 5% of head trauma patients manifest an associated injury to some portion of the visual system. Approximately 0.5-1.5% of head trauma patients will sustain *indirect* injury (as opposed to penetrating trauma) to the optic nerve, most often from an ipsilateral blow to the head (usually frontal, occasionally temporal, rarely occipital)<sup>29</sup>. The optic nerve may be divided into 4 segments: intraocular (1 mm in length), intraorbital (25-30 mm), intracanalicular (10 mm), and intracranial (10 mm). The intracanalicular segment is the most common one damaged with closed head injuries. Funduscopic abnormalities visible on initial exam indicates anterior injuries (injury to the intraocular segment (optic disc) or the 10-15 mm of the intraorbital segment immediately behind the globe where the central retinal artery is contained within the optic nerve), whereas posterior injuries (occurring posterior to this but anterior to the chiasm) takes 4-8 weeks to show signs of disc pallor and loss of the retinal nerve fiber layer.

**Treatment**<sup>29</sup>: No prospective study has been carried out. Optic nerve decompression has been advocated for indirect optic nerve injury, however, the results are not clearly better than expectant management with the exception that documented *delayed* visual loss appears to be a strong indication for surgery. Transethmoidal is the accepted route, and is usually done within 1-3 weeks from the trauma<sup>64</sup>. The use of "megadose steroids" may be appropriate as an adjunct to diagnosis and treatment.

### POST-TRAUMATIC HYPOPITUITARISM

Trauma is a rare cause of hypopituitarism. It may follow closed head injury (with or

without basilar skull fracture) or penetrating trauma<sup>65</sup>. In 20 cases in the literature<sup>66</sup> all had deficient growth hormone and gonadotropin, 95% had corticotropin deficiency, 85% had reduced TSH, 63% had elevated PRL. Only 40% had transient or permanent DI.

## 27.2.4. Exploratory burr holes

In a trauma patient, the clinical triad of altered mental status, unilateral pupillary dilatation with loss of light reflex, and contralateral hemiparesis is most often due to upper brainstem compression by uncatal transtentorial herniation which, in the majority of trauma cases, is due to an extraaxial intracranial hematoma. Furthermore, the prognosis of patients with traumatic herniation is poor. Outcome may possibly be improved slightly by increasing the rapidity with which decompression is undertaken, however, an upper limit of salvageability is probably still only ~ 20% satisfactory outcome.

Burr holes are primarily a diagnostic tool, as bleeding cannot be controlled and most acute hematomas are too congealed to be removed through a burr hole. However, if the burr hole is positive, it is possible that modest decompression may be performed, and then the definitive craniotomy can be undertaken incorporating the burr hole(s).

With widespread availability of quickly accessible CT scanning, exploratory burr holes are infrequently indicated.

### INDICATIONS

1. clinical criteria: based on deteriorating neurologic exam. Indications in E/R (rare): patient dying of rapid transtentorial herniation (*see below*) or brainstem compression that does not improve or stabilize with mannitol and hyperventilation<sup>67</sup>.
  - indicators of transtentorial herniation/brainstem compression:
    1. sudden drop in Glasgow Coma Scale (GCS) score
    2. one pupil fixes and dilates
    3. paralysis or decerebration (usually contralateral to blown pupil)
  - recommended situations where criteria should be applied:
    1. neurologically stable patient undergoes witnessed deterioration as described above
    2. awake patient undergoes same process in transport, and changes are well documented by competent medical or paramedical personnel
2. other criteria
  - A. some patients needing emergent surgery for systemic injuries (e.g. positive peritoneal lavage + hemodynamic instability) where there is not time for a brain CT (*see Patients with associated severe systemic injuries*, page 863)

### MANAGEMENT

Controversial. The following should serve only as guidelines:

1. if patient fits the above criteria (emergent operation for systemic injuries or deterioration with failure to improve with mannitol and hyperventilation), and CT scan cannot be performed and interpreted immediately, then treatment should not wait for CT scan
  - A. in general, if the O.R. can be immediately available, burr holes are preferably done there (equipped to handle craniotomy, better lighting and sterility, dedicated scrub nurse...) especially in older patients (> 30 yrs) not involved in MVAs (*see Literature below*). This may more rapidly diagnose and treat extraaxial hematomas in herniating patients, although no difference in outcome has been proven
  - B. if delay in getting to the O.R. is foreseen, emergency burr holes in the E/R should be performed
2. placement of burr-hole(s) as outlined under *Technique below*

### TECHNIQUE

#### Position

Shoulder roll, head turned with side to be explored up. Three pin skull-fixation used if concern about possible aneurysm or AVM (to allow for retractors and increased stability) or if additional stability is desired (e.g. with unstable cervical fractures), otherwise a horse-shoe head-holder suffices and saves time and permits rapid access to the other side.

## Choice of side for initial burr hole

Start with a temporal burr hole (*see below*) on the side:

1. ipsilateral to a blown pupil. This will be on the correct side in > 85% of epidurals<sup>68</sup> and other extra-axial mass lesions<sup>69</sup>
2. if both pupils are dilated, use the side of the first dilating pupil (if known)
3. if pupils are equal, or it is not known which side dilated first, place on side of obvious external trauma
4. if no localizing clues, place hole on left side (to evaluate and decompress the dominant hemisphere)

## Approach

Burr holes are placed along a path that can be connected to form a "trauma flap" if a craniotomy becomes necessary (*see Figure 27-1*). The "trauma flap" is so-called because it provides wide access to most of the cerebral convexity permitting complete evacuation of acute blood clot and control of most bleeding.

First outline the trauma flap with a skin marker:

1. start at the zygomatic arch < 1 cm anterior to the tragus (saves the branch of the facial nerve to the frontalis muscle and the anterior branch of the superficial temporal artery)
2. proceed superiorly and then curve posteriorly at the level of top of the pinna
3. 4-6 cm behind the pinna it is taken superiorly
4. 1-2 cm ipsilateral to the midline (sagittal suture) curve anteriorly to end behind the hairline

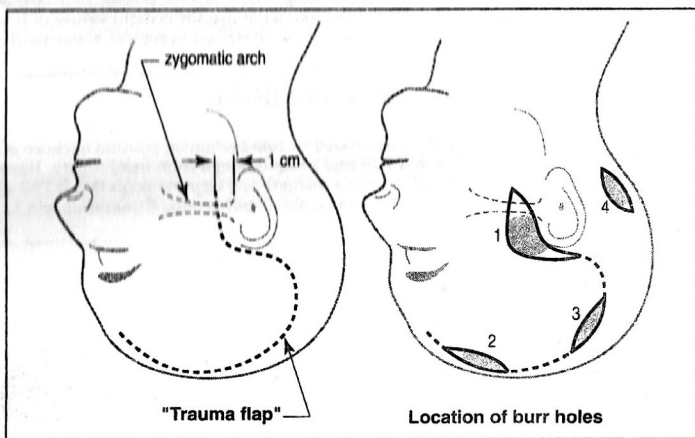


Figure 27-1 Technique to convert burr-hole(s) into trauma flap (adapted<sup>69, 70</sup>)

## Burr hole locations

1. first (temporal) burr-hole: over middle cranial fossa (#1 in *Figure 27-1*) just superior to the zygomatic arch. Provides access to middle fossa (the most common site of epidural hematoma) and usually allows access to most convexity subdural hematomas, as well as proximity to middle meningeal artery in region of pterion
2. if no epidural hematoma, the dura is opened if it has bluish discoloration (suggests subdural hematoma) or if there is a strong suspicion of a mass lesion on that side
3. if completely negative, usually perform temporal burr hole on contralateral side
4. if negative, further burr holes should be undertaken if a CT cannot now be done
5. proceed to ipsilateral frontal burr hole (#2 in *Figure 27-1*)
6. subsequent burr holes may be placed at parietal region (#3 in *Figure 27-1*) and lastly in posterior fossa (#4 in *Figure 27-1*)

## LITERATURE

In 100 trauma patients undergoing transtentorial herniation or brainstem compression<sup>68</sup>, exploratory burr holes (bilateral temporal, frontal and parietal, done in the O.R.) were positive in 56%. Lower rates in younger patients (< 30 yrs) and those in

MVAs (as opposed to falls or assaults). SDH was the most common extraaxial mass lesion (alone and unilateral in 70%, bilateral in 11%, and in combination with EDH or ICH in > 9%).

When burr holes were positive, the first burr hole was on the correct side 86% of the time when placed as above. Six patients had significant extraaxial hematomas missed with exploratory burr holes (mostly due to incomplete burr hole exploration). Only 3 patients had the above neurologic findings as a result of intraparenchymal hematomas.

### Outcome

Mean follow-up: 11 mos (range: 1-37). 70 of the 100 patients died. No morbidity or mortality was directly attributable to the burr holes. Four patients with good outcome and 4 with moderate disability had positive burr holes.

## 27.3. Neuromonitoring

This section considers neuromonitoring instrumentation that can be done primarily at the patient's bedside. The bulk of neuromonitoring literature deals with intracranial pressure (ICP). Other parameters that can be monitored include: jugular venous oxygen monitoring (page 874), regional CBF (page 875), brain tissue oxygen tension (page 874), and brain metabolites (pyruvate, lactate, glucose...) (page 875).

The role of adjunctive monitoring is currently unknown. Unanswered questions include: should neuromonitoring be disease specific (e.g. is SAH different from TBI), which monitors provide additional unique information, what are the critical values of the monitored entity, and what interventions should be undertaken to correct abnormalities?

### 27.3.1. Intracranial pressure (ICP)

Intracranial pressure (ICP) is discussed in this section on trauma because of the close relationship between elevated ICP and brain damage from head injury. However, factors involved in diagnosing and treating intracranial hypertension (IC-HTN) also may pertain (with modifications) to brain tumors, dural venous thrombosis, etc.

#### 27.3.1.1. General information about ICP

#### CEREBRAL PERFUSION PRESSURE (CPP) AND CEREBRAL AUTOREGULATION

Secondary brain injury (i.e. following the initial trauma) is attributable in part to cerebral ischemia (see *Secondary injury*, page 854). The critical parameter for brain function and survival is not actually ICP, rather it is adequate cerebral blood flow (CBF) to meet CMRO<sub>2</sub> demands (for a discussion of CBF & CMRO<sub>2</sub>, see page 1010). CBF is difficult to quantitate, and can only be measured continuously at the bedside with specialized equipment and difficulty<sup>71</sup>. However, CBF depends on cerebral perfusion pressure (CPP), which is related to ICP (which is more easily measured) as shown in Eq 27-1.

$$\left\{ \begin{array}{c} \text{cerebral perfusion} \\ \text{pressure} \end{array} \right\} = \left\{ \begin{array}{c} \text{mean arterial} \\ \text{pressure}^* \end{array} \right\} - \left\{ \begin{array}{c} \text{intracranial} \\ \text{pressure} \end{array} \right\}$$

or, expressed in symbols:

$$CPP = MAP^* - ICP$$

Eq 27-1

\* note: the actual pressure of interest is the **mean carotid pressure (MCP)** which may be approximated as the MAP with the transducer zeroed ~ at the level of the foramen of Monro<sup>72</sup>

Normal adult CPP is > 50 mm Hg. **Cerebral autoregulation** is a mechanism whereby over a wide range, large changes in systemic BP produce only small changes in CBF. Due to autoregulation, CPP would have to drop below 40 in a normal brain before CBF would be impaired.

In the head injured patient, recent evidence suggests that elevated ICP (≥ 20 mm Hg) may be more detrimental than changes in CPP (as long as CPP is > 60 mm Hg<sup>73</sup>)<sup>18</sup> (higher levels of CPP were not protective against significant ICP elevations<sup>18</sup>).

## INTRACRANIAL PRESSURE

The following are approximations to help simplify understanding ICP (these are only models, and as such are not entirely accurate):

1. **normal** intracranial constituents (and approximate volumes):
  - A. brain parenchyma (which also contains extracellular fluid): 1400 ml
  - B. cerebral blood volume (CBV): 150 ml
  - C. cerebrospinal fluid (CSF): 150 ml
2. these volumes are contained in an inelastic, completely closed skull
3. pressure is distributed evenly throughout the intracranial cavity
4. the modified **Monro-Kellie** doctrine<sup>74</sup> states that the sum of the intracranial volumes (CBV, brain, CSF, and other constituents (e.g. tumor, hematoma...)) is constant, and that an increase in any one of these must be offset by an equal decrease in another
- ◆ the mechanism: there is a pressure equilibrium in the skull. If the pressure from one intracranial constituent increases (as when that component increases in volume), it causes the pressure inside the skull (ICP) to increase. When this increased ICP exceeds the pressure required to force one of the other constituents out through the foramen magnum (FM) (the only true effective opening in the intact skull) that other component will decrease in size via that route until a new equilibrium is established. The craniospinal axis can buffer small increases in volume with no change or only a slight increase in ICP. If the expansion continues, then the new equilibrium will be at a higher ICP. The result:
  - at pressures slightly above normal, if there is no obstruction to CSF flow (obstructive hydrocephalus), CSF can be displaced from the ventricles and subarachnoid spaces and exit the intracranial compartment via the FM
  - intravenous blood can also be displaced through the FM via the IJVs
  - as pressure continues to rise, arterial blood is displaced and CPP decreases, eventually producing diffuse cerebral ischemia. At pressures equal to mean arterial pressure, arterial blood will be unable to enter the skull through the FM, producing complete cessation of blood flow to the brain, with resultant massive infarction
  - increased brain edema, or an expanding mass (e.g. hematoma) can push brain parenchyma downward into the foramen magnum (cerebral herniation) although brain tissue cannot actually exit the skull

### NORMAL ICP

The normal range of ICP varies with age. Values for pediatrics are not well established. Guidelines are shown in Table 27-16.

Table 27-16 Normal ICP

Age group	Normal range (mm Hg)
adults and older children*	< 10-15
young children	3-7
term infants†	1.5-6

\* the age of transition from "young" to "older" child is not precisely defined

† may be subatmospheric in newborns<sup>75</sup>

### INTRACRANIAL HYPERTENSION (IC-HTN)

Traumatic IC-HTN may be due any of the following (alone or in various combinations):

1. cerebral edema
2. hyperemia: the normal response to head injury<sup>76</sup>. Possibly due to vasomotor paralysis (loss of cerebral autoregulation). May be more significant than edema in raising ICP<sup>17</sup> (see page 902)
3. traumatically induced masses
  - A. epidural hematoma
  - B. subdural hematoma
  - C. intraparenchymal hemorrhage (hemorrhagic contusion)
  - D. foreign body (e.g. bullet)
  - E. depressed skull fracture
4. hydrocephalus due to obstruction of CSF absorption or circulation
5. hypoventilation (causing hypercarbia → vasodilatation)
6. systemic hypertension (HTN)
7. venous sinus thrombosis
8. increased muscle tone and valsalva maneuver as a result of agitation or posturing → increased intrathoracic pressure → increased jugular venous pressure → reduced venous outflow from head
9. sustained posttraumatic seizures (status epilepticus)

A **secondary increase in ICP** is sometimes observed 3-10 days following the trauma, and may be associated with a worse prognosis<sup>77</sup>. Possible causes include:



1. delayed hematoma formation
  - A. delayed epidural hematoma: *see page 896*
  - B. delayed acute subdural hematoma: *see page 899*
  - C. delayed traumatic intracerebral hemorrhage<sup>41</sup> (or hemorrhagic contusions) with perilesional edema: usually in older patients, may cause sudden deterioration. May be severe enough to require evacuation (*see page 893*)
2. cerebral vasospasm<sup>78</sup>
3. severe adult respiratory distress syndrome (ARDS) with hypoventilation
4. delayed edema formation: more common in pediatric patients
5. hyponatremia

### Indications to treat IC-HTN

Various cutoff values are used at different centers above which treatment measures for intracranial hypertension (IC-HTN) are initiated. Although 15, 20 and 25 have been quoted, most centers use ICP  $\geq 20$ -25 mm Hg as the upper limit<sup>50</sup>. There is high mortality and worse outcome<sup>18</sup> among patients with ICP persistently  $> 20$  compared to 20% in those where ICP could be kept  $< 20$ <sup>79</sup>. Better control may be possible by treating early rather than waiting and trying to control higher ICPs or when plateau waves occur<sup>27</sup>.

"Deadly" ICP (in adult), i.e. likely to be fatal if uncontrolled:  $> 25$ -30 mm Hg.

### Cushing's triad

Cushing's triad is shown in Table 27-17, and may be seen with IC-HTN regardless of cause. However, the full triad is only seen in  $\approx 33\%$  of cases of IC-HTN.

**Table 27-17 Cushing's triad**

- A. hypertension
- B. bradycardia
- C. respiratory irregularity

### CT scan and elevated ICP

Whereas CT findings may be correlated with a risk of IC-HTN, no combination of CT findings has been shown to allow accurate estimates of actual ICP. 60% of patients with closed head injury and an abnormal CT<sup>A</sup> will have IC-HTN<sup>80</sup>.

Only 13% of patients with a normal CT scan will have IC-HTN<sup>80</sup>. However, patients with a normal CT AND 2 or more risk factors identified in Table 27-18 will have  $\approx 60\%$  risk of IC-HTN. If only 1 or none are present, ICP will be increased in only 4%.

**Table 27-18 Risk factors for IC-HTN with a normal CT**

- age  $> 40$  yrs
- SBP  $< 90$  mm Hg
- decerebrate or decorticate posturing on motor exam (unilateral or bilateral)

## 27.3.1.2. ICP monitoring

### INDICATIONS FOR ICP MONITORING

#### PRACTICE GUIDELINE 27-9 INDICATIONS FOR ICP MONITORING

For salvageable patients with severe traumatic brain injury (GCS  $\leq 8$  after cardiopulmonary resuscitation)

**Level II**<sup>82</sup>: with an abnormal admitting brain CT<sup>A</sup>

**Level III**<sup>82</sup>: with a normal admitting brain CT, but with  $\geq 2$  of the risk factors for IC-HTN in Table 27-18

- ★ 1. neurologic criteria: *see PRACTICE GUIDELINE 27-9* above
- some centers monitor patients who don't follow commands. Rationale: patients who follow commands (GCS  $\geq 9$ ) are at low risk for IC-HTN, and one can follow sequential neurologic exams in these patients and institute further evaluation or treatment based on neurologic deterioration
  - some monitor patients who don't localize, and follow neuro exam on others
2. multiple systems injured with altered level of consciousness (especially where therapies for other injuries may have deleterious effects on ICP, e.g. high levels of PEEP or the need for large volumes of IV fluids or the need for heavy sedation)
3. with traumatic intracranial mass (EDH, SDH, depressed skull fracture...)
- A. a physician may choose to monitor ICP in some of these patients<sup>81, 83</sup>
  - B. post-op, subsequent to removal of the mass

A. "abnormal" CT: demonstrates hematomas (EDH, SDH or ICH), contusions<sup>80</sup>, compression of basal cisterns (*see page 909*), herniation or swelling<sup>81, 82</sup>

4. non-traumatic indications for ICP monitoring:
  - A. some centers monitor ICP in patients with acute fulminant liver failure with an INR > 1.5 and Grade III of IV coma. A recent study shows that a subarachnoid bolt may be inserted after administration of factor VII 40 mcg/kg IV over 1-2 minutes (the bold is inserted as soon as possible (usually within 15 minutes and no more than 2 hours after administration)) without significant risk of hemorrhage. All patients were treated with hypothermia; other ICP treatment measures were used for refractory IC-HTN

### CONTRAINDICATIONS (RELATIVE)

1. "awake" patient: monitor usually not necessary, can follow neuro exam
2. coagulopathy (including DIC): frequently seen in severe head injury. If an ICP monitor is essential, take steps to correct coagulopathy (FFP, platelets...) and consider subarachnoid bolt or epidural monitor (an IVC or intraparenchymal monitor is contraindicated) (for recommended range of PT or INR, see page 37)

### DURATION OF MONITORING

D/C monitor when ICP normal x 48-72 hrs after withdrawal of ICP therapy. Caution: IC-HTN may have delayed onset (often starts on day 2-3, and day 9-11 is a common second peak especially in peds). Also see *Delayed deterioration*, page 854. Avoid a false sense of security imparted by a normal early ICP.

### COMPLICATIONS OF ICP MONITORS

See Table 27-19 for a summary of complication rates for various types of monitors<sup>50</sup>.

1. infection: see below
2. hemorrhage<sup>50</sup>: overall incidence is 1.4% for all devices (see Table 27-19 for breakdown). Risk of significant hematoma requiring surgical evacuation is ~ 0.5-2.5%<sup>80, 86, 87</sup>
3. malfunction or obstruction: with fluid coupled devices, higher rates of obstruction occur at ICPs > 50 mm Hg
4. malposition: 3% of IVCs require operative repositioning

Table 27-19 Complication rates with various types of ICP monitors

Monitor type	Bacterial colonization*	Hemorrhage	Malfunction or obstruction
IVC	ave: 10-17% range <sup>84, 85</sup> : 0-40%	1.1%	6.3%
subarachnoid bolt	ave: 5% range: 0-10%	0	16%
subdural	ave: 4% range: 1-10%	0	10.5%
parenchymal	ave: 14% (two reports, 12% & 17%)	2.8%	9-40%

\* some studies report this as infection, but do not distinguish between clinically significant infection and colonization of ICP monitor

### INFECTION WITH ICP MONITORS

Colonization of the monitoring device is much more common than clinically significant infection (ventriculitis or meningitis). See Table 27-19 for colonization rates. Fever, leukocytosis and CSF pleocytosis have low predictive value (CSF cultures are more helpful). Range of reported infection rates: 1-27%<sup>88</sup>.

#### PRACTICE GUIDELINE 27-10 INFECTION PROPHYLAXIS WITH ICP MONITORS

**Level III<sup>82</sup>:** neither prophylactic antibiotics nor routine ventricular catheter exchange is recommended to reduce infection

Identified risk factors for infection include<sup>85, 88-90</sup>:

1. intracerebral, subarachnoid or intraventricular hemorrhage
2. ICP > 20 mm Hg
3. duration of monitoring: contradictory results in literature. One study found an increased risk with monitor duration > 5 days (infection risk reaches 42% by day #11)<sup>86, 89</sup>. Another found no correlation with monitoring duration<sup>91</sup>. A analysis<sup>85</sup> found a non-linear increase of risk during the first 10-12 days after which the rate diminished rapidly
4. neurosurgical operation: including operations for depressed skull fracture

5. irrigation of system
6. leakage around IVCs
7. open skull fractures (including basilar skull fractures with CSF leak)
8. other infections: septicemia, pneumonia

Factors not associated with increased incidence of infection:

1. insertion of IVC in neuro intensive care unit (instead of O.R.)
2. previous IVC
3. drainage of CSF
4. use of steroids

Treatment of infection:

Removal of device if at all possible (if continued ICP monitoring is required consideration may be given to inserting a monitor at another site) and appropriate antibiotics.

## TYPES OF MONITORS

1. **intraventricular catheter (IVC):** AKA external ventricular drainage (**EVD**), connected to an external pressure transducer via fluid-filled tubing. The standard by which others are judged (also see *Intraventricular catheter (IVC)* below)<sup>A</sup>
  - A. advantages:
    1. most accurate (can be recalibrated to minimize measurement drift)<sup>92</sup>
    2. lower cost
    3. in addition to measuring pressure, allows therapeutic CSF drainage
  - B. disadvantages
    1. may be difficult to insert into compressed or displaced ventricles
    2. obstruction of the fluid column (e.g. by blood clot) may cause inaccuracy
    3. some effort is required to check and maintain function (e.g. see *IVC problems*, page 873 and *IVC trouble shooting* on page 873)
    4. transducer must be consistently maintained at a fixed reference point relative to patient's head (must be moved as HOB is raised/lowered)
2. **intraparenchymal monitor** (e.g. Camino labs or Honeywell/Phillips<sup>93, 94</sup>): similar to IVC but more expensive. Some are subject to measurement drift<sup>95, 96</sup>, others may not be<sup>97</sup>
3. less accurate monitors
  - A. **subarachnoid screw** (bolt): risk of infection 1%, rises after 3 days. At high ICPs (often when needed most) surface of brain may occlude lumen → false readings (usually lower than actual, may still show = normal waveform)
  - B. **subdural**: may utilize a fluid coupled catheter (e.g. Cordis Cup catheter), fiberoptic tipped catheter, or strain gauge tipped catheter
  - C. **epidural**: may utilize a fluid coupled catheter, or fiberoptic tipped catheter (e.g. Ladd fiberoptic). Accuracy is questionable
  - D. in infants, one can utilize an open anterior fontanelle (**AF**):
    1. **fontanometry**<sup>98</sup>: probably not very accurate
    2. **aplanation principle**: may be used in suitable circumstances (viz.: if the fontanelle is concave with the infant upright, and convex when flat or head down) to estimate the ICP within 1 cm H<sub>2</sub>O<sup>75</sup>. The infant is placed supine, and the AF is visualized and palpated while the head is raised and lowered. When the AF is flat, the ICP equals atmospheric pressure, and ICP can be estimated in cm H<sub>2</sub>O as the distance from the AF to the point where the venous pressure is 0 (for a recumbent infant, the midpoint of the clavicle usually suffices). If the AF is not concave with the infant erect, then this method cannot be used because either the ICP exceeds the distance from the AF to the venous zero point, or the scalp may be too thick

**Conversion factors:** between mm Hg and cm H<sub>2</sub>O are shown in *Eq 27-2* and *Eq 27-3* (the density of mercury is 13.6 times that of water, and CSF is fairly close to water).

$$1 \text{ mm Hg (torr)} = 1.36 \text{ cm H}_2\text{O}$$

**Eq 27-2**

$$1 \text{ cm H}_2\text{O} = 0.735 \text{ mm Hg (torr)}$$

**Eq 27-3**

A. other options for IVCs utilize transducers tipped with fiberoptic or strain gauge devices which are located within the intraventricular catheter; in this discussion, "IVC" does not refer to this type

## INTRAVENTRICULAR CATHETER (IVC)

See *Types of monitors* above for some basic information.

### Insertion technique

For technique to place catheter in frontal horn, see *Kocher's point* on page 207. The right side is usually used unless specific reasons to use the left are present (e.g. blood clot in right lateral ventricle which might occlude IVC).

### Set-up

Figure 27-2 shows a typical external ventricular drainage (EVD) system/ventriculostomy ICP monitor. Not every system will have the same components. Note that the effect of having an opening on the top of the drip chamber (through an air-filter) is the same as having the drip nozzle open to air, and therefore as long as this filter is not wet or plugged the pressure in the IVC is regulated by the height of the nozzle (as read on the pressure scale; note that the "0" is level with the nozzle).

The external auditory canal (EAC) is often used as a convenient external landmark for "0" (approximates the level of the foramen of Monro). In Figure 27-2 the drip chamber is illustrated at 8 cm above the EAC.

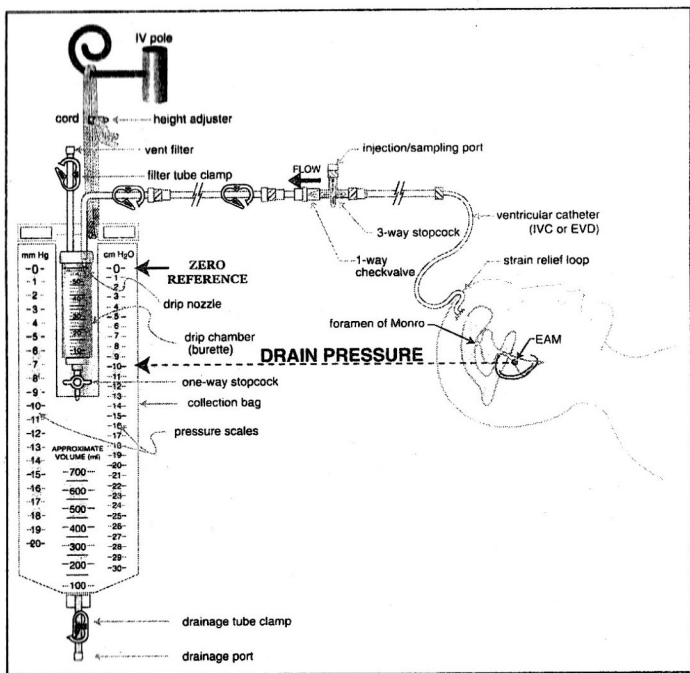


Figure 27-2 Medtronic® ventricular drainage system/ICP monitor

## TYPES OF ICP WAVEFORMS

### NORMAL WAVEFORMS

The normal ICP waveform (as occurs with normal blood pressure and in the absence of IC-HTN) as illustrated in Figure 27-3 is rarely seen since ICP is usually monitored only when it is elevated. The origin of the variations seen in the normal tracing is somewhat in dispute. One explanation describes these two types of waveforms<sup>90</sup>:

1. small pulsations transmitted from the systemic blood pressure to the intracranial cavity
  - A. large (1-2 mm Hg) peak corresponding to the arterial systolic pressure

- 
- The diagram illustrates the relationship between respiration, ICP, CVP, and A-line. At the top, a smooth curve represents the respiratory cycle, with peaks labeled 'inspiration' and troughs labeled 'expiration'. A bracket on the right side of this curve is labeled 'respiration'. Below this, a jagged line represents the ICP waveform, which shows a clear sawtooth pattern corresponding to the respiratory cycle. A dashed line connects a portion of the ICP waveform to a circular inset. Inside the inset, three waveforms are shown: CVP (Cardiac Venous Pressure), ICP (Intracranial Pressure), and A-line (Arterial Pressure). The CVP waveform shows a regular, rhythmic pattern. The ICP waveform shows a sawtooth pattern. The A-line waveform shows a regular, rhythmic pattern. A label 'A' wave' points to a specific peak in the CVP waveform. Vertical dashed lines connect the peaks of the CVP waveform to the peaks of the ICP waveform, showing a temporal relationship between the two.

any time there is a change in: ICP (increase or decrease), neuro exam, or CSF output (for systems open to drainage).

1. check for presence of good waveform with respiratory variations and transmitted pulse pressures
2. IVCs: to check for patency, open the system to drain and lower the drip chamber below level of head and observe for 2-3 drops of CSF (normally do not allow more than this to drain)
3. for systems open to drainage:
  - A. volume of CSF in drip chamber should be indicated every hour with a mark on a piece of tape on the drip chamber, and the volume should increase with time unless ICP is less than the height of the drip chamber (under these circumstances the system would usually not be left open to drainage).  
NB: the maximum expected output from a ventriculostomy would be ~ 450-700 ml per day in a situation where none of the produced CSF is absorbed by the patient. This is not commonly encountered. A typical amount of drainage would be ~ 75 ml every 8 hrs
  - B. drip chamber should be emptied into drainage bag regularly (e.g. q 4 or 8 hours) and any time the chamber begins to get full (record volume)
4. in cases where there is a question whether the monitor is actually reflecting ICP, lowering the HOB towards 0° should increase ICP. Gentle pressure on both jugular veins simultaneously should also cause a gradual rise in ICP over 5-15 seconds that should drop back down to baseline when the pressure is released

### IVC problems

The following represents some of the error or pitfalls that commonly occur with external ventricular drainage. Some also apply to ICP monitoring in general.

1. air filter on drip chamber gets wet (prevents air from passing through filter)
  - A. result: fluid cannot drain freely into drip chamber (the pressure is no longer regulated by the height of the drip nozzle)
    1. if the outflow from the drip chamber is clamped, then no flow at all
    2. if the clamp on the drip chamber outlet is open, then the pressure is actually regulated by the height of the nozzle in the collection bag and not the nozzle in the drip chamber
  - B. solution: if a fresh filter is available, then replace the wet one. Otherwise one must improvise (with the risk of exposing the system to contamination): e.g. replace the wet filter with a filter from an IV set, or with a sterile gauze taped over the opening
2. air filter on collection bag gets wet: this will make it difficult to empty the drip chamber into the bag
  - A. this is not usually an urgent problem unless the drip chamber is full and the collection bag is distended tensely with air
  - B. the filter will dry out with time and will usually start to work again
  - C. if it is necessary to empty the drip chamber before the filter is dry, then using sterile technique insert a needle into the bag drainage port and decompress the bag of fluid and air
3. improper connections: a pressurized irrigation bag with or without heparinized solution should never be connected to an ICP monitor
4. changing position of head of bed: must move drip chamber up or down to keep it level with the same external landmarks (e.g. level of auditory canal):
  - A. when open to drainage, this will assure the correct pressure will be maintained
  - B. when opened to pressure transducer, will maintain correct zero
5. when open to drain, pressure reading from transducer is not meaningful: the pressure cannot exceed the height of the drip chamber in this situation
6. drip chamber falls to floor:
  - A. overdrainage, possible seizures and/or subdural hematoma formation
  - B. solution: securely tape chamber to pole, bed-rail..., check position regularly

### IVC trouble shooting

See also *IVC problems* above.

1. IVC no longer works
  - A. manifestation of problem:
    1. dampening or loss of normal waveform
    2. no fluid drains into drip chamber (applies only when catheter has been opened to drain)
  - B. possible causes:

1. occlusion of catheter proximal to transducer
  - a. slide clamp closed or stopcock closed
  - b. catheter occluded by brain particles, blood cells, protein
2. IVC pulled out of ventricle
- C. test: temporarily lower drip nozzle and watch for 2-3 drops CSF
- D. solution:
  1. verify all clamps are open
  2. flush no more than 1.5 ml of non-bacteriostatic saline (AKA preservative-free saline) with very gentle pressure into ventricular catheter (NB: in elevated ICP the compliance of the brain is abnormally low and small volumes can cause large pressure changes)
    - a. if no return then brain or clot is probably plugging catheter. If it is known that the ventricles are ~ completely collapsed then the IVC may be OK and CSF should still drain over time. Otherwise this is a non-functioning catheter, and if a monitor/drain is still indicated then a new catheter may need to be inserted (CT may be considered first if the status of the ventricles is not known). If catheter is clotted by intraventricular hemorrhage, rt-PA may sometimes be used<sup>39</sup> (see page 1130)
2. ICP waveform dampened
  - A. possible causes:
    1. occlusion of catheter proximal to transducer: *see above*
    2. IVC pulled out of ventricle: no fluid will drain
    3. air in system:
      - a. solution: allow CSF to drain and expel air
      - b. caution: do not allow excessive amount of CSF to drain (may allow obstruction of catheter, subdural formation...). Do not inject fluid to flush air into brain
    4. following decompressive craniectomy: due to the fact that the monitor is no longer in a closed space, this is a normal finding in this setting

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## 27.3.2. Adjuncts to ICP monitoring

### JUGULAR VENOUS OXYGEN MONITORING

Parameters related to oxygen content of the blood in the jugular veins are global in nature and are insensitive to focal pathology. Requires retrograde placement of catheter near to the origin of the internal jugular vein at the base of the skull. Parameters that can be measured:

1. jugular venous oxygen saturation ( $SjVO_2$ ): measured continuously with special fiberoptic catheter. Normal  $SjVO_2$ :  $\geq 60\%$ . Desaturations to  $< 50\%$  suggest ischemia. Multiple desaturations ( $< 50\%$ ), or sustained ( $\geq 10$  minutes) or profound desaturation episodes are associated with poor outcome<sup>101, 102</sup>. Sustained desaturations should prompt an evaluation for correctable etiologies: kinking of jugular vein, anemia, increased ICP, poor catheter position, CPP  $< 60$  mm Hg, vasospasm, surgical lesion,  $PaCO_2 < 28$  mm Hg. High  $SjVO_2 > 75\%$  may indicate hyperemia or infarcted tissue and is also associated with poor outcome<sup>103</sup>
2. jugular vein oxygen content ( $CVO_2$ ). Requires intermittent sampling of blood
3. **arterial-jugular venous oxygen content difference ( $AVdO_2$ )**<sup>104</sup>:  $AVdO_2 > 9$  ml/dl (vol%) probably indicates global cerebral ischemia<sup>105, 106</sup>, while values  $< 4$  ml/dl indicate cerebral hyperemia<sup>107</sup> ("luxury perfusion" in excess of the brain's metabolic requirement<sup>106</sup>)

### BRAIN TISSUE OXYGEN TENSION MONITORING ( $P_{Bt}O_2$ )

Monitored e.g. with Licox® probe. The likelihood of death increases with longer times of brain tissue oxygen tension ( $P_{Bt}O_2$ )  $< 15$  mm Hg or even a brief drop of  $P_{Bt}O_2 < 6$ <sup>108</sup>. Initial  $P_{Bt}O_2 < 10$  mm Hg for  $> 30$  minutes correlates with increased risk of death or bad outcome<sup>109</sup>. Also, see *PRACTICE GUIDELINE 27-13*.

Probe placement:

- TBI: assumed to be a diffuse process, often placed on least injured side

- SAH: placed in vascular distributions at greatest risk of vasospasm
  - A. ACA (with ACA or a-comm aneurysm): standard frontal placement ( $\approx$  2-3 cm off midline on appropriate side)
  - B. MCA (with ICA or MCA aneurysm): 4.5-5.5 cm off midline
  - C. ACA-MCA watershed area: 3 cm lateral to midline
- ICH: usually placed near the site of the hemorrhage

Effect of  $p_{\text{Bt}}\text{O}_2$  monitoring/intervention on outcome: no randomized studies

1. in TBI<sup>110</sup>: goal was to maintain  $p_{\text{Bt}}\text{O}_2 > 25$  mm Hg. Adding  $p_{\text{Bt}}\text{O}_2$  monitoring resulted in improved outcome. May have been result of increased attentiveness ("Hawthorne effect")
2. in SAH<sup>111</sup>: a moving correlation coefficient (ORx) between CPP and  $p_{\text{Bt}}\text{O}_2$  was used to label high ORx as disturbed autoregulation, and this value on post SAH days 5 & 6 had predictive value for delayed infarction

Management suggestions for  $p_{\text{Bt}}\text{O}_2 < 15$ -20 mmHg:

1. consider jugular venous  $\text{O}_2$  saturation monitor or lactate microdialysis monitor for confirmation
2. consider CBF study to determine generalizability of  $p_{\text{Bt}}\text{O}_2$  monitor reading
3. treatment: proceed to each tier as needed
  - tier 1
    1. keep body temperature  $< 37.5^\circ\text{C}$
    2. increase CPP to  $> 60$  mmHg (use fluids preferentially to pressors until CVP  $> 8$  cm  $\text{H}_2\text{O}$ , then use pressors)
  - tier 2
    1. increase  $\text{FiO}_2$  to 60%
    2. increase  $\text{paCO}_2$  to 45-50 mmHg
    3. transfuse PRBCs until Hgb  $> 10$  g/dL
  - tier 3
    1. increase  $\text{FiO}_2$  to 100%
    2. consider increasing PEEP to increase  $\text{PaO}_2$  if  $\text{FiO}_2$  is at 100%
    3. decrease ICP to  $< 10$  mmHg (drain CSF, mannitol, sedation...)

## BEDSIDE MONITORING OF REGIONAL CBF (rCBF)

Thermal diffusion flowmetry permits continuous rCBF monitoring by assessing thermal convection due to tissue blood flow. The probe tip is inserted into the white matter of the brain. Commercially available systems include Hemedex® monitoring system (Codman) utilizing the QFLOW 500® probe which is ~~is~~ not MRI compatible.

Probe placement: issues similar to those discussed for  $p_{\text{Bt}}\text{O}_2$  (see above).

Readout:

1. **K value** (thermal conduction): range for white matter is 4.9-5.8 mW/cm $^\circ\text{C}$  (the monitor suppresses CBF readings if the K value is outside this range)
  - A.  $K < 4.9$ : the probe tip is probably out of the brain tissue or white matter - the probe should be advanced 1-2 mm
  - B.  $K > 5.8$  the tip is probably too deep, near a blood vessel, or in the ventricle or epidural or subdural space - the probe should be retracted 1-2 mm
2. **CBF**
  - A. normal white matter: 18-25 ml/100g-min
    1. white matter CBF  $< 15$ : may indicate vasospasm or ischemia
    2. white matter CBF  $< 10$ : may indicate infarction
  - B. normal gray matter: 67-80 ml/100g-min

Observational data: in a small study of SAH (n=5) and TBI (n=3)<sup>112</sup> there was good correlation between rCBF and  $p_{\text{Bt}}\text{O}_2$  91% of the time. Monitoring was not possible 36% of the time due to patient fever (wherein the system prevents monitoring).

## CEREBRAL MICRODIALYSIS

Compounds assayed include: lactate, pyruvate, lactate/pyruvate ratio, glucose, glutamate, urea and electrolytes including  $\text{K}^+$  & calcium. Some observational data:

1. lactate levels increase during episodes of SjVO $_2$  desaturation<sup>113</sup>
2. decreased extracellular glucose was associated with increased mortality<sup>114</sup>



## 27.3.3. Treatment measures for elevated ICP

This section presents a general protocol for treating documented (or sometimes clinically suspected) intracranial hypertension (IC-HTN). Guidelines promulgated by the Brain Trauma Foundation<sup>46, 50, 115, 116</sup> are generally followed. Unless otherwise stated, guidelines are for adult patients ( $\geq 18$  years age).

### TREATMENT THRESHOLDS

#### Intracranial pressure treatment thresholds

The optimal ICP at which to begin treatment is not known. Generally accepted level: ICP  $\geq 20$ -25 mm Hg<sup>117</sup>. **PRACTICE GUIDELINE 27-11** shows the Brain Trauma Foundation guideline. \* Caution: patients can herniate even at ICP  $< 20$ <sup>118</sup> (depends on location of intracranial mass).

#### PRACTICE GUIDELINE 27-11 ICP TREATMENT THRESHOLD

**Level II**<sup>117</sup>: treatment for IC-HTN should be initiated for ICP  $> 20$  mm Hg

**Level III**<sup>117</sup>: the need for treatment should be based on ICP in combination with clinical examination & brain CT findings

#### Cerebral perfusion pressure (CPP)

The optimal value for CPP has yet to be determined. The threshold for ischemia is in the 50-60 mm Hg range. Because of systemic toxicity, paradigms of maintaining CPP  $> 70$  mm Hg have been superseded. **PRACTICE GUIDELINE 27-12** outlines current recommendations regarding CPP.

#### PRACTICE GUIDELINE 27-12 CEREBRAL PERFUSION PRESSURE ISSUES

**Level II**<sup>119</sup>: \* avoid aggressive use of fluids and pressors to maintain CPP  $> 70$  mm Hg (because of risk of adult respiratory distress syndrome (ARDS))

**Level III**<sup>119</sup>: \* avoid CPP  $< 50$  mm Hg\*

**Level III**: ancillary monitoring of CBF, oxygenation or metabolism assists CPP management

\* in order to avoid CPP  $< 50$  mm Hg, it may be best to start treatment when CPP falls  $< 60$ <sup>119, 120</sup>

#### Brain oxygenation parameters

Suggestions for treatment thresholds are shown in **PRACTICE GUIDELINE 27-13**. It remains to be determined which interventions are useful to achieve this, and whether this improves outcome.

#### PRACTICE GUIDELINE 27-13 BRAIN OXYGEN MONITORING

**Level III**<sup>121</sup>: jugular venous O<sub>2</sub> saturation  $< 50\%$  or brain tissue oxygen tension (p<sub>Bt</sub>O<sub>2</sub>)  $< 15$  mm Hg are treatment thresholds

### ICP MANAGEMENT PROTOCOL

Table 27-20 summarizes a protocol (see *Measures to lower ICP* below for details).

Dosages are given for an average adult, unless specified as mg/kg. Treatment may be initiated prior to insertion of a monitor if there is acute neurologic deterioration or clinical signs of IC-HTN, but continued treatment requires documentation of persistent IC-HTN.

For persistent IC-HTN consider "second tier" therapies shown on page 879.

Additional measures which may be used to treat an acute ICP crisis are shown in Table 27-21.

**Table 27-20 Summary of measures to control IC-HTN\***

Goals: keep ICP < 20 mm Hg, and CPP  $\geq$  50 mm Hg<sup>117, 119</sup>

Step	Rationale/Remedy
<b>GENERAL MEASURES (should be utilized routinely)</b>	
elevate HOB to 30-45°	↓ ICP by enhancing venous outflow, but also reduces mean carotid pressure → no net change in CBF
keep neck straight, avoid tight trach tape	constriction of jugular venous outflow causes ↑ ICP
avoid hypotension (SBP < 90 mm Hg)	<ul style="list-style-type: none"> <li>normalize intravascular volume</li> <li>use pressors if needed</li> </ul>
control hypertension if present	<ul style="list-style-type: none"> <li>nitroprusside if not tachycardic</li> <li>beta-blocker if tachycardic (labetalol, esmolol...)</li> <li>avoid overtreatment → hypotension</li> </ul>
avoid hypoxia (PaO <sub>2</sub> < 60 mm Hg or O <sub>2</sub> sat < 90%) (maintain airway and adequate oxygenation)	hypoxia may cause further ischemic brain injury
ventilate to normocarbica (PaCO <sub>2</sub> = 35-40 mm Hg)	avoid prophylactic hyperventilation (page 880)
light sedation: e.g. codeine 30-60 mg IM q 4 hrs PRN	(same as heavy sedation, see below)
controversial: prophylactic hypothermia. If used, hold at target temp > 48 hrs	↓ CMRO <sub>2</sub> - efficacy not rigorously proven (page 883)
unenhanced head CT scan for ICP problems†	rule out surgical condition
<b>SPECIFIC MEASURES FOR IC-HTN</b>	
(proceed to successive steps if documented IC-HTN persists - each step is <b>ADDED</b> to the previous measure)	
heavy sedation (e.g. fentanyl 1-2 ml or MSO <sub>4</sub> 2-4 mg IV q 1 hr) and/or paralysis (e.g. vecuronium 8-10mg IV)	reduces elevated sympathetic tone and HTN induced by movement, tensing abdominal musculature...
drain 3-5 ml CSF if IVC present	reduces intracranial volume
hyperventilate to PaCO <sub>2</sub> = 30-35 mm Hg ("blows off" CO <sub>2</sub> )	↓ PaCO <sub>2</sub> → ↓ CBF → ↓ ICP
mannitol 0.25-1 gm/kg, then 0.25 mg/kg q 6 hrs, increase dose if IC-HTN persists & serum osmol $\leq$ 320 (NB: skip this step if hypovolemia or hypotension)	expands plasma volume, increases serum tonicity which draws fluid out of brain, may improve rheologic properties of blood
if there is "osmotic room" (i.e. serum osmol < 320) bolus with 10-20 ml of 23.4% hypertonic saline (HS)	some patients refractory to mannitol will respond to HS
hyperventilate to PaCO <sub>2</sub> = 25-30 mm Hg	monitor SjVO <sub>2</sub> (see page 874) or CBF if possible
If IC-HTN persists, consider unenhanced head CT† & EEG‡. Proceed to "second tier" therapy (see page 879).	

\* see text for details (beginning on page 878). As IC-HTN subsides, carefully withdraw treatment

† if IC-HTN persists, and especially for a sudden unexplained rise in ICP or loss of previously controlled ICP, give strong consideration to repeating cranial CT to rule out a surgical condition, i.e. "clot" (SDH, EDH, or ICH) or hydrocephalus

‡ EEG to rule-out subclinical status epilepticus which is a rare cause of sustained IC-HTN

## ICP MANAGEMENT PROTOCOL DETAILS

Goals of therapy:

1. keep ICP < 20 mm Hg (prevents "plateau waves" from compromising cerebral blood-flow (CBF) and causing cerebral ischemia and/or brain death<sup>122</sup>)
2. keep CPP  $\geq$  50 mm Hg<sup>119</sup>. The primary goal is to control ICP, simultaneously, CPP should be supported by maintaining adequate MAP<sup>123</sup>.

## SURGICAL TREATMENT

1. see surgical indications for subdural (page 896), epidural (page 895) or intraparenchymal hematoma (page 892) or posterior fossa mass lesions (page 905)
2. patients with hemorrhagic contusions ("pulsed brain") showing progressive deterioration may benefit from surgically excision of portions of the contused brain tissue especially if not eloquent brain (see page 880)
3. decompressive craniectomy may be considered for IC-HTN that cannot be controlled medically

## GENERAL CARE

### Major goals:

1. avoid hypoxia ( $pO_2 < 60$  mm Hg)
2. avoid hypotension (SBP  $\leq 90$  mm Hg): 67% positive-predictive value (PPV) for poor outcome (79% PPV when combined with hypoxia)<sup>124</sup>

### Details of general treatment measures

1. prophylaxis against steroid ulcers (if steroids are used) and Cushing's (stress) ulcers (seen in severe head injury and in increased ICP, accompanied by hypergastrinemia)<sup>125-129</sup> for all patients (see

*Prophylaxis for stress ulcers*, page 52)

- A. elevating gastric pH: titrated antacid and/or  $H_2$  antagonist (e.g. ranitidine 50 mg IV q 8 hrs). Avoid cimetidine if phenytoin is also being given
  - B. sucralfate
2. aggressive control of fever (fever is a potent stimulus to increase CBF, and may also increase plateau waves)<sup>122</sup>
  3. arterial line for BP monitoring and frequent ABGs
  4. CVP or PA line if high doses of mannitol are needed (goal: keep patient euvolemic)
  5. IV fluids
    - A. choice of fluids:
      1. isolated head injury: IVF of choice is isotonic (e.g. NS + 20 mEq KCl/L)
      2. avoid hypotonic solutions (e.g. lactated ringers) which may impair cerebral compliance<sup>130</sup>
    - B. fluid volume:
      1. provide adequate fluid resuscitation to avoid hypotension
      2. normalization of intravascular fluid volume is not detrimental to ICP
      3. although fluid restriction reduces the amount of mannitol needed to control ICP<sup>131</sup>, the concept of "running patients dry" is obsolete<sup>132</sup>
      4. if mannitol is required, patient should be maintained at euvolemia
      5. also exercise caution in restricting fluids following SAH (see *Cerebral salt wasting*, page 13)
      6. if injuries to other systems are present (e.g. perforated viscus), they may dictate fluid management
    - C. pressors (e.g. dopamine) are preferable to IV fluid boluses in head injury

**Table 27-21 Measures to treat an acute ICP crisis\***

Step	Rationale
check airway, position... (see general measures in Table 27-20). For resistant or sudden IC-HTN, consider STAT unenhanced head CT	
be sure patient is sedated and paralyzed (see Table 27-20)	(see Table 27-20)
drain 3-5 ml CSF if IVC present	↓ intracranial volume
mannitol† 1 gm/kg IV bolus or 10-20 ml of 23.4% saline	↑ plasma volume → ↑ CBF → ↓ ICP, ↑ serum osmolality → ↓ brain water
hyperventilate with Ambu® bag (always keep $PaCO_2 > 25$ mm Hg)	"blow off" (reduce) $PaCO_2$ → ↓ CBF → ↓ ICP. CAUTION: due to reduced CBF, use for no more than several minutes (see page 880)
pentobarbital‡ 100 mg slow IV or thiopental 2.5 mg/kg IV over 10 minutes	sedates, ↓ ICP (NB: also myocardial depressant → ↓ MAP), treats seizures, may be neuroprotective

\* for measures to treat ICP that is trending up over a longer period, see Table 27-20 or information starting on page 878

† skip this step and go to hyperventilation if hypotensive, volume depleted, or if serum osmolality  $> 320$  mOsm/L

‡ the availability of pentobarbital in the U.S. has been reduced, and other sedatives may need to be substituted, see page 884

## MEASURES TO LOWER ICP

### General measures that should be routine

1. positioning:
  - A. elevate HOB 30-45° (see below)
  - B. keep head midline (to prevent kinking jugular veins)
2. light sedation: codeine 30-60 mg IM q 4 hrs PRN, or lorazepam (Ativan®) 1-2 mg IV q 4-6 hrs PRN
3. avoid hypotension (SBP  $< 90$  mm Hg): normalize intravascular volume, support with pressors if needed
4. control HTN (in ICH, aim for patient's baseline, see *Initial management of ICH*, page 1126)

5. prevent hyperglycemia: (aggravates cerebral edema) usually present in head injury<sup>133, 134</sup>, may be exacerbated by steroids
6. intubation: for GCS  $\leq 8$  or respiratory distress. Give IV lidocaine first (see *Adjunctive measures* below) and antibiotics (see *PRACTICE GUIDELINE 27-5*, page 861)
7. avoid hyperventilation: keep PaCO<sub>2</sub> at the low end of eucapnia (35 mm Hg)
8. prophylactic hypothermia: non-statistically significant trend suggests reduced mortality<sup>135</sup>. Maintain target temperature for > 48 hours (see page 883)

## Measures to use for documented IC-HTN

First, check *General measures that should be routine* above. Proceed to each step if IC-HTN persists.

1. heavy sedation and/or paralysis when necessary (also assists treatment of HTN) e.g. when patient is agitated, or to blunt the elevation of ICP that occurs with certain maneuvers such as moving the patient to CT table. Caution: with heavy sedation or paralysis, the ability to follow the neurologic exam is lost (follow ICPs)
  - A. for heavy sedation (intubation recommended to avoid respiratory depression  $\rightarrow$  elevation of PaCO<sub>2</sub>  $\rightarrow$   $\uparrow$  ICP): e.g. one of the following:
    1. MSO<sub>4</sub>: **Rx** 2-4 mg/hr IV drip
    2. fentanyl: **Rx** 1-2 ml IV q 1 hr (or 2-5  $\mu$ g/kg/hr IV drip)
    3. sufentanil: **Rx** 10-30  $\mu$ g test dose, then 0.05-2  $\mu$ g/kg/hr IV drip
    4. midazolam (Versed®): **Rx** 2 mg test dose, then 2-4 mg/hr IV drip
    5. propofol drip (see page 24): 0.5 mg/kg test dose, then 20-75  $\mu$ g/kg/min IV drip \* avoid high-dose propofol (do not exceed 83  $\mu$ g/kg/min)
    6. "low dose" pentobarbital (adult: 100 mg IV q 4 hrs; peds: 2-5 mg/kg IV q 4 hrs)
  - B. paralysis (intubation mandatory): e.g. vecuronium 8-10 mg IV q 2-3 hrs
2. CSF drainage (when IVC is being utilized to measure ICP): 3-5 ml of CSF should be drained with the drip chamber at  $\leq 10$  cm above EAC. Works immediately by removal of CSF (reducing intracranial volume) and possibly by allowing edema fluid to drain into ventricles<sup>136</sup> (latter point is controversial)
3. "osmotic therapy" when there is evidence of IC-HTN:
  - A. mannitol (also see below) 0.25-1 gm/kg bolus (over < 20 mins) followed by 0.25 gm/kg IVP (over 20 min) q 6 hrs PRN ICP > 20. Recent literature suggests that 1.4 gm/kg initial dose is more effective. May "alternate" with: furosemide (Lasix®) (also see below): adult 10-20 mg IV q 6 hrs PRN ICP > 20. Peds: 1 mg/kg, 6 mg max IV q 6 hrs PRN ICP > 20
  - B. keep patient euvolemic to slightly hypervolemic
  - C. if IC-HTN persists and serum osmolality is < 320 mOsm/L, increase mannitol up to 1 gm/kg, and shorten the dosing interval
  - D. if ICP remains refractory to mannitol, consider hypertonic saline, either continuous 3% saline infusion or as bolus of 10-20 ml of 23.4% saline (D/C after  $\approx$  72 hours to avoid rebound edema)
  - E. hold osmotic therapy if serum osmolality is  $\geq 320$  mOsm/L (higher tonicity may have no advantage and risks renal dysfunction (see below)) or SBP < 100
4. hyperventilation (HPV) to PaCO<sub>2</sub> = 30-35 mm Hg (for details, see below)
  - A. \* do not use prophylactically
  - B. \* avoid aggressive HPV (PaCO<sub>2</sub>  $\leq 25$  mm Hg) at all times
  - C. use only for
    1. short periods for acute neurologic deterioration
    2. or chronically for documented IC-HTN unresponsive to sedation, paralysis, CSF drainage and osmotic therapy
  - D. avoid HPV during the first 24 hrs after injury if possible
5. \* steroids: the routine use of glucocorticoids is not recommended for treatment of patients with head injuries (see below)

## "Second tier" therapy for persistent IC-HTN

If IC-HTN remains refractory to the above measures, and especially if there is loss of previously controlled ICP, strong consideration should be given to repeating a head CT to rule out a surgical condition before proceeding with "second tier" therapies which are either effective but with significant risks (e.g. high-dose barbiturates), or are unproven in terms of benefit on outcome. Also consider an EEG to rule-out status epilepticus that is not clinically evident (see page 405 for treatment measures for status epilepticus; some medications are effective for both seizures and IC-HTN, e.g. pentobarbital, propofol...).

1. high dose barbiturate therapy: initiate if ICP remains > 20-25 mm Hg (see *High-dose barbiturate therapy*, page 883)
2. hyperventilate to  $\text{PaCO}_2 = 25\text{-}30$  mm Hg. Monitoring  $\text{SjVO}_2$ ,  $\text{AVdO}_2$ , and/or CBF is recommended (see *below*)
3. hypothermia<sup>137, 138</sup>: patients must be monitored for a drop in cardiac index, thrombocytopenia, elevated creatinine clearance, and pancreatitis. Avoid shivering which raises ICP<sup>138</sup>
4. decompressive surgery:
  - A. decompressive craniectomy removal of portion of calvaria<sup>139</sup>. Controversial (may enhance cerebral edema formation<sup>140</sup>). Craniectomy decreased ICP to < 20 mm Hg in 85%<sup>141</sup> regardless of pupillary response to light, timing of craniectomy, brain shift and age. Outcomes were improved when IC-HTN responded<sup>82, 141, 142</sup>. Further randomized trials are indicated. Early decompressive craniectomy may be considered in patients undergoing emergent surgery (for fracture, EDH, SDH...)<sup>143</sup>. Flap must be at least 12 cm in diameter, and duraplasty is mandatory. Also, see *Hemicraniectomy for malignant MCA territory infarction*, page 1022
  - B. removal of large areas of contused hemorrhagic brain (makes room immediately; removes region of disrupted BBB). If contused, consider temporal tip lobectomy (no more than 4-5 cm on dominant side, 6-7 cm on non-dominant) (total temporal lobectomy<sup>144</sup> is probably too aggressive) or frontal lobectomy. Has not shown great therapeutic promise
5. lumbar drainage: showing some promise. Watch for "cerebral sag"
6. hypertensive therapy

### ADJUNCTIVE MEASURES

1. **lidocaine**: 1.5 mg/kg IVP (watch for hypotension, reduce dose if necessary) at least one minute before endotracheal intubation or suctioning. Blunts the rise in ICP as well as tachycardia and systemic HTN (based on patients with brain tumors undergoing intubation under light barbiturate-nitrous oxide anesthesia; extrapolation to trauma patients is unproven)<sup>145</sup>
2. high frequency (jet) ventilation: consider if high levels of positive end-expiratory pressure (**PEEP**) are required<sup>146</sup> (NB: patients with reduced lung compliance, e.g. pulmonary edema, transmit more of PEEP through lungs to thoracic vessels and may raise ICP).  $\text{PEEP} \leq 10$  cm  $\text{H}_2\text{O}$  does not cause clinically significant increases in ICP<sup>147</sup>. Higher levels of  $\text{PEEP} > 15\text{-}20$  are not recommended. Also, rapid elimination of PEEP may cause a sudden increase in circulating blood volume which may exacerbate cerebral edema and also elevate ICP

### DETAILS OF SOME MEASURES OUTLINED ABOVE

#### ELEVATING HEAD OF BED (HOB)

Early data indicated that keeping the HOB at 30-45° optimized the trade-off between the following two factors as the HOB is elevated: reducing ICP (by enhancing venous outflow and by promoting displacement of CSF from the intracranial compartment to the spinal compartment) and reducing MAP (and thus CPP) at the level of the carotid arteries.

Recent data<sup>148</sup> indicate that although mean carotid pressure (**MCP**) is reduced, the ICP is also reduced and the CBF is unaffected by elevating the HOB to 30°. The onset of action of raising the HOB is immediate.

#### HYPERVENTILATION

Intraarterial carbon dioxide ( $\text{PaCO}_2$ ) is the most potent cerebrovascular vasodilator. Hyperventilation (**HPV**) lowers ICP by reducing  $\text{PaCO}_2$  which causes cerebral vasoconstriction, thus reducing the cerebral blood volume (**CBV**)<sup>149</sup>. Of concern, vasoconstriction also lowers cerebral blood flow (**CBF**) which could produce focal ischemia in areas with preserved cerebral autoregulation as a result of shunting<sup>150, 151</sup>. However, ischemia does not necessarily follow as the  $\text{O}_2$  extraction fraction (**OEF**) may also increase, up to a point<sup>152</sup>.

✖ Hyperventilation (**HPV**), is to be used in moderation only in specific situations<sup>50</sup> (see *below*). Prophylactic<sup>A</sup> HPV may actually be associated with a worse outcome<sup>154</sup>. When indicated, use HPV only to  $\text{PaCO}_2 = 30\text{-}35$  mm Hg (see *Caveats for hyperventilation*

below). CBF in severe head trauma patients is already about half of normal during the first 24 hrs after injury<sup>155-158</sup>. In one study, the use of HPV to  $\text{PaCO}_2 = 30$  mm Hg within 8-14 hrs of severe head injury did not impair *global* cerebral metabolism<sup>152</sup>, but *focal* changes were not studied. Hyperventilation to  $\text{PaCO}_2 < 30$  mm Hg further reduces CBF, but does not consistently reduce ICP and may cause loss of cerebral autoregulation<sup>106</sup>. If carefully monitored, there may be occasion to use this. A summary of the ranges of  $\text{PaCO}_2$  and the recommendations is shown in Table 27-22.

#### PRACTICE GUIDELINE 27-14 HYPERVENTILATION FOR ICP MANAGEMENT\*

**Level I**<sup>153</sup>: in the absence of IC-HTN, chronic prolonged hyperventilation (HPV) ( $\text{PaCO}_2 \leq 25$  mm Hg) should be avoided

**Level II**<sup>153</sup>: *prophylactic* hyperventilation ( $\text{PaCO}_2 \leq 25$  mm Hg) is not recommended

#### Level III

- HPV may be necessary for brief periods when there is acute neurologic deterioration, or for longer periods if there is IC-HTN refractory to sedation, paralysis, CSF drainage and osmotic diuretics<sup>153</sup>
- HPV should be avoided  $\leq 24$  hrs after head injury<sup>53</sup>
- if HPV is used, jugular venous oxygen saturation ( $\text{SjVO}_2$ ) (see page 874) or  $\text{P}_{\text{brO}_2}$  (see page 874) should be measured to monitor brain  $\text{O}_2$  delivery<sup>53</sup>

\* for prophylactic hyperventilation, see PRACTICE GUIDELINE 27-6, page 861

Reducing  $\text{PaCO}_2$  from 35 to 29 mm Hg lowers ICP 25-30% in most patients. Onset of action:  $\leq 30$  seconds. Peak effect at  $\approx 8$  mins. Duration of effect is occasionally as short as 15-20 mins. Effect may be blunted by 1 hour (based on patients with intracranial tumors), after which it is difficult to return to normocarbica without rebound elevation of ICP<sup>159, 160</sup>. Thus, HPV must be weaned slowly<sup>122</sup>.

#### Indications for hyperventilation (HPV)

1. HPV for brief periods (minutes) at the following times
  - A. prior to insertion of ICP monitor: if there are clinical signs of IC-HTN (see Table 27-14, page 861)
  - B. after insertion of a monitor: if there is a sudden increase in ICP and/or acute neurologic deterioration, HPV may be used while evaluating patient for a treatable condition (e.g. delayed intracranial hematoma)
2. HPV for longer periods: when there is documented IC-HTN unresponsive to sedation, paralytics, CSF drainage (when available) and osmotic diuretics
3. HPV may be appropriate for IC-HTN resulting primarily from hyperemia (see page 902)

#### Caveats for hyperventilation

1. avoid during the first 5 days after head injury if possible (especially first 24 hrs)
2. do not use prophylactically (i.e. without appropriate indications, see above)
3. if documented IC-HTN is unresponsive to other measures, hyperventilate only to  $\text{PaCO}_2 = 30-35$  mm Hg
4. if prolonged HPV to  $\text{PaCO}_2$  of 25-30 mm Hg is deemed necessary, consider monitoring  $\text{SjVO}_2$ ,  $\text{AVdO}_2$ , or CBF to rule-out cerebral ischemia (see page 874)

A. *prophylactic* HPV implies cases where there are no clinical signs of IC-HTN and where IC-HTN unresponsive to other measures has not been documented by ICP monitoring

**Table 27-22 Summary of recommendations for  $\text{PaCO}_2$  following head trauma (see text for details)**

$\text{PaCO}_2$ (mm Hg)	Description
35-40	normocarbica. Use routinely
30-35	hyperventilation. Do not use prophylactically. Use only as follows: briefly for clinical evidence of IC-HTN (neurologic deterioration) or chronically for documented IC-HTN unresponsive to other measures
25-30	augmented hyperventilation. A second tier treatment. Use only when other methods fail to control IC-HTN. Additional monitoring recommended to R/O cerebral ischemia
< 25	aggressive hyperventilation. No documented benefit. Significant potential for ischemia

- do not reduce  $\text{PaCO}_2 < 25$  mm Hg (except for very brief periods of a few minutes)

## MANNITOL

### PRACTICE GUIDELINE 27-15 MANNITOL IN SEVERE TRAUMATIC BRAIN INJURY

#### Level II<sup>54, 161</sup>

- mannitol is effective for control of IC-HTN after severe TBI\*
- intermittent boluses may be more effective than continuous infusion
- effective doses range from 0.25-1 gm/kg body weight
- avoid hypotension (SBP < 90 mm Hg)

#### Level III<sup>161</sup>

- indications: signs of transtentorial herniation or progressive neurological deterioration not attributable to systemic pathology
- euvolemia should be maintained (hypovolemia should be avoided) by fluid replacement. An indwelling urinary catheter is essential
- serum osmolality should be kept < 320 mOsm when there is concern about renal failure

\* current information did not allow recommendations regarding hypertonic saline to be made<sup>54</sup>

The mechanism(s) by which mannitol provides its beneficial effects is still controversial, but probably includes some combination of the following

- lowering ICP
  - immediate plasma expansion<sup>162-164</sup>: reduces the hematocrit and blood viscosity (improved rheology) which increases CBF and  $\text{O}_2$  delivery. This reduces ICP within a few minutes, and is most marked in patients with CPP < 70 mm Hg
  - osmotic effect: increased serum tonicity draws edema fluid from cerebral parenchyma. Takes 15-30 minutes until gradients are established<sup>162</sup>. Effect lasts 1.5-6 hrs, depending on the clinical condition<sup>50, 165, 166</sup>
- supports the microcirculation by improving blood rheology (*see above*)
- possible free radical scavenging<sup>167</sup>

With bolus administration, onset of ICP lowering effect occurs in 1-5 minutes; peaks at 20-60 minutes. When urgent reduction of ICP is needed, an initial dose of 1 gm/kg should be given over 30 minutes. When long-term reduction of ICP is intended, the infusion time should be lengthened to 60 minutes<sup>168</sup> and the dose reduced (e.g. 0.25-0.5 gm/kg q 6 hrs).

### Cautions with mannitol

- mannitol opens the BBB, and mannitol that has crossed the BBB may draw fluid into the CNS (this may be minimized by repeated bolus administration vs. continuous infusion<sup>163, 169</sup>) which can aggravate vasogenic cerebral edema<sup>170</sup>. Thus, when it is time to D/C mannitol, it should be tapered to prevent ICP rebound<sup>168</sup>
- caution: corticosteroids + phenytoin + mannitol may cause hyperosmolar nonketotic state with high mortality<sup>122</sup>
- excessively vigorous bolus administration may → HTN and if autoregulation is defective → increased CBF which may promote herniation rather than prevent it<sup>171</sup>
- high doses of mannitol carries the risk of acute renal failure (acute tubular necrosis), especially in the following<sup>76, 172</sup>: serum osmolality > 320 mOsm/L, use of other potentially nephrotoxic drugs, sepsis, pre-existing renal disease
- large doses prevents diagnosing DI by use of urinary osmols or SG (*see page 16*)
- because it may further increase CBF<sup>173</sup>, the use of mannitol may be deleterious when IC-HTN is due to hyperemia (*see page 902*)

### FUROSEMIDE

The use of furosemide (Lasix®) has been advocated, but little data exists to support this<sup>50</sup>. Loop acting diuretics may reduce ICP<sup>174</sup> by reducing cerebral edema<sup>175</sup> (possibly by increasing serum tonicity), and may also slow the production of CSF<sup>176</sup>. They also act synergistically with mannitol<sup>177</sup> (*see Mannitol above*).

**Rx:** 10-20 mg IV q 6 hrs, may be alternated with mannitol such that the patient receives one or the other q 3 hrs. Hold if serum osmolality > 320 mOsm/L.

## HYPERTONIC SALINE (HS)

May reduce ICP in patients refractory to mannitol<sup>178, 179</sup>, although no improvement in outcome over mannitol has been demonstrated<sup>179, 180</sup>. Potentially deleterious effect on stroke penumbra in animal studies. Studies<sup>181, 182</sup> not adequate to make recommendations regarding use<sup>54</sup>.

**Rx:** Continuous infusion: 3% saline at 25-50 ml/hr may be given through a peripheral IV. Bolus: 10-20 ml of 7.5-23.4% saline must be given through a central line. HS should be discontinued after ~ 72 hours to avoid rebound edema<sup>179</sup>. Hold if serum osmolality > 320 mOsm/L.

## PROPHYLACTIC HYPOTHERMIA

### PRACTICE GUIDELINE 27-16 PROPHYLACTIC HYPOTHERMIA

#### Level III<sup>135</sup>: prophylactic hypothermia:

- improves the chances of having a moderate to good outcome (4-5 on the Glasgow Outcome Score, *see page 1183*) at the end of the follow-up period when target temperatures of 32-35° C\* (91.4-95° F) were used
- showed a non-significant trend suggesting that it lowers mortality when the target temperature is maintained for > 48 hrs†

\* no clear relationship was found for cooling duration or rewarming rate

† the actual target temperature and rewarming rate did not influence mortality

## STEROIDS

### PRACTICE GUIDELINE 27-17 GLUCOCORTICOIDS IN SEVERE HEAD INJURY

**Level I<sup>183</sup>:** the use of glucocorticoids (steroids) is not recommended for improving outcome or reducing ICP in patients with severe TBI (except in patients with known depletion of endogenous adrenal hormones<sup>184, 185</sup>). High-dose methylprednisolone is associated with increased mortality and is contraindicated<sup>183</sup>

Although glucocorticoids reduce **vasogenic cerebral edema** (e.g. surrounding brain tumors) and may be effective in lowering ICP in pseudotumor cerebri, they have little effect on **cytotoxic cerebral edema** which is more prevalent following trauma (see *Cerebral edema*, page 109).

Significant side effects may occur<sup>186</sup> including coagulopathies, hyperglycemia<sup>187</sup> with its undesirable effect on cerebral edema (see *Possible deleterious side effects of steroids*, page 33), and increased incidence of infection. High-dose methylprednisolone is associated with increased mortality<sup>188</sup>.

Non-glucocorticoid steroids (e.g. 21-aminosteroids, AKA lazaroids, including tirilazad<sup>189, 190</sup>) and the synthetic glucocorticoid triamcinolone<sup>191</sup> have also failed to show overall benefit.

## 27.3.3.1. High-dose barbiturate therapy

### PRACTICE GUIDELINE 27-18 BARBITURATES IN SEVERE HEAD INJURY

**Level II<sup>192</sup>:** \* *prophylactic* use of barbiturates for burst suppression EEG is not recommended

**Level II<sup>192</sup>:** high-dose barbiturates are recommended for IC-HTN refractory to maximal medical and surgical ICP lowering therapy. Patients should be hemodynamically stable before and during treatment

Theoretical benefits of barbiturates in head injury derive from vasoconstriction in normal areas (shunting blood to ischemic brain tissue), decreased metabolic demand for O<sub>2</sub> (CMRO<sub>2</sub>) with accompanying reduction of CBF, free radical scavenging, reduced intracellular calcium, and lysosomal stabilization<sup>193</sup>. There is little question that barbiturates lower ICP, even when other treatments have failed<sup>194</sup>, but regarding outcome, studies have shown both benefits<sup>195, 196</sup> and lack of same<sup>197, 198</sup>. Patients that do respond have a lower mortality (33%) than those in whom ICP control could not be accomplished (75%)<sup>196</sup>.

The limiting factor for therapy is usually hypotension due to barbiturate induced reduction of sympathetic tone<sup>199</sup> (p 354) (causing peripheral vasodilatation) and direct mild



myocardial depression. Hypotension occurs in ~ 50% of patients in spite of adequate blood volume and use of dopamine<sup>197</sup>.

NB: the ability to follow the neurologic exam is lost, and one must follow ICP.

**"Barbiturate coma" vs. high-dose therapy:** If barbiturates are given until there is burst suppression on EEG, this is considered true "barbiturate coma". This results in near maximal reductions in CMRO<sub>2</sub> and CBF<sup>60</sup>. However, most regimens should technically be called "high dose intravenous therapy" since they simply try to establish target serum barbiturate levels (e.g. 3-4 mg% for pentobarbital), even though there is poor correlation between serum level, therapeutic benefit, and systemic complications<sup>60</sup>.

Adjunctive measures to administration of high-dose barbiturates:

1. consider a Swan-Ganz (PA) catheter placed during the first hour of loading dose
2. high-dose barbiturates cause paralytic ileus: therefore NG tube to suction & IV hyperalimentation are usually needed

## INDICATIONS

The use of barbiturates should be reserved for situations where the ICP cannot be controlled by the previously outlined measures<sup>196</sup>, as there is evidence that prophylactic barbiturates do not favorably alter outcome, and are associated with significant side effects, mostly hypotension<sup>197</sup>, that can cause neurologic deterioration.

## CHOICE OF AGENTS

A number of agents have been studied, however, there is inadequate data to recommend one drug over another. The most information is available on pentobarbital (*see below*). Alternative agents which have not been as well studied: thiopental (*see below*), phenobarbital (*see page 413*) & propofol (*see page 885*).

### pentobarbital (Nembutal®)

### DRUG INFO

Pentobarbital has a fast onset (full effects within ~ 15 minutes), short duration of action (3-4 hrs), and a half life of 15-48 hrs.

## Protocols for pentobarbital therapy in adults

There are many protocols. A simple one from a randomized clinical trial<sup>200</sup>:

- loading dose:
  - A. pentobarbital 10 mg/kg IV over 30 minutes
  - B. then 5 mg/kg q 1 hr x 3 doses
- maintenance: 1 mg/kg/hr

A more elaborate protocol:

1. loading dose: pentobarbital 10 mg/kg/hr IV over 4 hrs as follows:
  - A. FIRST HOUR: 2.5 mg/kg slow IVP q 15 min x 4 doses (total: 10 mg/kg in first hr), follow BP closely
  - B. next 3 hours: 10 mg/kg/hr continuous infusion (put 2500 mg in 250 ml of appropriate IVF, run at K ml/hr x 3 hrs (K = patient's weight in kg))
2. maintenance: 1.5 mg/kg/hr infusion (put 250 mg in 250 ml IVF and run at 1.5 x K ml/hr)
3. check serum pentobarbital level 1 hr after loading dose completed; usually 3.5-5.0 mg%
4. check serum pentobarbital level q day thereafter
5. if level ever > 5 mg% and ICP acceptable, reduce dose
6. baseline brain stem auditory evoked response (BAER) early in treatment. May be omitted on clinical grounds. Repeat BAER if pentobarbital level ever > 6 mg%. Reduce dose if BAER deteriorates (caution: hemotympanum may interfere with BAER)

**Table 27-23 CNS effects of various pentobarbital levels\***

Degree of CNS depression	mg%	µg/ml
level for valid brain death exam	≤ 1	≤ 10
sedated, relaxed, easily aroused	0.05-0.3	0.5-3
heavy sedation, difficult to arouse, respiratory depression	2	20
"coma" level (burst suppression occurs in most patients)	5	50

\* levels reported are for intolerant patients; there is significant variability between patients and tolerant patients may not be sedated even at levels as high as 100 µg/ml

- goal: ICP < 24 mm Hg and pentobarbital level 3-5 mg%. Consider discontinuing pentobarbital due to ineffectiveness if ICP still > 24 with adequate drug levels x 24 hrs
- if ICP < 20 mm Hg, continue treatment x 48 hrs, then taper dose. Backtrack if ICP rises

Neuro function takes ~ 2 days off pentobarbital to return (see Table 27-23). Level should be  $\approx 10$   $\mu\text{g/ml}$  before brain death exam is valid.

### thiopental (Pentothal®)

DRUG INFO

May be useful when a rapidly acting barbiturate is needed (e.g. intra-op) or when large doses of pentobarbital are not available. One of many protocols follows (note: thiopental has not been as well studied for this indication, but is theoretically similar to pentobarbital<sup>[201, 202]</sup>):

- loading dose: thiopental 5 mg/kg (range: 3-5) IV over 10 minutes
- follow with continuous infusion of 5 mg/kg/hr (range: 3-5) for 24 hours
- may need to rebolus with 2.5 mg/kg as needed for ICP control
- after 24 hours, fat stores become saturated, reduce infusion to 2.5 mg/kg/hr
- titrate to control ICP or use EEG to monitor for electrocerebral silence
- "therapeutic" serum level: 6-8.5 mg/dl

### propofol (Diprivan®)

DRUG INFO

#### PRACTICE GUIDELINE 27-19 PROPOFOL IN SEVERE HEAD INJURY

**Level II**<sup>192</sup>: propofol may control ICP after several hours of dosing, but it does not improve mortality or 6 month outcome. \* Caution: high-dose propofol (total dose > 100 mg/kg for > 48 hrs) can cause significant morbidity

**Rx:** 0.5 mg/kg test dose, then 20-75  $\mu\text{g/kg/min}$  infusion. Increase by 5-10  $\mu\text{g/kg/min}$  q 5-10 minutes PRN ICP control (do not exceed 83  $\mu\text{g/kg/hr}$  = 5 mg/kg/hr).

**SIDE EFFECTS:** include Propofol Infusion Syndrome (see page 25). Use with caution at doses > 5 mg/kg/hr or at any dose for > 48 hrs.

## 27.4. Skull fractures

Table 27-24 shows some differentiating features to distinguish linear skull fractures. See also *Indications for CT and admission criteria for TBI* on page 856.

90% of pediatric skull fractures are linear and involve the calvaria.

**Diastatic fractures** extend into and separate sutures. More common in young children<sup>203</sup>.

Table 27-24 Differentiating linear skull fractures from normal plain film findings

Feature	Linear skull fracture	Vessel groove	Suture line
density	dark black	grey	grey
course	straight	curving	follows course of known suture lines
branching	usually none	often branching	joins other suture lines
width	very thin	thicker than fracture	jagged, wide

### 27.4.1. Depressed skull fractures

Classified as either closed (**simple fracture**) or open (**compound fracture**).

#### ADULT

See **PRACTICE GUIDELINE 27-20** for surgical management guidelines. Some additional observations regarding surgery to elevate a depressed skull fracture in an adult:

- consider surgery for depressed skull fractures with deficit referable to underlying brain
- \* more conservative treatment is recommended for fractures overlying a major

## PRACTICE GUIDELINE 27-20 SURGICAL MANAGEMENT OF DEPRESSED SKULL FRACTURES

### Indications for surgery

#### Level III<sup>204</sup>:

- open (compound) fractures
  - A. surgery for fractures depressed > thickness of calvaria and those not meeting criteria for nonsurgical management listed below
  - B. nonsurgical management may be considered if
    1. there is no evidence (clinical or CT) of dural penetration (CSF leak, intradural pneumocephalus on CT...)
    2. and no significant intracranial hematoma
    3. and depression is < 1 cm
    4. and no frontal sinus involvement
    5. and no wound infection or gross contamination
    6. and no gross cosmetic deformity
- closed (simple) depressed fractures: may be managed surgically or non-surgically

### Timing of surgery

**Level III<sup>204</sup>:** early surgery to reduce risk of infection

### Surgical methods

#### Level III<sup>204</sup>:

- elevation and debridement are recommended
- option: if there is no evidence of wound infection, primary bone replacement
- antibiotics should be used for all compound depressed fractures

There is no evidence that elevating a depressed skull fracture will reduce the subsequent development of posttraumatic seizures<sup>205</sup>, which are probably more related to the initial brain injury.

### PEDIATRIC<sup>206</sup>

Most common in frontal and parietal bones. One third are closed, and these tend to occur in younger children as a result of the thinner, more deformable skull. Open fractures tended to occur with MVAs, closed fractures tended to follow accidents at home. Dural lacerations are more common in compound fractures.

### Simple depressed skull fractures

No difference in outcome (seizures, neurologic dysfunction or cosmetic appearance) in surgical vs. nonsurgical treatment. In the younger child, remodelling of the skull as a result of brain growth tends to smooth out the deformity.

Indications for surgery for pediatric simple depressed skull fracture:

1. definite evidence of dural penetration
2. persistent cosmetic defect in the older child after the swelling has subsided
3. ± focal neurologic deficit related to the fracture (this group has a higher incidence of dural laceration, although it is usually trivial)

### "Ping-pong ball" fractures<sup>207</sup>

A green-stick type of fracture → caving in of a focal area of the skull as in a crushed area of a ping-pong ball. Usually seen only in the newborn due to the plasticity of the skull.

### Indications for surgery

No treatment is necessary when these occur in the temporoparietal region in the absence of underlying brain injury as the deformity will usually correct as the skull grows.

1. radiographic evidence of intraparenchymal bone fragments
2. associated neurologic deficit (rare)
3. signs of increased intracranial pressure
4. signs of CSF leak deep to the galea

A. exception: depressed fractures overlying and depressing one of the dural sinuses may be dangerous to elevate, and if the patient is neurologically intact, and no indication for operation (e.g. CSF leak mandates surgery) may be best managed conservatively

5. difficulty with long-term follow-up

#### Technique

Frontally located lesions may be corrected for cosmesis by a small linear incision behind the hairline, opening the cranium adjacent to the depression, and pushing it back out e.g. with a Penfield #3 dissector.

#### Booking the case - craniotomy: for depressed skull fracture

Also see defaults & disclaimers (page v).



1. position: (depends on location of the fracture)
2. post-op: ICU
3. blood: type & screen (for severe fractures: type and cross 2 U PRBC)
4. consent (in lay terms for the patient - not all-inclusive):
  - A. procedure: surgery in the area of the skull fracture to bone fragments that may have been displaced, to repair the covering of the brain, to remove any foreign material that can be identified and any permanently damaged brain tissue (i.e. dead brain tissue), remove any blood clot and stop any bleeding identified, possible placement of intracranial pressure monitor. If a large opening has to be left in the skull, it may require surgery to correct in a number of months (3 or more)
  - B. alternatives: nonsurgical management
  - C. complications: (usual craniotomy complications - see page v) plus any permanent brain injury that has already occurred is not likely to recover, seizures may occur (with or without the surgery), hydrocephalus, infection (including delayed infection/abscess)

#### Technical considerations of surgery

Surgical goals (modified<sup>208</sup>)

1. debridement of skin edges
2. elevation of bone fragments
3. repair of dural laceration
4. debridement of devitalized brain
5. reconstruction of the skull
6. skin closure

#### Techniques

1. with open (compound) contaminated fractures, it may be necessary to excise depressed bone. In these cases or when air sinuses are involved, to minimize the risk of infecting the flap, some surgeons follow the patient for 6-12 months to rule out infection before performing a cosmetic cranioplasty. There has been no documented increase in infection with replacement of bone fragments; soaking the fragments in povidone-iodine has been recommended<sup>208</sup>
2. elevating the bone may be facilitated by drilling burr holes around the periphery and either using rongeurs or craniotome to excise the depressed portion
3. in cases where laceration of a major dural sinus is suspected and surgery is mandated, adequate preparation must be made for dural sinus repair<sup>209</sup> (NB: the SSS is often to the right of the sagittal suture - see page 87)
  - A. prepare for massive blood loss
  - B. have small Fogarty catheter ready to temporarily occlude sinus
  - C. have dural shunt ready (Kapp-Gielchinsky shunt, if available, has an inflatable balloon at both ends)
  - D. prep out saphenous vein area for vein graft
  - E. bone fragments that may have lacerated sinus should be removed last

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## 27.4.2. Basal skull fractures

Most basal (AKA basilar) skull fractures (BSF) are extensions of fractures through the cranial vault.

#### DIAGNOSIS

##### Radiographic diagnosis

CT scan is often poor for directly demonstrating BSF. Plain skull x-rays and clinical criteria (see below) are usually more sensitive. Sensitivity of CT diagnosis can be in-

creased by the use of bone windows together with thin cuts ( $\leq 5$  mm) and coronal images. BSF appear as linear lucencies through the skull base.

Indirect radiographic findings (on CT or plain films) that suggest BSF include: pneumocephalus (diagnostic of BSF in the absence of an open fracture of the cranial vault), air/fluid level within or opacification of air sinus with fluid (suggestive). Other related findings include: fractures of the cribriform plate or orbital roof.

### Clinical diagnosis

Some of these signs may take several hours to develop. Signs include:

1. CSF otorrhea or rhinorrhea
2. hemotympanum or laceration of external auditory canal
3. postauricular ecchymoses (Battle's sign)
4. periorbital ecchymoses (raccoon's eyes) in the absence of direct orbital trauma, especially if bilateral
5. cranial nerve injury:
  - A. VII and/or VIII: usually associated with temporal bone fracture
  - B. olfactory nerve (Cr. N. I) injury: often occurs with anterior fossa BSF and results in anosmia, this fracture may extend to the optic canal and cause injury to the optic nerve (Cr. N. II)
  - C. VI injury: can occur with fractures through the clivus (see below)

Severe basilar skull fractures may produce shearing injuries to the pituitary gland.

### TREATMENT

**NG tubes:** \* Caution: cases have been reported where an NG tube has been passed intracranially<sup>210-212</sup> and is associated with fatal outcome in 64% of cases. Possible mechanisms include: a cribriform plate that is thin (congenitally or due to chronic sinusitis) or fractured (due to a frontal basal skull fracture or a comminuted fracture through the skull base).

Suggested contraindications to blind placement of an NG tube include: trauma with possible basal skull fracture, ongoing or history of previous CSF rhinorrhea, meningitis with chronic sinusitis.

**Prophylactic antibiotics:** The routine use of prophylactic antibiotics is controversial. This remains true even in the presence of a CSF fistula (see *CSF fistula (cranial)*, page 300). However, most ENT physicians recommend treating fractures through the nasal sinuses as open contaminated fractures, and they use broad spectrum antibiotics (e.g. ciprofloxacin) for 7-10 days.

**Treatment of the BSF:** Most do not require treatment by themselves. However, conditions that may be associated with BSF which may require specific management include:

1. "traumatic aneurysms"<sup>213</sup> (see *Traumatic aneurysms*, page 1081)
2. posttraumatic carotid-cavernous fistula (see *Carotid-cavernous fistula*, page 1113)
3. CSF fistula: operative treatment may be required for persistent CSF rhinorrhea (see *CSF fistula (cranial)*, page 300)
4. meningitis or cerebral abscess: may occur with BSF into air sinuses (frontal or mastoid) even in the absence of an identifiable CSF leak. May even occur many years after the BSF was sustained (see *Post craniospinal trauma meningitis (post-traumatic meningitis)*, page 344)
5. cosmetic deformities
6. posttraumatic facial palsy (see *Temporal bone fractures* below)

### TEMPORAL BONE FRACTURES

Although often mixed, there are two basic types of temporal bone fractures:

1. longitudinal fracture: more common (70-90%). Usually through petro-squamosal suture, parallel to and through EAC. Can often be diagnosed on otoscopic inspection of the EAC. Usually passes between cochlea and semicircular canals (SCC) sparing the VII and VIII nerves, but may disrupt ossicular chain
2. transverse fracture: perpendicular to EAC. Often passes through cochlea and may place stretch on geniculate ganglion, resulting in VIII and VII nerve deficits respectively

## POSTTRAUMATIC FACIAL PALSY

Posttraumatic unilateral peripheral facial nerve palsy may be associated with petrous bone fractures as noted above.

### Management

Management is often complicated by multiplicity of injuries (including head injury requiring endotracheal intubation) making it difficult to determine the time of onset of facial palsy. Guidelines:

1. regardless of time of onset:
  - A. steroids (glucocorticoids) are often utilized (efficacy unproven)
  - B. consultation with ENT physician is usually indicated
2. immediate onset of unilateral peripheral facial palsy: facial EMG (AKA electroneuronography<sup>214</sup> or ENOG) takes at least 72 hrs to become abnormal. These cases are often followed and are possible candidates for surgical VII nerve decompression if no improvement occurs with steroids (timing of surgery is controversial, but is usually not done emergently)
3. delayed onset of unilateral peripheral facial palsy: follow serial ENOGs, if continued nerve deterioration occurs while on steroids, and activity on ENOG drops to less than 10% of the contralateral side, surgical decompression may be considered (controversial, thought to improve recovery from  $\approx 40\%$  to  $\approx 75\%$  of cases)

## CLIVAL FRACTURES<sup>215</sup>

3 categories (75% are longitudinal or transverse):

1. longitudinal: may be associated with injuries of vertebrobasilar vessels including:
  - A. dissection or occlusion: may cause brain stem infarction
  - B. traumatic aneurysms
2. transverse: may be associated with injuries to the anterior circulation
3. oblique

Clival fractures are highly lethal. May be associated with:

1. cranial nerve deficits: especially III through VI; bitemporal hemianopsia
2. CSF leak
3. diabetes insipidus

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## 27.4.3. Craniofacial fractures

### FRONTAL SINUS FRACTURES

Frontal sinus fractures account for 5-15% of facial fractures.

In the presence of a frontal sinus fracture, intracranial air (pneumocephalus) on CT even without a clinically evident CSF leak, must be presumed to be due to dural laceration (although it could also be due to a basal skull fracture, see *Pneumocephalus* below).

Anesthesia of the forehead may occur due to supratrochlear and/or supraorbital nerve involvement.

The risks of posterior wall fractures are not immediate, but may be delayed (some even by months or years) and include:

1. brain abscess
2. CSF leak with risk of meningitis
3. cyst or mucocoele formation: injured frontal sinus mucosa has a higher predilection for mucocoele formation than other sinuses<sup>216</sup>. Mucocoeles may also develop as a result of frontonasal duct obstruction due to fracture or chronic inflammation. Mucocoeles are prone to infection (mucopyocoele) which can erode bone and expose dura with risk of infection

### SURGICAL CONSIDERATIONS

#### Indications

Linear fractures of the anterior wall of the frontal sinus are treated expectantly.

Indications for exploration of posterior wall fractures is controversial<sup>217</sup>. Some argue that a few mm of displacement, or that CSF fistula that resolves may not require exploration. Others vehemently disagree.

## Technique

In the presence of a traumatic forehead laceration, the frontal sinus may be exposed through judicious incorporation of the laceration in a forehead incision. Without such a laceration, either a bicoronal (souttar) skin incision or a butterfly incision (through the lower part of the eyebrows, crossing the midline near the glabella) is used.

**Dealing with frontal sinus:** ✱ Simple packing of the sinus (with bone wax, Gelfoam®, muscle or fat) increases the possibility of infection or mucocele formation.

The rear wall of the sinus is removed (so-called cranialization of the frontal sinus). The sinus is then exenterated (mucosa is stripped from sinus wall down to the nasofrontal duct, the mucosa is inverted over itself in the region of the duct and is packed down into the duct, temporalis muscle plugs are then packed into the frontonasal ducts<sup>217</sup>), then the bony wall of the sinus is drilled with a diamond burr to remove tiny remnants of mucosa found in the surface of bone that may proliferate and form a mucocele<sup>216</sup>. If there is any remnant of sinus, it may then be packed with abdominal fat that fills all corners of the cavity. Post-op risks related to frontal sinus injury include: infection, mucocele formation and CSF leak.

In the presence of pneumocephalus, if no obvious dural laceration is found the dural undersurface of the frontal lobes should be checked for leaks. Extradural inspection and repair is rarely indicated; the act of lifting the dura off the floor of the frontal fossa in the region of the ethmoid sinuses often creates lacerations<sup>218</sup>. Intradural repair is accomplished using a graft (fascia lata is most desirable; periosteum is thinner but is often acceptable) which is held in place with sutures and must extend all the way back to the ridge of the sphenoid wing (fibrin glue may be a helpful adjunct).

A periosteal flap is placed across the floor of the frontal fossa to help isolate the dura from the frontal sinus and to prevent CSF fistula.

## LEFORT FRACTURES

Complex fractures through inherently weak "cleavage planes" resulting in an unstable segment ("floating face"). Shown in Figure 27-5 (usually occur as variants of this basic scheme).

- **LeFort I:** transverse AKA transmaxillary fracture. Fracture line crosses pterygoid plate and maxilla just above the apices of the upper teeth. May enter maxillary sinus(es)
- **LeFort II:** pyramidal. Fracture extends upward across inferior orbital rim and orbital floor to medial orbital wall, then across nasofrontal suture. Often from downward blow to the nasal area
- **LeFort III:** craniofacial dislocation. Involves zygomatic arches, zygomaticofrontal suture, nasofrontal suture, pterygoid plates, and orbital floors (separating maxilla from cranium). Requires significant force, therefore often associated with other injuries, including brain injuries

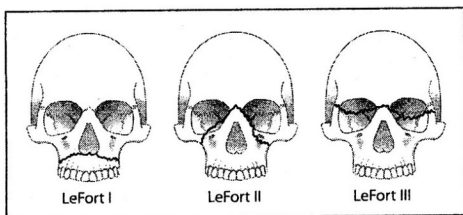


Figure 27-5 LeFort fractures

## PNEUMOCEPHALUS

AKA (intra)cranial aerocele, AKA pneumatocoele, is defined as the presence of intracranial gas. It is critical to distinguish this from **tension pneumocephalus** which is gas under pressure (*see below*). The gas may be located in any of the following compartments: epidural, subdural, subarachnoid, intraparenchymal, intraventricular.

**Presentation:** H/A in 38%, N/V, seizures, dizziness, and obtundation<sup>219</sup>. An intracranial succussion splash is a rare (occurring in ~ 7%) but pathognomonic finding. Tension pneumocephalus may additionally cause signs and symptoms just as any mass (may cause focal deficit or increased ICP).

### Etiologies of pneumocephalus:

Anything that can cause a CSF leak can produce associated pneumocephalus (*see page 301*).

1. skull defects
  - A. post neurosurgical procedure
    1. craniotomy: risk is higher when patient is operated in the sitting position<sup>220</sup>
    2. shunt insertion<sup>221, 222</sup>
    3. burr-hole drainage of chronic subdural hematoma<sup>223, 224</sup>: incidence is probably < 2.5%<sup>224</sup> although higher rates have been reported
  - B. posttraumatic
    1. fracture through air sinus: including basal skull fracture
    2. open fracture over convexity (usually with dural laceration)
  - C. congenital skull defects: including defect in tegmen tympani<sup>225</sup>
  - D. neoplasm (osteoma<sup>226</sup>, epidermoid<sup>227</sup>, pituitary tumor): usually caused by tumor erosion through floor of sella into sphenoid sinus
2. infection
  - A. with gas-producing organisms
  - B. mastoiditis
3. post invasive procedure:
  - A. lumbar puncture
  - B. ventriculostomy
  - C. spinal anesthesia<sup>228</sup>
4. spinal trauma (LP could be included here as well)
5. barotrauma<sup>229</sup>: e.g. with scuba diving (possibly through a defect in the tegmen tympani)
6. may be potentiated by a CSF drainage device in the presence of a CSF leak<sup>230</sup>

### Differential diagnosis (things that can mimic pneumocephalus):

Although intracranial low-density on CT may be associated with epidermoid, lipoma, or CSF, nothing is as intensely black as air. This can often be better appreciated on bone-windows than on soft-tissue windows.

### Tension pneumocephalus

Intracranial gas can develop elevated pressure in the following settings:

1. when nitrous oxide anesthesia is not discontinued prior to closure of the dura<sup>231</sup> (see *nitrous oxide* ( $N_2O$ ), page 2)
2. "ball-valve" effect due to an opening to the intracranial compartment with soft tissue (e.g. brain) that may permit air to enter but prevent exit of air or CSF
3. when trapped room temperature air expands with warming to body temperature: a modest increase of only  $\approx 4\%$  results from this effect<sup>232</sup>
4. in the presence of continued production by gas-producing organisms

### Diagnosis

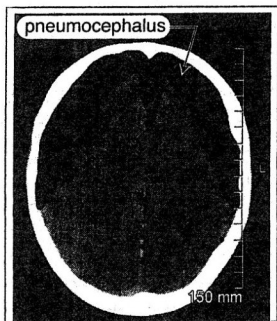
Pneumocephalus is most easily diagnosed on CT<sup>233</sup> which can detect quantities of air as low as 0.5 ml. Air appears dark black (darker than CSF) and has a Hounsfield coefficient of  $-1000$ . One characteristic finding with bilateral pneumocephalus is the **Mt. Fuji sign** in which the two frontal poles appear peaked and are surrounded by and separated by air, resembling the silhouette of the twin peaks of Mt. Fuji<sup>224</sup> (see Figure 27-6). Intracranial gas may also be evident on plain skull x-rays.

Since simple pneumocephalus usually does not require treatment, it is critical to differentiate it from tension pneumocephalus, which may need to be evacuated if symptomatic. It may be difficult to distinguish the two; brain that has been compressed e.g. by a chronic subdural may not expand immediately post-op and the "gas gap" may mimic the appearance of gas under pressure.

### Treatment

When pneumocephalus is due to gas-producing organisms, treatment of the primary infection is initiated and the pneumocephalus is usually followed.

Treatment of non-infectious simple pneumocephalus depends on the whether or not the presence of a CSF leak is suspected. If there is no leak the gas will be resorbed with time, and if the mass effect is not severe it may simply be followed. If a CSF leak is sus-



**Figure 27-6** Mt. Fuji sign with bilateral pneumocephalus. Axial noncontrast CT scan



pected, management is as with any CSF fistula (see *CSF fistula (cranial)*, page 300).

Treatment of significant or symptomatic post-op pneumocephalus by breathing 100% O<sub>2</sub> via a nonrebreather mask increases the rate of resorption<sup>234</sup> (100% FiO<sub>2</sub> can be tolerated for 24-48 hours without serious pulmonary toxicity<sup>235</sup>).

Tension pneumocephalus producing significant symptoms must be evacuated. The urgency is similar to that of an intracranial hematoma. Dramatic and rapid improvement may occur with the release of gas under pressure. Options include placement of new twist drill or burr holes, or insertion of a spinal needle through a pre-existing burr hole (e.g. following a craniotomy).

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## 27.4.4. Skull fractures in pediatric patients

This section deals with some special concerns of skull fractures in pediatrics. Also see *Child abuse*, page 918.

### POSTTRAUMATIC LEPTOMENINGEAL CYSTS

AKA **growing skull fractures**. Posttraumatic leptomeningeal cysts (PTLMC) are distinct from arachnoid cysts (AKA leptomeningeal cysts, which are not posttraumatic). PTLMC consists of a fracture line that widens with time. Although usually asymptomatic, the cyst may cause mass effect with neurologic deficit. Traumatic aneurysm is another rare complication<sup>236</sup>.

PTLMCs are very rare, occurring in 0.05-0.6% of skull fractures<sup>237, 238</sup>. Usually requires both a widely separated fracture AND a dural tear. Mean age at injury: < 1 year, over 90% occur before age 3 years<sup>239</sup> (formation may require the presence of a rapidly growing brain<sup>240</sup>) although rare adult cases have been described<sup>241-243</sup> (a total of 5 cases in the literature as of 1998<sup>243</sup>). Most often presents as scalp mass, although there are two reports of presentation with head pain alone<sup>241</sup>. PTLMC rarely occur > 6 mos out from the injury. Some children may develop a skull fracture that seems to grow during the initial few weeks, that is not accompanied by a subgaleal mass, and that heal spontaneously within several months; the term "pseudogrowing fracture" has been suggested for these<sup>244</sup>.

X-ray appearance: widening of fracture and scalloping (or saucerizing) of edges.

### TREATMENT

Treatment of true PTLMC is surgical, with dural closure mandatory. Since the dural defect is usually larger than the bony defect, it may be advantageous to perform a craniotomy around the fracture site, repair the dural defect, and replace the bone<sup>242</sup>. Pseudogrowing fractures should be followed with x-rays and operated only if expansion persists beyond several months or if a subgaleal mass is present.

### SCREENING FOR DEVELOPMENT OF PTLMC

If early growth of a fracture line with no subgaleal mass is noted, repeat skull films in 1-2 months before operating (to rule-out pseudogrowing fracture). In young patients with separated skull fractures (the width of the initial fracture is rarely mentioned), consider obtaining follow-up skull film 6-12 mos post-trauma. However, since most PTLMCs are brought to medical attention when the palpable mass is noticed, routine follow-up x-rays may not be cost-effective.

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## 27.5. Posttraumatic parenchymal injuries

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For techniques of decompressive craniectomies, see page 165.

### CEREBRAL EDEMA

Surgical decompression is occasionally an option (see *PRACTICE GUIDELINE 27-21*).

**Indications and timing for surgery**

**Level III**<sup>245</sup>: bifrontal decompressive craniectomy within 48 hrs of injury is a treatment option for patients with diffuse, medically refractory posttraumatic cerebral edema and associated IC-HTN

**DIFFUSE INJURIES**

Patients with severe diffuse injuries occasionally may be considered for decompressive craniectomy (see *PRACTICE GUIDELINE 27-22*).

**PRACTICE GUIDELINE 27-22 DIFFUSE INJURIES****Indications for surgery**

**Level III**<sup>245</sup>: decompressive craniectomy is an option for patients with refractory IC-HTN and diffuse parenchymal injury with clinical and radiographic evidence for impending transtentorial herniation

**27.5.1. Hemorrhagic contusion**

AKA traumatic intracerebral hemorrhage (**TICH**). Often considered as high density areas on CT (some exclude areas < 1 cm diameter<sup>246</sup>). TICH usually produce much less mass effect than their apparent size. Most commonly occur in areas where sudden deceleration of the head causes the brain to impact on bony prominences (e.g. temporal, frontal and occipital poles) in coup or contrecoup fashion.

TICH often enlarge and/or coalesce with time as seen on serial CTs. They also may appear in a delayed fashion (see *Delayed traumatic intracerebral hemorrhage (DTICH)* below). CT scans months later often show surprisingly minimal or no encephalomalacia.

**Treatment****PRACTICE GUIDELINE 27-23 SURGICAL MANAGEMENT OF TICH**

**Level III**<sup>245</sup>: Indications for surgery for TICH:

- surgical evacuation for:
  - A. progressive neurological deterioration referable to the TICH, medically refractory IC-HTN, or signs of mass effect on CT
  - B. or TICH volume > 50 cm<sup>3</sup>
  - C. or GCS = 6-8 with frontal or temporal TICH volume > 20 cm<sup>3</sup> with midline shift (MLS - see page 909) ≥ 5 mm and/or compressed basal cisterns on CT (see page 909)
- nonoperative management with intensive monitoring and serial imaging: may be used for TICH without neurologic compromise and no significant mass effect on CT and controlled ICP

**DELAYED TRAUMATIC INTRACEREBRAL HEMORRHAGE (DTICH)**

In patients with GCS ≤ 8, incidence is ≈ 10%<sup>247, 248</sup> (reported incidence varies with resolution of CT scanner<sup>41</sup>, timing of scan, and definition). Most DTICH occur within 72 hrs of the trauma<sup>248</sup>. Some patients seem to be doing well and then present with an apoplectic event (although DTICH accounted only for 12% of patients who "talk and deteriorate"<sup>249</sup>).

Factors that contribute to formation of DTICH include local or systemic coagulopathy, hemorrhage into an area of necrotic brain softening, coalescence of extravasated microhematomas<sup>250</sup>.

Treatment is the same as for TICH (see above).

Outcome for patients with DTICH described in the literature is generally poor, with a mortality ranging from 50-75%<sup>250</sup>.

## 27.6. Epidural hematoma

Incidence of epidural hematoma (EDH): 1% of head trauma admissions (which is ~ 50% the incidence of acute subdurals). Ratio of male:female = 4:1. Usually occurs in young adults, and is rare before age 2 yrs or after age 60 (perhaps because the dura is more adherent to the inner table in these groups).

Dogma was that a temporoparietal skull fracture disrupts the middle meningeal artery as it exits its bony groove at the pterion, causing arterial bleeding that dissects the dura from the inner table. Alternatively, dissection of the dura from the inner table may occur first, followed by bleeding into the space thus created.

Source of bleeding: 85% = arterial bleeding (the middle meningeal artery is the most common source of middle fossa EDHs). Many of the remainder of cases are due to bleeding from middle meningeal vein or dural sinus.

70% occur laterally over the hemispheres with their epicenter at the pterion, the rest occur in the frontal, occipital, and posterior fossa (5-10% each).

### PRESENTATION WITH EDH

"Textbook" presentation (< 10%-27% have this classical presentation<sup>68</sup>):

- brief posttraumatic loss of consciousness (LOC)
- followed by a "lucid interval" for several hours
- then, obtundation, contralateral hemiparesis, ipsilateral pupillary dilatation

Deterioration usually occurs over a few hours, but may take days and rarely, weeks (longer intervals may be associated with venous bleeding).

Other presenting findings: H/A, vomiting, seizure (may be unilateral), hemi-hyperreflexia + unilateral Babinski sign, and elevated CSF pressure (LP is seldom used any longer). Bradycardia is usually a late finding. In peds, EDH should be suspected if there is a 10% drop in hematocrit after admission.

Contralateral hemiparesis is not uniformly seen, especially with EDH in locations other than laterally over the hemisphere. Shift of the brain stem away from the mass may produce compression of the opposite cerebral peduncle on tentorial notch which can produce *ipsilateral* hemiparesis (so called **Kernohan's phenomenon** or Kernohan's notch phenomenon)<sup>251</sup>, a false localizing sign.

60% of patients with EDH have a dilated pupil, 85% of which are *ipsilateral*.

No initial loss of consciousness occurs in 60%. No lucid interval in 20%. Beware: lucid interval may also be seen in other conditions (including subdural hematoma).

**Differential diagnosis:** Includes a posttraumatic disorder described by Denny-Brown consisting of a "lucid interval" followed by bradycardia, brief periods of restlessness and vomiting, without intracranial hypertension or mass. Children especially may have H/A, and may become drowsy and confused. Theory: a form of vagal syncope, but CT must be done to rule-out EDH.

### EVALUATION

#### Plain skull x-rays

No fracture is identified in 40% of EDH. In these cases the patient's age was almost always < 30 yrs.

#### CT scan in EDH

"Classic" CT appearance occurs in 84% of cases: high density biconvex (lenticular) shape adjacent to the skull. In 11% the side against the skull is convex and that along the brain is straight, and in 5% it is crescent shaped (resembling subdural hematoma)<sup>252</sup>. An EDH may cross the falx (distinct from SDH which is limited to one side of the falx) but is usually limited by skull sutures. EDH usually has uniformly density, sharply defined edges on multiple cuts, high attenuation (undiluted blood), contiguous with inner table, usually confined to small segment of calvaria. Mass effect is frequent. Occasionally, an epidural may be isodense with brain and may not show up unless IV contrast is given<sup>252</sup>.

### MORTALITY WITH EDH

Overall: 20-55% (higher rates in older series). Optimal diagnosis and treatment

within few hours results in 5-10% estimated mortality (12% in a recent CT era series<sup>253</sup>). Mortality without lucid interval double that with. Bilateral Babinski's or decerebration pre-op → worse prognosis. Death is usually due to respiratory arrest from uncal herniation causing injury to the midbrain.

20% of patients with EDH on CT also have ASDH at autopsy or operation. Mortality with both lesions concurrently is higher, reported range: 25-90%.

## TREATMENT OF EDH

### MEDICAL

CT may detect small EDHs and can be used to follow them. However, in most cases, EDH is a surgical condition (see *Surgical indications and timing* (see **PRACTICE GUIDELINE 27-24**) below).

Nonsurgical management may be attempted in the following:

Small ( $\leq 1$  cm maximal thickness) subacute or chronic EDH<sup>254</sup>, with minimal neurological signs/symptoms (e.g. slight lethargy, H/A) and no evidence of herniation. Although medical management of p-fossa EDHs has been reported, these are more dangerous and surgery is recommended.

In 50% of cases there will be a slight transient increase in size between days 5-16, and some patients required emergency craniotomy when signs of herniation occurred<sup>255</sup>.

### Management

Management includes: admit, observe (in monitored bed if possible). Optional: steroids for several days, then taper. Follow-up CT: in 1 wk if clinically stable. Repeat in 1-3 mos if patient becomes asymptomatic (to document resolution). Prompt surgery if signs of local mass effect, signs of herniation (increasing drowsiness, pupil changes, hemiparesis...) or cardiorespiratory abnormalities.

### SURGICAL

#### Surgical indications and timing (see **PRACTICE GUIDELINE 27-24**)

EDH in pediatric patients is riskier than adults since there is less room for clot. Threshold for surgery in pediatrics should be very low.

#### PRACTICE GUIDELINE 27-24 SURGICAL MANAGEMENT OF EDH

##### Indications for surgery

###### Level III<sup>256</sup>:

- EDH volume  $> 30 \text{ cm}^3$  should be evacuated regardless of GCS
- EDH with the *all* of the following characteristics can be managed non-surgically with serial CT scans and close neurological observation in a neurosurgical center:
  - A. volume  $< 30 \text{ cm}^3$
  - B. *and* thickness  $< 15 \text{ mm}$
  - C. *and* with midline shift (MLS)  $< 5 \text{ mm}$  (see page 909)
  - D. *and* GCS  $> 8$
  - E. *and* no focal neurologic deficit

##### Timing of surgery

**Level III<sup>256</sup>:** it is strongly recommended that patients with an acute EDH and GCS  $< 9$  and anisocoria undergo surgical evacuation ASAP

### Booking the case - craniotomy for acute EDH/SDH

Also see defaults & disclaimers (page v).

1. position: (depends on location of bleed, usually supine)
2. blood: type & screen (for severe SDH: T & C 2 U PRBC)
3. post-op: ICU
4. consent (in lay terms for the patient - not all-inclusive):
  - A. procedure: surgery through the skull to remove blood clot, stop any bleeding identified, possible placement of intracranial pressure monitor
  - B. alternatives: nonsurgical management
  - C. complications: (usual craniotomy complications - see page v) *plus* further bleeding which may cause problems (especially in patients taking blood thinners, antiplatelet drugs including aspirin, or those with coagulation ab-



normalities or previous bleeds) and may require further surgery, any permanent brain injury that has already occurred is not likely to recover, hydrocephalus

### **Surgical technical issues**

Evacuation is performed in the O.R. unless the patient herniates in E/R. Objectives:

1. clot removal: lowers ICP and eliminates focal mass effect. Blood is usually thick coagulum, thus exposure must provide access to most of clot. Craniotomy permits more complete evacuation of hematoma than e.g. burr holes<sup>256</sup>
2. hemostasis: coagulate bleeding soft tissue (dural veins & arteries). Apply bone wax to intra-diploic bleeders (e.g. middle meningeal artery). Also requires large exposure
3. prevent reaccumulation: (some bleeding may recur, and dura is now detached from inner table) place dural tack-up sutures to edges of craniotomy and use central "tenting" suture

### **DELAYED EPIDURAL HEMATOMA (DEDH)**

**Definition:** an EDH that is not present on the initial CT scan, but is found on subsequent CT. Comprise 9-10% of all EDHs in several series<sup>257, 258</sup>.

Theoretical risk factors for DEDH include the following (NB: many of these risk factors may be incurred after the patient is admitted following a negative initial CT):

1. lowering ICP either medically (e.g. osmotic diuretics) and/or surgically (e.g. evacuating contralateral hematoma) which reduces tamponading effect
2. rapidly correcting shock (hemodynamic "surge" may cause DEDH)<sup>259</sup>
3. coagulopathies

DEDH have been reported in mild head injury (GCS > 12) infrequently<sup>260</sup>. Presence of a skull fracture has been identified as a common feature of DEDH<sup>260</sup>.

Key to diagnosis: high index of suspicion. Avoid a false sense of security imparted by an initial "nonsurgical" CT. 6 of 7 patients in one series improved or remained unchanged neurologically despite enlarging EDH (most eventually deteriorate). 1 of 5 with an ICP monitor did not have a heralding increase in ICP. May develop once an intracranial lesion is surgically treated, as occurred in 5 of 7 patients within 24 hrs of evacuation of another EDH. 6 of 7 patients had known skull fractures in the region where the delayed EDH developed<sup>258</sup>, but none of 3 had a skull fracture in another report<sup>259</sup>.

### **POSTERIOR FOSSA EPIDURAL HEMATOMA**

Comprise ~ 5% of EDH<sup>261, 262</sup>. More common in 1st two decades of life. Although as many as 84% have occipital skull fractures, only ~ 3% of children with occipital skull fractures develop p-fossa EDH. The source of bleeding is usually not found, but there is a high incidence of tears of the dural sinuses. Cerebellar signs are surprisingly lacking or subtle in most. For surgical indications, *see page 905*. Overall mortality is ~ 26% (mortality was higher in patients with an associated intracranial lesion).

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## **27.7. Subdural hematoma**

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### **27.7.1. Acute subdural hematoma**

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The magnitude of impact damage (as opposed to secondary damage, *see page 854*) is usually much higher in acute subdural hematoma (**ASDH**) than in epidural hematomas, which generally makes this lesion much more lethal. There is often associated underlying brain injury, which may be less common with EDH. Symptoms may be due to compression of the underlying brain with midline shift, in addition to parenchymal brain injury and possibly cerebral edema<sup>263, 264</sup>.

Two common causes of traumatic ASDH:

1. accumulation around parenchymal laceration (usually frontal or temporal lobe). There is usually severe underlying primary brain injury. Often no "lucid inter-

- val". Focal signs usually occur later and are less prominent than with EDH
- surface or bridging vessel torn from cerebral acceleration-deceleration during violent head motion. With this etiology, primary brain damage may be less severe, a lucid interval may occur with later rapid deterioration

ASDH may also occur in patients receiving anticoagulation therapy<sup>265, 266</sup>, usually with but sometimes without a history of trauma (the trauma may be minor). Receiving anticoagulation therapy increases the risk of ASDH 7-fold in males and 26-fold in females<sup>265</sup>.

### CT SCAN IN ASDH

Crescentic mass of increased attenuation adjacent to inner table. Edema is often present. Usually over convexity, but may also be interhemispheric, along tentorium, or in p-fossa. Membrane formation begins by about 4 days after injury<sup>267</sup>. Changes with time on CT (see Table 27-25): isodense after  $\approx$  2 wks, only clues may be obliteration of sulci and lateralizing shift, the latter may be absent if bilateral. Subsequently becomes hypodense to brain (see *Chronic subdural hematoma*, page 899).

**Table 27-25 ASDH density changes on CT with time**

Category	Time frame	Density on CT
acute	1 to 3 days	hyperdense
subacute	4 days to 2 or 3 wks	$\approx$ isodense
chronic	usually > 3 wks and < 3-4 mos	hypodense (approaching density of CSF)
	after about 1-2 months	may become lenticular shaped (similar to epidural hematoma) with density > CSF, < fresh blood

Differences from EDH: SDH is more diffuse, less uniform, usually concave over brain surface, often less dense (from mixing with CSF<sup>268</sup>), and bridging subdural veins (from brain surface to the skull) may be seen.

### TREATMENT

Level III surgical indications are shown in *PRACTICE GUIDELINE 27-25*. Other factors that should be considered:

- presence of anticoagulants or platelet inhibitors: patients in good neurologic condition may be better served by reversing these agents prior to operating (to increase the safety of surgery)
- location of hematoma: in general, a SDH high over the convexity is less threatening than a temporal/parietal SDH of the same volume that also has MLS
- patient's baseline level of function, DNR status...
- while the guidelines suggest evacuating SDH < 10 mm thick in some circumstances, clots that are smaller than this may simply be an epiphenomenon

#### **PRACTICE GUIDELINE 27-25 SURGICAL MANAGEMENT OF ASDH**

##### **Indications for surgery**

###### **Level III<sup>268</sup>:**

- ASDH with thickness > 10 mm or midline shift (MLS) > 5 mm (on CT) should be evacuated regardless of GCS
- ASDH with thickness < 10 mm\* and MLS < 5 mm should undergo surgical evacuation if:
  - GCS drops by  $\geq$  2 points from injury to admission
  - and/or the pupils are asymmetric or fixed and dilated
  - and/or ICP is > 20 mm Hg
- monitor ICP in all patients with ASDH and GCS < 9

##### **Timing of surgery**

**Level III<sup>268</sup>:** ASDH meeting surgical criteria should be evacuated ASAP†

##### **Surgical methods**

**Level III<sup>268</sup>:** ASDH meeting the above criteria for surgery should be evacuated via craniotomy‡ with or without bone flap removal and duraplasty

\* see text regarding the evacuation of ASDH < 10 mm thick

† for issues regarding timing of surgery, see text

‡ a large craniotomy flap is often required to evacuate the thick coagulum and to gain access to possible bleeding sites.

## Booking the case - acute subdural hematoma

As for acute epidural hematoma (see page 895).



### Technical considerations

The actual bleeding site is often not identified at the time of surgery. One may start with a small linear dural opening to effect clot removal and enlarge it as needed and only if brain swelling seems controllable.

### MORBIDITY AND MORTALITY WITH ASDH

#### Mortality

Range: 50-90% (a significant percentage of this mortality is from the underlying brain injury, and not the ASDH itself).

Traditionally thought to be higher in aged patients (60%). 90-100% in patients on anticoagulants<sup>266</sup>.

#### "Four hour rule"

Based on a 1981 series of 82 patients with ASDH<sup>269</sup>, it had been widely held that:

1. patients operated within 4 hrs of injury had 30% mortality, compared to 90% mortality if surgery was delayed > 4 hrs
2. functional survival (Glasgow Outcome Scale  $\geq 4$ , see Table 34-3, page 1183) rate of 65% could be achieved with surgery within 4 hrs
3. other factors related to outcome in this series include:
  - A. post-op ICP: 79% of patients with functional recovery had post-op ICPs that didn't exceed 20 mm Hg, whereas only 30% of patients who died had ICP < 20 mm Hg
  - B. initial neuro exam
  - C. age was not a factor (ASDH tend to occur in older patients than EDH)

However, the magnitude of the importance of rapid surgical treatment is still controversial. A study of 101 patients with ASDH found overall mortality of 66%, and functional recovery of 19%<sup>270</sup>. Postoperative seizures occurred in 9%, and did not correlate with outcome. The following was determined:

1. delay to surgery: delays > 4 hours increased mortality from 59% to 69% and decreased functional survival (Glasgow Outcome Scale  $\geq 4$ , see page 1183) from 26% to 16%. These differences suggested a trend but were not statistically significant
2. the following variables were identified as strongly influencing outcome:
  - A. mechanism of injury: the worst outcome was with motorcycle accidents, with 100% mortality in unhelmeted patients, 33% in helmeted
  - B. age: correlated with outcome only > 65 yrs age, with 82% mortality and 5% functional survival in this group (other series had similar results<sup>271</sup>)
  - C. neurologic condition on admission: the ratio of mortality to functional survival rate related to the admission Glasgow Coma Scale (GCS) is shown in Table 27-26
  - D. postoperative ICP: patients with peak ICPs < 20 mm Hg had 40% mortality, and no patient with ICP > 45 had a functional survival

Of all the above factors, only the time to surgery and postoperative ICP can be directly influenced by the treating neurosurgeon.

**Table 27-26 Outcome as related to admission GCS**

GCS	Mortality	Functional survival
3	90%	5%
4	76%	10%
5	62%	18%
6 & 7	51%	44%

### INTERHEMISPHERIC SUBDURAL HEMATOMA

Subdural hematoma along the falx between the two cerebral hemispheres (older term: interhemispheric scissure).

May occur in children<sup>272</sup>, possibly associated with child abuse<sup>273</sup>.

In adults, a consequence of head trauma in 79-91%, ruptured aneurysm<sup>274</sup> in ~ 12%, surgery in the vicinity of the corpus callosum, and rarely spontaneously<sup>275</sup>.

Incidence is unknown. Spontaneous cases should be investigated for possible underlying aneurysm. Occasionally may be bilateral, sometimes may be delayed (see below)

Most often are asymptomatic, or may present with the so-called "falx syndrome" - paresis or focal seizures contralateral to the hematoma. Other presentations: gait ataxia, dementia, language disturbance, oculomotor palsies.

## Treatment

Controversial. Small asymptomatic cases may be managed expectantly. Surgery should be considered for progressive neurological deterioration. Approached through a parasagittal craniotomy. ✖ Surgery for these lesions can be treacherous - there is risk of venous infarction and often you find you are dealing with a superior sagittal sinus injury.

## Outcome

Reported mortality: 25-42%. Mortality is higher in the presence of altered levels of consciousness. Mortality rate may actually be lower (24%) than with all-comers<sup>275</sup>. This is significantly lower than SDH in other sites (*see above*).

## DELAYED ACUTE SUBDURAL HEMATOMA (DASDH)

DASDHs have received less attention than delayed epidural or intraparenchymal hematomas. Incidence is  $\approx 0.5\%$  of operatively treated ASDHs<sup>250</sup>.

**Definition:** ASDH not present on an initial CT (or MRI) that shows up on a subsequent study. Indications for treatment are the same as for ASDH. Neurologically stable patients with a small DASDH and medically controllable ICP are managed expectantly.

## INFANTILE ACUTE SUBDURAL HEMATOMA

Infantile acute subdural hematoma (**IASDH**) is often considered as a special case of SDH. Roughly defined as an acute SDH in an infant due to minor head trauma without initial loss of consciousness or cerebral contusion<sup>276</sup>, possibly due to rupture of a bridging vein. The most common trauma is a fall backwards from sitting or standing. The infants will often cry immediately and then (usually within minutes to 1 hour) develop a generalized seizure. Patients are usually < 2 yrs old (most are 6-12 mos, the age when they first begin to pull themselves up or walk)<sup>277</sup>.

These clots are rarely pure blood, and are often mixed with fluid. 75% are bilateral or have contralateral subdural fluid collections. It is speculated that IASDH may represent acute bleeding into a preexisting fluid collection<sup>277</sup>.

Skull fractures are rare. In one series, retinal and preretinal hemorrhages were seen in all 26 patients<sup>276</sup>.

## Treatment

Treatment is guided by clinical condition and size of hematoma. Minimally symptomatic cases (vomiting, irritability, no altered level of consciousness and no motor disturbance) with liquefied hematoma may be treated with percutaneous subdural tap, which may be repeated several times as needed. Chronically persistent cases may require a subduroperitoneal shunt.

More symptomatic cases with high density clot on CT require craniotomy. A subdural membrane similar to those seen in adult chronic SDH is not unusual<sup>277</sup>. Caution: these patients are at risk of developing intraoperative hypovolemic shock.

## Outcome

8% morbidity and mortality rate in one series<sup>276</sup>. Much better prognosis than ASDH of all ages probably because of the absence of cerebral contusion in IASDH.

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## 27.7.2. Chronic subdural hematoma

Chronic subdural hematomas (**CSDH**) generally occur in the elderly, with the average age being  $\approx 63$  yrs (exception: subdural collections of infancy, *see page 904*). Head trauma is identified in < 50% (sometimes rather trivial trauma). Other risk factors: alcohol abuse, seizures, CSF shunts, coagulopathies (including therapeutic anticoagulation<sup>266</sup>), and patients at risk for falls (e.g. with hemiplegia from previous CVA). CSDHs are bilateral in  $\approx 20$ -25% of cases<sup>278, 279</sup>.

Hematoma thickness tends to be larger in older patients due to a decrease in brain weight and increase in subdural space with age<sup>280</sup>.

Classically CSDHs contains dark "motor oil" fluid which does not clot<sup>281</sup>. When the subdural fluid is clear (CSF), the collection is termed a subdural hygroma (*see page 903*).

## Pathophysiology

Many CSDH probably start out as acute subdurals. Blood within the subdural space



evokes an inflammatory response. Within days, fibroblasts invade the clot, and form neomembranes on the inner (cortical) and outer (dural) surface. This is followed by ingrowth of neocapillaries, enzymatic fibrinolysis, and liquefaction of blood clot. Fibrin degradation products are reincorporated into new clots and inhibit hemostasis. The course of CSDH is determined by the balance of plasma effusion and/or rebleeding from the neomembranes on the one hand and reabsorption of fluid on the other<sup>282, 283</sup>.

## Presentation

Patients may present with minor symptoms of headache, confusion, language difficulties (e.g. word-finding difficulties or speech arrest, usually with dominant hemisphere lesions), or TIA-like symptoms (*see page 1201*). Or, they may develop varying degrees of coma, hemiplegia, or seizures (focal, or less often generalized). Often, the diagnosis may be unexpected prior to imaging. Specialized clinical grading systems have been published, but are not widely used.

## TREATMENT

1. seizure prophylaxis: used by some. Fully load with phenytoin (17 mg/kg slow IV, *see phenytoin (PHT) (Dilantin®)*, page 409) and follow with 100 mg slow IV q 8 hrs. It may be safe to discontinue after a week or so if there are no seizure. If late seizure occurs with or without prior use of AEDs, longer-term therapy is required. Some feel that the incidence of side effects from AEDs approximates the incidence of seizures and therefore they do not recommend prophylactic AEDs
2. coagulopathies (and iatrogenic anticoagulation) should be reversed
3. surgical evacuation of hematoma indicated for:
  - A. symptomatic lesions: including focal deficit, mental status changes...
  - B. or subdurals with maximum thickness greater than ~ 1 cm

## SURGICAL CONSIDERATIONS

### Booking the case - craniotomy: for chronic subdural

Also see defaults & disclaimers (*page v*).

1. position: (usually supine), horseshoe headrest
2. post-op: ICU
3. consent (in lay terms for the patient - not all-inclusive):
  - A. procedure: surgery through the skull to remove blood clot, stop any bleeding identified, placement a drainage tube to allow further fluid to drain after surgery for a day or so
  - B. alternatives: nonsurgical management
  - C. complications: (usual craniotomy complications - *see page v*) *plus* further bleeding which may cause problems (especially in patients taking blood thinners, antiplatelet drugs including aspirin, or those with coagulation abnormalities or previous bleeds) and may require further surgery, hydrocephalus



### Surgical options

There is not uniform agreement on the best method to treat CSDHs. For details of techniques (burr holes, whether or not to use subdural drain...) *see below*.

1. placing two burr holes, and irrigating through and through with tepid saline until the fluid runs clear
2. single "large" burr hole with irrigation and aspiration: *see below*
3. single burr hole drainage with placement of a subdural drain, maintained for 24-48 hrs (removed when output becomes negligible)
4. twist drill craniostomy: *see below* (note that small "twist drill" drainage without subdural drain has higher recurrence rate than e.g. burr holes)
5. formal craniotomy with excision of subdural membrane (may be necessary in cases which persistently recur after above procedures, possibly due to seepage from the subdural membrane). Still a safe and valid technique<sup>284</sup>. No attempt should be made to remove the deep membrane adherent to the surface of brain

Techniques that promote continued drainage after the immediate procedure and that may thus reduce residual fluid and prevent reaccumulation:

1. use of a subdural drain: (*see below*)
2. using a generous burr hole under the temporalis muscle: (*see below*)
3. bed-rest restriction with the head of the bed flat (1 pillow is permitted) with mild overhydration for 24-48 hours post-op (or if a drain is used, until 24-48 hours after

it is removed). May promote expansion of the brain and expulsion of residual subdural fluid. Allowing patients to sit up to 30-40° immediately post-op was associated with higher radiographic recurrence rate (2.3% for those kept flat, vs. 19% for those who sat up) but usually did not require reoperation<sup>285</sup>

4. some advocate continuous lumbar subarachnoid infusion when the brain fails to expand, however there are possible complications<sup>223</sup>

#### *TWIST DRILL CRANIOSTOMY FOR CHRONIC SUBDURALS*

This method is thought to decompress the brain more slowly and avoids the presumed rapid pressure shifts that occurs following other methods, which may be associated with complications such as intraparenchymal (intracerebral) hemorrhage. May even be performed at the bedside under local anesthesia.

A 0.5 cm incision is made in the scalp in the rostral portion of the hematoma, and then a twist drill hole is placed at a 45° angle to the skull, aimed in the direction of the longitudinal axis of the collection. If the drill does not penetrate the dura, this is done with an 18 Ga. spinal needle. A ventricular catheter is inserted into the subdural space, and is drained to a standard ventriculostomy drainage bag maintained 20 cm below the level of the craniostomy site<sup>286-288</sup> (see *Subdural drain* below). The patient is kept flat in bed (see *above*). Serial CTs assess the adequacy of drainage. The catheter is removed when at least ~ 20% of the collection is drained and when the patient shows signs of improvement, which occurs within a range of 1-7 days (mean of 2.1 days). Some include a low pressure shunt valve in the system to prevent reflux of fluid or air.

#### *BURR HOLES FOR CHRONIC SUBDURAL HEMATOMAS*

To prevent recurrence, the use of small burr holes (without a subdural drain) is not recommended. A generous (> 2.5 cm diameter - it is recommended that one actually measure this) subtemporal craniectomy should be performed, and bipolar coagulation is used to shrink the edges of the dura and subdural membrane back to the full width of the bony opening (do not try to separate these two layers as this may promote bleeding). This allows continued drainage of fluid into the temporalis muscle. A piece of Gelfoam® may be placed over the opening to help prevent fresh blood from oozing into the opening.

#### *SUBDURAL DRAIN*

Use of a subdural drain is associated with a decrease in need for repeat surgery from 19% to 10%<sup>289</sup>. If a subdural drain is used, a closed drainage system is recommended. Difficulties may occur with ventriculostomy catheters because the holes are small and are restricted to the tip region (so-designed to keep choroid plexus from plugging the catheter when inserted into the ventricles when used as intended as a CSF shunt), especially with thick "oily" fluid (on the positive side, slow drainage may be desirable). The drainage bag is maintained ~ 50-80 cm below the level of the head<sup>288, 290</sup>. An alternative is a small Jackson-Pratt® drain using "thumb-print" indentation of the suction bulb which provides good drainage with a self-contained one-way valve (however, there may be a risk of excessive negative pressure with overcompression of the bulb).

Post-op, the patient is kept flat (see *above*). Prophylactic antibiotics may be given until ~ 24-48 hrs following removal of the drain, at which time the HOB is gradually elevated. CT scan prior to removal of the drain (or shortly after removal) may be helpful to establish a baseline for later comparison in the event of deterioration.

There is a case report of administration of urokinase through a subdural drain to treat reaccumulation of clot following evacuation<sup>291</sup>.

#### **OUTCOME**

There is clinical improvement when the subdural pressure is reduced to close to zero, which usually occurs after ~ 20% of the collection is removed<sup>288</sup>.

Patients who have high subdural fluid pressure tend to have more rapid brain expansion and clinical improvement than patients with low pressures<sup>290</sup>.

Residual subdural fluid collections after treatment are common, but clinical improvement does not require complete resolution of the fluid collection on CT. CTs showed persistent fluid in 78% of cases on post-op day 10, and in 15% after 40 days<sup>290</sup>, and may take up to 6 months for complete resolution. Recommendation: do not treat persistent fluid collections evident on CT (especially before ~ 20 days post-op) unless it increases in size on CT or if the patient shows no recovery or deteriorates.

76% of 114 patients were successfully treated with a single drainage procedure us-

ing a twist drill craniostomy with subdural ventricular catheter, and 90% with one or two procedures<sup>286</sup>. These statistics are slightly better than twist drill craniostomy with aspiration alone (i.e. no drain).

### Complications of surgical treatment

Although these collections often appear innocuous, severe complications may occur, and include:

1. seizures (including intractable status epilepticus)
2. intracerebral hemorrhage (ICH): occurs in 0.7-5%<sup>292</sup>. Very devastating in this setting: one third of these patients die and one third are severely disabled
3. failure of the brain to re-expand and/or reaccumulation of the subdural fluid
4. tension pneumocephalus
5. subdural empyema: may also occur with untreated subdurals<sup>293</sup>

In 60% of patients  $\geq$  age 75 yrs (and in no patients  $<$  75 yrs), rapid decompression is associated with **hyperemia** in the cortex immediately beneath the hematoma, which may be related to the complications of ICH or seizures<sup>292</sup>. All complications are more common in elderly or debilitated patients.

Overall mortality with surgical treatment for CSDH is 0-8%<sup>292</sup>. In a series of 104 patients treated mostly with craniostomy<sup>294</sup>, mortality was  $\approx$  4%, all of which occurred in patients  $>$  60 yrs old and were due to accompanying disease. Another large personal series reported 0.5% mortality<sup>295</sup>. Worsening of neurologic status following drainage occurs in  $\approx$  4%<sup>294</sup>.

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## 27.7.3. Spontaneous subdural hematoma

Occasionally patients with no identifiable trauma will present with severe H/A with or without associated findings (nausea, seizures, lethargy, focal findings including possible ipsilateral hemiparesis<sup>296</sup>...) and CT or MRI discloses a subdural hematoma that may be acute, subacute or chronic in appearance. The onset of symptoms is often sudden<sup>296</sup>.

### RISK FACTORS

Risk factors identified in a review of 21 cases in the literature<sup>297</sup> include:

1. hypertension: present in 7 cases
2. vascular abnormalities: arteriovenous malformation (AVM), aneurysm<sup>298</sup>
3. neoplasm
4. infection: including meningitis, tuberculosis
5. substance abuse: alcoholism, cocaine<sup>299</sup>
6. hypovitaminosis: especially vitamin C deficiency<sup>300</sup>
7. coagulopathies, including:
  - A. iatrogenic (anticoagulation e.g. with warfarin)
  - B. Ginkgo biloba (GB) extract: EGb761 and LI1379. Contains ginkgolides (especially Type B) which are inhibitors of platelet activating factor (PAF) at high concentrations<sup>301</sup>, also cause vasodilation and decreased blood viscosity. There have been case reports showing temporal relationship of hemorrhage to intake of GB<sup>302</sup>, especially at higher doses over long periods of time. However, no consistent alteration was demonstrable in 29 measurable coagulation/clotting variables after 7 days<sup>303</sup> (bleeding time was mildly prolonged in some case reports<sup>302, 304</sup>). Some individuals may possibly be more susceptible to the supplement, and there may be as-yet uncharacterized interactions with other entities (such as alcohol, aspirin...) but studies so far have been unrevealing<sup>305</sup>
  - C. factor XIII deficiency (protransglutaminase)<sup>306, 307</sup>. In peds: history may include report of bleeding from umbilical cord at birth. Check factor XIII levels as coagulation parameters may be normal or only slightly elevated
8. seemingly innocuous insults (e.g. bending over) or injuries resulting in no direct trauma to the head (e.g. whiplash injuries)
9. intracranial hypotension: spontaneous, after epidural anesthesia, lumbar puncture, or VP shunt<sup>308, 309</sup>

### ETIOLOGY

The bleeding site was determined in 14 of the 21 cases, and was arterial in each, typically involving a cortical branch of the MCA in the area of the sylvian fissure<sup>297</sup> where

there is a large number of branches to a wide cortical area.

Possible mechanisms for arterial rupture in idiopathic acute subdural hematoma (ASDH) include tears occur secondary to sudden head movements or trivial head trauma of the following<sup>310, 311</sup>:

1. small artery at perpendicular branch point off a cortical artery
2. small artery connecting the dura and cortex
3. adhesions between cortical artery and dura

#### TREATMENT

As for traumatic SDH. If symptomatic and/or  $> 1$  cm thick, surgical evacuation is the treatment of choice. For subacute to chronic subdurals, burr-hole evacuation is usually adequate (see above). For acute SDH, a craniotomy is usually required, and should expose the sylvian fissure to identify bleeding point(s). Microsurgical repair of arterial wall has been described<sup>311</sup>.

## 27.7.4. Traumatic subdural hygroma

From the Greek *hygros* meaning wet. AKA traumatic subdural effusion, AKA hydro-ma. Excess fluid in the subdural space (may be clear, blood tinged, or xanthochromic and under variable pressure) is almost always associated with head trauma, especially alcohol-related falls or assaults<sup>312</sup>. Skull fractures were found in 39% of cases. Distinct from chronic subdural hematoma, which is usually associated with underlying cerebral contusion, and usually contains darker clots or brownish fluid ("motor oil" fluid), and may show membrane formation adjacent to inner surface of dura (hygromas lack membranes).

"Simple hygroma" refers to a hygroma without significant accompanying conditions. "Complex hygroma" refers to hygromas with associated significant subdural hematoma, epidural hematoma, or intracerebral hemorrhage.

On CT, the density of the fluid is similar to that of CSF.

#### PATHOGENESIS

Mechanism of formation of hygroma is probably a tear in the arachnoid membrane with resultant CSF leakage into the subdural compartment. Hygroma fluid contains pre-albumin, which is also found in CSF but not in subdural hematomas. The most likely locations of arachnoid tears are in the sylvian fissure or the chiasmatic cistern. Another possible mechanism is post-meningitis effusion (especially influenza meningitis).

May be under high pressure. May increase in size (possibly due to a flap-valve mechanism) and exert mass effect, with the possibility of significant morbidity. Cerebral atrophy was present in 19% of patients with simple hygromas.

**Table 27-27 Major clinical features of traumatic subdural hygromas<sup>312</sup>**

Type of hygroma	Simple	Complex	Total
number of patients	66	14	80
spontaneous eye opening	74%	57%	71%
disorientation or stupor	65%	57%	64%
mental status change without focal signs	52%	50%	51%
neurological plateau with deficit or delayed deterioration	42%	7%	36%
seizures (usually generalized)	36%	43%	38%
hemiparesis	32%	21%	30%
neck stiffness	26%	14%	24%
anisocoria (maintained light reflex)	15%	7%	14%
headache	14%	14%	14%
alert (no mental status change)	8%	0%	6%
hemiplegia	6%	14%	8%
comatose (responsive to pain only)	3%	43%	10%

#### PRESENTATION

Table 27-27 shows clinical findings of subdural hygromas. Many present without focal findings. Complex hygromas usually present more acutely and require more urgent treatment.

#### TREATMENT

Asymptomatic hygromas do not require treatment. Recurrence following simple burr-hole drainage is common. Many surgeons maintain a subdural drain for 24-48 hrs

post-op. Recurrent cases may require either a craniotomy to locate the site of CSF leak (may be very difficult), or a subdural-peritoneal shunt may be placed.

## OUTCOME

Outcome may be more related to accompanying injuries than to the hygroma itself.

5 of 9 patients with complex hygromas and subdural hematoma died. For simple hygromas, morbidity was 20% (12% for decreased mental status without focal findings, 32% if hemiparesis/plegia was present).

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## 27.7.5. Extraaxial fluid collections in children

### Differential diagnosis

1. benign subdural collection in infants (*see below*)
2. chronic symptomatic extraaxial fluid collections or effusions (*see below*)
3. cerebral atrophy: should not contain xanthochromic fluid with elevated protein
4. "external hydrocephalus": ventricles often enlarged, fluid is CSF (*see page 307*)
5. normal variant of enlarged subarachnoid spaces and interhemispheric fissure
6. acute subdural hematoma: high density (fresh blood) on CT (occasionally these will appear as low density collections in children with low hematocrits). Will usually be unilateral (the others above are usually bilateral). These lesions may occur as birth injuries, and typically present with seizures, pallor, tense fontanelle, poor respirations, hypotension, and retinal hemorrhages
7. "craniocerebral disproportion" (head too large for the brain)<sup>313</sup>: extracerebral spaces enlarged up to 1.5 cm in thickness and filled with CSF-like fluid (possibly CSF), ventricles at upper limits of normal, deep sulci, widened interhemispheric fissure, normal intracranial pressure. Patients are developmentally normal. May be the same as benign extra-axial fluid of infancy (*see below*). Making this diagnosis with certainty is difficult in first few months of life

### BENIGN SUBDURAL COLLECTIONS OF INFANCY

Benign subdural collections (or effusions) of infancy<sup>314, 315</sup>, are perhaps better characterized by the term **benign extra-axial fluid collections of infancy**, since it is difficult to distinguish whether they are subdural or subarachnoid<sup>316</sup>. They appear on CT as peripheral hypodensities over the frontal lobes in infants. Imaging may also show dilatation of the interhemispheric fissure, cortical sulci<sup>317</sup>, and sylvian fissure. Ventricles are usually normal or slightly enlarged, with no evidence of transependymal absorption. Brain size is normal. Transillumination is increased over both frontal regions. The fluid is usually clear yellow (xanthochromic) with high protein content. The etiology of these is unclear, some cases may be due to perinatal trauma. They are more common in term infants than preemies. Must be differentiated from external hydrocephalus (*see page 307*).

**Presentation:** Mean age of presentation is ~ 4 months<sup>316</sup>.

May show: signs of elevated intracranial pressure (tense or large fontanelle, accelerated head growth crossing percentile curves), developmental delay usually as a result of poor head control due to the large size (developmental delay without macrocrania runs counter to the concept of "benign"<sup>316</sup>), frontal bossing, jitteriness. The poor head control may lead to positional flattening. Other symptoms, such as seizures (possibly focal) are indicative of symptomatic collections (*see below*). Large collections in the *absence* of macrocrania are more suggestive of cerebral atrophy.

**Treatment:** Most cases gradually resolve spontaneously, often within 8-9 months. A single subdural tap (*see page 201*) for diagnostic purposes (to differentiate from cortical atrophy and to rule out infection) may be done, and may accelerate the rate of disappearance. Repeat physical exams with OFC measurements should be done at ~ 3-6 month intervals. Head growth usually parallels or approaches normal curves by ~ 1-2 yrs age, and by 30-36 months orbital-frontal head circumference (OFC) approaches normal percentiles for height and weight. They usually catch up developmentally as OFCs normalize.

## SYMPTOMATIC CHRONIC EXTRAAXIAL FLUID COLLECTIONS IN CHILDREN

Variously classified as hematomas (chronic subdural hematoma), effusions, or hygromas, with differing definitions associated with each. Since the appearance on imaging and the treatment is similar, Litofsky et al. proposed that they all be classified as extraaxial fluid collections<sup>318</sup>. The difference between these lesions and "benign" subdural effusions (*see above*) may simply be the degree of clinical manifestation.

### Etiologies

The following etiologies were listed in a series of 103 cases<sup>318</sup>:

1. 36% were thought to be the result of trauma (22 were victims of child abuse)
2. 22% followed bacterial meningitis (post-infectious)
3. 19 occurred after placement or revision of a shunt (*see page 327*)
4. no cause could be identified in 17 patients

Other causes include<sup>313</sup>:

1. tumors: extracerebral or intracerebral
2. post-asphyxia with hypoxic brain damage and cerebral atrophy
3. defects of hemostasis: vitamin K deficiency...

### Signs and symptoms

Symptoms include: seizure (26%), large head (22%), vomiting (20%), irritability (13%), lethargy (13%), headache (older children), poor feeding, respiratory arrest...

Signs include: full fontanelle (30%), macrocrania (25%), fever (17%), lethargy (13%), hemiparesis (12%), retinal hemorrhages, coma, papilledema, developmental delay...

### Evaluation

CT/MRI usually shows ventricular compression and obliteration of the cerebral sulci, unlike with benign subdural collections. The "cortical vein sign" (*see page 308*) helps distinguish this from external hydrocephalus.

### Treatment

Options include:

1. observation: follow-up with serial OFC measurements, ultrasound and CT/MRI
2. serial percutaneous subdural taps (*see page 201*): some patients require as many as 16 taps<sup>319</sup>. Some series show good results and others show low success rate<sup>320, 321</sup>
3. burr hole drainage: may include long-term external drainage. Simple burr hole drainage may not be effective with severe cranioccephalic disproportion as the brain will not expand to obliterate the extra-axial space
4. subdural-peritoneal shunt: unilateral shunt is usually adequate even for bilateral effusions<sup>318, 321, 322</sup> (no study is required to demonstrate communication between the 2 sides<sup>318, 323</sup>). An extremely low pressure system should be utilized. The general practice is to remove the shunt after 2-3 months of drainage (once the collections are obliterated) to reduce the risk of associated mineralization of the dura and arachnoid and possible risk of seizures (these shunts are easily removed at this time, but may be more difficult to remove at a later date)<sup>324</sup>

Other recommendations:

At least one percutaneous tap should be performed to rule-out infection.

Many authors recommend observation for the patient with no symptoms or with only enlarging head and developmental delay.

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## 27.8. Traumatic posterior fossa mass lesions

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Less than 3% of head injuries involve the posterior fossa<sup>325</sup>. Epidural hematomas constitute the majority of these (*see page 896*). Other entities (subdural hematoma, intraparenchymal hematoma<sup>326</sup>) comprise the small remainder. **PRACTICE GUIDELINE 27-26** shows surgical management recommendations. Any of these can cause hydrocephalus<sup>325</sup>.

Most parenchymal hemorrhages managed nonsurgically were < 3 cm diameter.

## Indications for surgery

### Level III<sup>327</sup>:

- symptomatic posterior fossa mass lesions or those with mass effect on CT\* should be surgically removed
- asymptomatic lesions without mass effect on CT may be managed with close observation and serial imaging

## Timing of surgery

**Level III<sup>327</sup>:** p-fossa mass lesions meeting surgical criteria should be evacuated ASAP due to the potential for rapid deterioration

## Surgical methods

**Level III<sup>327</sup>:** suboccipital craniectomy is the recommended procedure

\* mass effect on CT: defined as dislocation, compression or obliteration of the 4th ventricle; compression or loss of basal cisterns (see page 909), or the presence of obstructive hydrocephalus

# 27.9. Posttraumatic hydrocephalus

Hydrocephalus was found in 40% of 61 patients with severe head injury (GCS = 3-8) and in 27% of 34 patients with moderate head injury (GCS = 9-13)<sup>328</sup>. Hydrocephalus developed by 4 weeks after injury in 58% and by 2 months in 70%<sup>328</sup>. There was no statistically significant relationship between posttraumatic hydrocephalus and age, the presence of SAH, or type of lesion (focal or diffuse). Posttraumatic hydrocephalus was associated with worse outcome<sup>328</sup>.

## Differentiating true hydrocephalus from hydrocephalus ex vacuo

Delayed ventricular enlargement months to years after TBI may instead be due to atrophy (hydrocephalus ex vacuo) secondary to diffuse axonal injury, and may not represent true hydrocephalus. It may not be possible to accurately differentiate these two conditions, and the decision to shunt may therefore be difficult (similar to the dilemma in patients with NPH vs. atrophy).

Factors favoring hydrocephalus, for which shunt should be considered:

1. elevated pressure on 1 or more LPs
2. papilledema on funduscopic exam
3. symptoms of headache/pressure
4. findings of "transependymal absorption" on CT or T2WI MRI (see page 310)
5. ± patient's whose neurologic recovery seems worse than expected
6. provocative tests have been recommended<sup>329</sup> (see page 332)

Patients with enlarged ventricles who are asymptomatic and are doing well following their head injury should be managed expectantly.

## HYDROCEPHALUS AFTER TRAUMATIC SUBARACHNOID HEMORRHAGE

Incidence of clinically symptomatic hydrocephalus within 3 months of traumatic subarachnoid hemorrhage (tSAH) is ≈ 12%<sup>330</sup>. In this series of 301 tSAH patients, multivariate analysis showed the risk of developing hydrocephalus increased with age, intraventricular hemorrhage, blood thickness ≥ 5 mm, and diffuse distribution of blood (vs. focal distribution). There was no correlation with gender, admission GCS score, basal location of tSAH, or use of decompressive craniectomy<sup>330</sup>. NB: this is potentially confusing, univariate analysis shows the risk of hydrocephalus increases with increasing severity of TBI.

## 27.10. Aspects of general care in severe TBI

### 27.10.1. Airway management

See page 861 for indications for, and use of antibiotics with, intubation.

#### PRACTICE GUIDELINE 27-27 TIMING OF TRACHEOSTOMY

**Level II**<sup>52</sup>: early tracheostomy reduces the number of days of mechanical ventilation but does not affect mortality or incidence of pneumonia

#### PRACTICE GUIDELINE 27-28 TIMING OF EXTUBATION

**Level III**<sup>52</sup>: early extubation for patients meeting extubation criteria does not increase the risk of pneumonia

### 27.10.2. Deep-vein thrombosis (DVT) prophylaxis

Also, see page 42 for further details about thromboembolism in neurosurgical patients. The risk of developing DVT is  $\approx 20\%$  in untreated severe TBI<sup>331</sup>. **PRACTICE GUIDELINE 27-29** delineates some aspects of DVT prophylaxis.

#### PRACTICE GUIDELINE 27-29 DVT PROPHYLAXIS IN SEVERE TBI

**Level III**<sup>332</sup>:

- unless contraindicated, graduated compression stockings or intermittent compression boots are recommended until patients are ambulatory
- low molecular weight heparin (**LMWH**) (see page 39) or low-dose unfractionated heparin in conjunction with mechanical measures lowers the DVT risk, but a trend suggests they increase the risk of expansion of intracranial hemorrhage\*

\* there is insufficient evidence: to support use of one pharmacologic agent over another, or to define the optimal dose or timing of agents<sup>332</sup>

### 27.10.3. Nutrition in the head-injured patient

#### SUMMARY OF RECOMMENDATIONS (SEE TEXT FOR DETAILS)

##### PRACTICE GUIDELINE 27-30 NUTRITION

**Level II**<sup>333</sup>: full caloric replacement should be attained by post-trauma day 7

1. by post-trauma day 7, replace the following (enterally or parenterally):
  - A. non-paralyzed patients: 140% of predicted basal energy expenditure (BEE)
  - B. paralyzed patients: 100% of predicted BEE
2. provide  $\approx 15\%$  of calories as protein
3. nutritional replacement should begin within 72 hrs of head injury in order to achieve goal #1 by day 7
4. the enteral route is preferred (IV hyperalimentation is preferred if higher nitrogen intake is desired or if there is decreased gastric emptying)

#### RATIONALE

##### CALORIC REQUIREMENTS

Restored comatose patients with isolated head injury have a metabolic expenditure



that is 140% of normal for that patient (range: 120-250%)<sup>50, 334-336</sup>. Paralysis with muscle blocker or barbiturate coma reduced this excess expenditure in most patients to ~ 100-120% of normal, but some remained elevated by 20-30%<sup>337</sup>. Energy requirements rise during the first 2 weeks after injury, but it is not known for how long this elevation persists. Mortality is reduced in patients who receive full caloric replacement by day 7 after trauma<sup>338</sup>. Since it generally takes 2-3 days to get nutritional replacement up to speed whether the enteral or parenteral route is utilized<sup>50</sup>, it is recommended that nutritional supplementation begin within 72 hrs of head injury.

**Enteral vs. IV hyperalimentation:** Caloric replacement that can be achieved is similar between enteral or parenteral routes<sup>339</sup>. The enteral route is preferred because of reduced risk of hyperglycemia, infection and cost<sup>340</sup>. IV hyperalimentation may be utilized if higher nitrogen intake is desired or if there is decreased gastric emptying. No significant difference in serum albumin, weight loss, nitrogen balance, or final outcome was found between enteral and parenteral nutrition<sup>339</sup>.

Estimates of basal energy expenditure (BEE) can be obtained from the **Harris-Benedict equation**<sup>341</sup>, shown in Eq 27-4 through Eq 27-6, where W is weight in kg, H is height in cm, and A is age in years.

$$\text{Males: BEE} = 66.47 + 13.75 \times W + 5.0 \times H - 6.76 \times A \quad \text{Eq 27-4}$$

$$\text{Females: BEE} = 65.51 + 9.56 \times W + 1.85 \times H - 4.68 \times A \quad \text{Eq 27-5}$$

$$\text{Infants: BEE} = 22.1 + 31.05 \times W + 1.16 \times H \quad \text{Eq 27-6}$$

## Enteral nutrition

Isotonic solutions (such as Isocal® or Osmolyte®) should be used at full strength starting at 30 ml/hr. Check gastric residuals q 4 hrs and hold feedings if residuals exceed ~ 125 ml in an adult. Increase the rate by ~ 15-25 ml/hr every 12-24 hrs as tolerated until the desired rate is achieved<sup>342</sup>. Dilution is not recommended (may slow gastric emptying), but if it is desired, dilute with normal saline to reduce free water intake.

### Cautions:

NG tube feeding may interfere with absorption of phenytoin (see *phenytoin (PHT)* (*Dilantin*®), page 409). Reduced gastric emptying may be seen following head-injury<sup>343</sup> (NB: some may have temporarily elevated emptying) as well as in pentobarbital coma, patients may need IV hyperalimentation until the enteric route is usable. Others have described better tolerance of enteral feedings using jejunal administration<sup>344</sup>.

## NITROGEN BALANCE

As an estimate, for each gram of N excreted (mostly in the urine, however, some is also lost in the feces), 6.25 gm of protein have been catabolized. It is recommended that at least 15% of calories be supplied as protein. The percent of calories consumed (PCC) derived from protein can be calculated from Eq 27-7, where N is nitrogen in grams, and BEE is the basal energy expenditure<sup>334</sup> (see Eq 27-4 through Eq 27-6).

$$\text{PCC (from protein)} = \frac{N \text{ (gm N)} \times \frac{6.25 \text{ gm protein}}{\text{gm N}} \times \frac{4.0 \text{ kcal}}{\text{gm protein}}}{\text{BEE}} \times 100 \quad \text{Eq 27-7}$$

Thus, to supply PCC (protein) = 15% once the BEE is known, use Eq 27-8. Some enteral formulations include Magnacal® (PCC = 14%) and TraumaCal® (PCC = 22%).

$$N \text{ (gm N)} = 0.006 \times \text{BEE} \quad \text{Eq 27-8}$$

## 27.11. Outcome from head trauma

### 27.11.1. Age

In general, the degree of recovery from closed head injury is better in infants and

young children than in adults. In adults, decerebrate posturing or flaccidity with loss of pupillary or oculovestibular reflex is associated with a poor outcome in most cases, these findings are not as ominous in pediatrics.

## 27.11.2. Outcome prognosticators

The frequency of poor outcome from closed head injury is increased with persistent ICP > 20 mm Hg after hyperventilation, increasing age, impaired or absent pupillary light response or eye movement, hypotension (SBP < 90), hypercarbia, hypoxemia, or anemia<sup>79</sup>. This is probably due at least in part to the fact that some of these are markers for significant injury to other body systems. One of the most important predictors for poor outcome is the presence of a mass lesion requiring surgical removal<sup>345</sup>. High ICP during the first 24 hrs is also a poor prognosticator.

### MIDLINE SHIFT (MLS)

The presence of MLS correlates with a worse outcome. For the purpose of standardizing measurements in trauma, MLS is defined at the level of the foramen of Monro<sup>346</sup> as shown in Figure 27-7, and is calculated using Eq 27-9,

$$\text{midline shift (MLS)} = \frac{\text{BPD}}{2} - \text{SP} \quad \text{Eq 27-9}$$

where the midline is found by dividing the biparietal diameter (BPD) (the width of intracranial compartment at this location) by 2, and subtracting SP (the distance from the inner table to the septum pellucidum on the side of the shift). Measurements may be inaccurate if the vertical axis of the patient's head is not parallel to the long axis of the CT scanner. Midline shift may be associated with altered levels of consciousness (see page 280).

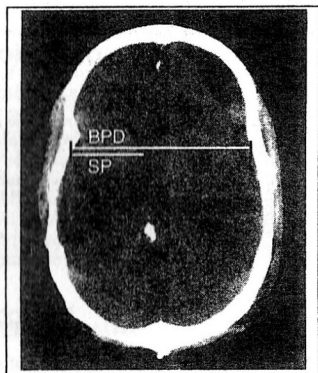


Figure 27-7 Measurement of midline shift (CT of a patient with subdural hematoma)

### OBLITERATION OF BASAL CISTERNS ON CT

The status of the basal cisterns<sup>A</sup> (BCs) is evaluated on axial CT scan at the level of the midbrain (see Figure 27-8) where they are divided into 3 limbs<sup>346</sup> (1 posterior limb = quadrigeminal cistern, 2 lateral limbs = posterior portion of the ambient cisterns). Possible findings:

1. open: all 3 limbs open
2. partially closed: 1 or 2 limbs obliterated
3. completely closed: 3 limbs obliterated

Compression or absence of the BCs carries a threefold risk of increased ICP, and the status of the BCs correlates with outcome<sup>346</sup>.

In a study of 218 patients with GCS ≤ 8, the BCs were classified on initial CT (within 48 hrs of admission) as: absent, compressed, normal, or not visualized (quality of CT too poor to tell)<sup>347</sup>. The relationship of the BCs to outcome is shown in Table 27-28.

18 patients had a shift of brain structures > 15 mm associated with absent BCs, all of them died. The status of the BCs were more important within each GOS score than across scores. Also, see Table 27-7, page 854 for more

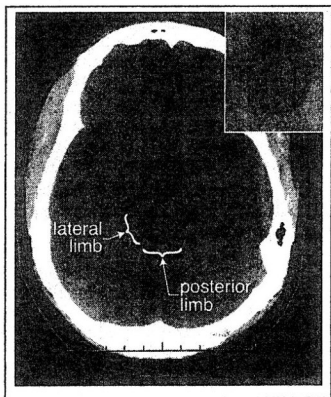


Figure 27-8 Basal cisterns CT demonstrating open basal cisterns (inset: example of = complete obliteration of BCs)

A. "basal cisterns" in the trauma literature are a subset of the perimesencephalic cisterns, see page 1085

**APOLIPOPROTEIN E  
(APOE) E4 ALLELE**

The presence of this genotype portends a worse prognosis following traumatic brain injury<sup>348</sup>. Furthermore, the incidence of severe brain injury in individuals with the apoE-4 allele greatly exceeds the rate of the allele in the general population<sup>349</sup>. This

allele is also a risk factor for Alzheimer's disease (*see below*) as well as for chronic traumatic encephalopathy (*see page 912*).

**Table 27-28 Correlation of GOS\* with basal cisterns**

Basal cisterns	--- OUTCOME* ---				
	Mortality	Vegetative	Severe disability	Moderate disability	Good
	(GOS 1)	(GOS 2)	(GOS 3)	(GOS 4)	(GOS 5)
normal	22%	6%	16%	21%	35%
compressed	39%	7%	18%	17%	19%
absent	77%	2%	6%	4%	11%
not-visualized	68%	0%	11%	9%	12%

\* GOS = Glasgow outcome scale, *see Table 34-3, page 1183*

### 27.11.3. Late complications from traumatic brain injury

Long term complications include:

1. posttraumatic seizures: (*see page 398*)
2. communicating hydrocephalus: incidence  $\approx$  3.9% of severe head injuries
3. posttraumatic syndrome (or postconcussive syndrome): *see below*
4. hypogonadotropic hypogonadism<sup>350</sup>; also *see page 863*
5. chronic traumatic encephalopathy (*see page 911*)
6. Alzheimer's disease (**AD**): head injury (especially if severe) promotes the deposition of amyloid proteins, especially in individuals possessing the apolipoprotein E (apoE)  $\epsilon$ 4 allele<sup>349</sup>, which may be related to the development of AD<sup>351-353</sup>

#### POSTCONCUSSIVE SYNDROME

Variouslly defined collection of symptoms (*see below*) that is usually considered as a possible sequelae to minor head trauma (although some of these features can be seen following more serious head trauma). Loss of consciousness is not a prerequisite.

Controversy exists over the relative contribution of actual organic dysfunction vs. psychological factors (including conversion reaction, secondary gain which may be for attention, financial reward, drug seeking...). Furthermore, the presence of some of these symptoms can undoubtedly lead to the development of others (e.g headache can cause difficulty concentrating and thus poor job performance and thence depression).

A paradox has been noted by clinicians that the complaints following minor head injury seem out of proportion when considered in the context of the frequency of complaints after serious head injury. It has also been noted that patients with early post-traumatic complaints generally improve with time, whereas the late development of symptoms is often associated with a more protracted and fulminant course.

Symptoms commonly considered part of this syndrome include the following (with headache, dizziness and memory difficulties being the most frequent):

1. somatic
  - A. headache
  - B. dizziness or light-headedness
  - C. visual disturbances: blurring is a common complaint
  - D. anosmia
  - E. hearing difficulties: tinnitus, reduced auditory acuity
  - F. balance difficulties
2. cognitive
  - A. difficulty concentrating
  - B. dementia: more common with multiple brain injuries than with a single concussion (*see Chronic traumatic encephalopathy, page 911*)
    1. loss of intellectual ability
    2. memory problems: usually impairs short-term memory
  - C. impaired judgement
3. psychosocial

- A. emotional difficulties: including depression, mood swings (emotional lability), euphoria/giddiness, easy irritability, lack of motivation, abulia
- B. personality changes
- C. loss of libido
- D. disruption of sleep/wake cycles, insomnia
- E. easy fatigability
- F. intolerance to light (photophobia) and/or loud (or even moderate) noise
- G. increased rate of job loss and divorce (may be related to any of above)

Basically any symptom can be ascribed to the condition. Other symptoms that may be described by patients which are generally not included in the definition:

1. fainting (vaso-vagal episodes): may need to rule out posttraumatic seizures, as well as other causes of syncope
2. altered sense of taste
3. dystonia<sup>354</sup>

## Treatment

Treatment for symptoms attributed to this syndrome tends to be supportive. Often times these patients obtain treatment from primary care physicians, neurologists, psychiatrists, and/or psychiatrists/psychologists. Neurosurgical involvement in the continuing care for these patients is usually at the discretion of the individual physician based on his or her practice patterns. Recovery follows a highly variable course.

Some symptoms may need to be evaluated for possible correctable late complications (seizures, hydrocephalus, CSF leak...). Alves and Jane<sup>355</sup> perform a head CT, MRI, BAER and neuropsychological battery if symptoms after minor head injury persist > 3 months. An EEG may be appropriate in cases where there is a question of seizures. If all studies are negative, "the authors tell the patient (and the lawyer) that there is no objective evidence for disease and that psychiatric evaluation is warranted." Non-correctable abnormalities on these studies prompt reassurance that significant symptoms should subside by 1 year, and that no specific treatment, other than psychological counselling, is helpful.

## CHRONIC TRAUMATIC ENCEPHALOPATHY

Often described in retired boxers, chronic traumatic encephalopathy (CTE) encompasses a spectrum of symptoms that range from mild to a severe form AKA **dementia pugilistica**<sup>356</sup>, or punch drunk syndrome. Symptoms involve motor, cognitive and psychiatric systems. CTE is distinct from post-traumatic dementia (which may follow a single closed head injury) or from post-traumatic Alzheimer's syndrome. Although generally accepted, not all authorities agree that repeated concussions have any long-term sequelae<sup>367</sup>.

There are some similarities with Alzheimer's disease (AD), including the presence of neurofibrillary tangles having similar microscopic characteristics and the development of amyloid angiopathy with the attendant risk of intracerebral hemorrhage<sup>368</sup>. EEG changes occur in one-third to one-half of professional boxers (diffuse slowing or low-voltage records).

**Clinical:** Clinical features of CTE are shown in Table 27-29<sup>356</sup> and include:

Table 27-29 CTE of boxing\*

Motor	Cognitive	Psychiatric
Early (~ 57%)		
dysarthria tremors mild incoordination especially non-dominant hand	decreased complex attention	emotional lability euphoria/hypomania irritability, suspiciousness ease of aggression & talkativeness
Middle (~ 17%)		
parkinsonism increased dysarthria, tremors, and incoordination	slowed mental speed mild deficits in memory, attention & executive ability	magnified personality decreased spontaneity paranoid, jealous inappropriate violent outbursts
Late (< 3%)		
pyramidal signs prominent parkinsonism prominent dysarthria, tremors & ataxia	prominent slowness of thought/speech amnesia attention deficits executive dysfunction	cheerful/silly decreased insight paranoid, psychotic disinhibited, violent possible Klüver-Bucy

\* in professional boxers with ≥ 20 bouts

1. cognitive: mental slowing and memory deficits (dementia)
2. personality changes: explosive behavior, morbid jealousy, pathological intoxication with alcohol, and paranoia
3. motor: cerebellar dysfunction, symptoms of Parkinson's disease, pyramidal tract dysfunction

Grading scales have been devised to rank patients as having probable, possible, and improbable CTE.

The chronic brain injury scale (CBIS) assesses involvement of motor, cognitive, and psychological axes as shown in *Table 27-30*.

**Risk factors** for dementia pugilistica in boxing<sup>356</sup>:

1. risk increases with length of boxing career, especially > 10 yrs
2. age at retirement: risk goes up after age 28 yrs
3. number of bouts: especially  $\geq 20$  (more important than the number of knock-outs)
4. boxing style: increased risk among poorer performers, those known as sluggers rather than "scientific" boxers, those known to be hard to knock out or known to take a punch and keep going
5. age at examination: long latency causes increased prevalence with age
6. and possibly, the number of head blows
7. risk increases in patients with the apolipoprotein E (apo E)  $\epsilon 4$  allele (as in Alzheimer's disease) as shown in *Table 27-31*
8. professional boxers (more risk than amateurs)

**Neuro-imaging:** The most common finding is cerebral atrophy. A cavum septum pellucidum (CSP) is observed in 13% of boxers<sup>359</sup>. CSP in this setting probably represents an acquired condition<sup>360</sup> and correlates with cerebral atrophy.

**Neuropathology** includes:

1. cerebral and cerebellar atrophy
2. neurofibrillary degeneration of cortical and subcortical areas
3. deposition of  $\beta$ -amyloid protein
  - A. forming diffuse amyloid plaques
  - B. in a subset of CTE patients this involves the vessel walls giving rise to cerebral amyloid angiopathy

**Table 27-30 Chronic brain injury scale**

Grade involvement of each of the following axes separately:	Scoring for each axis:
• motor	0 = none
• cognitive	1 = mild
• psychological	2 = moderate
	3 = severe
Sum total points	Severity
0	normal
1 - 2	mild
3 - 4	moderate
> 4	severe

**Table 27-31 Odds ratio for developing Alzheimer's disease**

Head injury	Apo E $\epsilon 4$ allele	Odds ratio
-	-	1
-	+	2
+	-	1
+	+	10

## 27.12. Gunshot wounds to the head

Gunshot wounds to the head (GSWH) account for the majority of penetrating brain injuries, and comprise  $\approx 35\%$  of deaths from brain injury in persons < 45 yrs old. GSWH are the most lethal type of head injury,  $\approx$  two thirds die at the scene, and GSWH ultimately are the proximal cause of death in > 90% of victims<sup>361</sup>.

### PRIMARY INJURY

Primary injury from GSWH results from a number of factors including:

1. injury to soft tissue
  - A. direct scalp and/or facial injuries
  - B. soft tissue and bacteria may be dragged intracranially, the devitalized tissue may also then support growth of the bacteria
  - C. pressure waves of gas combustion may cause injury if the weapon is close
2. comminuted fracture of bone: may injure subjacent vascular and/or cortical tissue (depressed skull fracture). May act as secondary missiles
3. cerebral injuries from missile
  - A. direct injury to brain tissue in path of bullet, exacerbated by
    1. fragmentation of bullet

2. ricochet off bone
3. deviations of the bullet from a straight path as it travels: tumbling (forward rotation - pitch), yaw (rotation about vertical axis), rotation (spin), nutation
4. deformation of bullet at impact: e.g. mushrooming
- B. injury to tissue by shock waves, cavitation
4. coup + contrecoup injury from missile impact on head (may cause injuries distant from bullet path)

Because of the complexities of ballistics (some of which are described above) there is often more damage even though the bullet slows (losing kinetic energy).

Extent of primary injury is related to impact velocity:

- impact velocity > 100 m/s: causes explosive intracranial injury that is uniformly fatal (NB: impact velocity is less than muzzle velocity)
- non-bullet missiles (e.g. grenade fragments) are considered low velocity
- low muzzle velocity bullets ( $\approx < 250$  m/s): as with most handguns. Tissue injury is caused primarily by laceration and maceration along a path slightly wider than missile diameter
- high muzzle velocity bullets ( $\approx 600-750$  m/s): from military weapons and hunting rifles. Causes additional damage by shock waves and temporary cavitation (tissue pushed away from the missile causes a conical cavity of injury that may exceed bullet diameter many-fold, and causes low-pressure region which may draw surface debris into the wound)

## SECONDARY INJURY

Cerebral edema occurs similar to closed head injury. ICP may rise rapidly within minutes (higher ICPs result from higher impact velocities). Cardiac output may also fall initially. Together,  $\uparrow$  ICP and  $\downarrow$  MAP adversely effect cerebral perfusion pressure.

Other common complicating factors include: DIC, intracranial hemorrhage from lacerated blood vessels.

## LATE COMPLICATIONS

Late complications include:

1. cerebral abscess: migration of bullet may be a tip-off (*see below*). Usually associated with retained contaminated material (bullet, bone, skin...) but may also result from persistent communication with nasal sinuses
2. traumatic aneurysm<sup>362</sup>
3. seizures
4. fragment migration
  - A. migration of a bullet: often indicates abscess<sup>363</sup> or, less commonly, a hematoma cavity. May also migrate within the ventricles
  - B. intraventricular fragments may migrate and cause obstructive hydrocephalus<sup>364</sup>
5. lead toxicity: more of an issue with bullet in disc space (*see page 998*)

## EVALUATION

Exam should describe visible entrance and exit wounds. In through-and-through missile wounds of the skull, the entrance wound is typically smaller than the exit wound due to bullet mushrooming. Entrance wounds may be especially small with direct contact of the muzzle to the head. At surgery or autopsy, the entrance wound will typically show bevelling of the inner table, whereas exit wounds have a beveled outer table.

## GRADING SYSTEMS

The Glasgow Coma Scale is still the most widely used system and allows better comparison between series than specialized scales for GSWs. See *Outcome* below.

## INITIAL STABILIZATION

### GENERAL MEASURES

1. CPR as required; endotracheal intubation if stuporous or airway compromised
2. additional injuries (e.g. chest wounds) identified and treated appropriately
3. usual precautions taken for spine injury
4. fluids as needed to replace estimated blood loss which may be variable: care to avoid excessive hydration (to minimize cerebral edema)
5. pressors to support MAP during and after fluid resuscitation

### TREATMENT OF THE INJURY

Neurological assessment as rapidly as possible and as thoroughly as time permits.

Decision by experienced neurosurgeon regarding the ultimate treatment of the patient will determine appropriate steps to be taken. Patients with little CNS function (in the absence of shock) are unlikely to benefit from craniotomy. Supportive measures are indicated in most cases (for possibility of organ donation, opportunity for family to adjust to situation, and requirements for observation period to determine actual brain death).

In patients considered for further treatment, rapid deterioration at any point with signs of herniation requires immediate surgical intervention. As time permits, the following should be undertaken:

- initial steps
  - A. control bleeding from scalp and associated wounds (hemostats on scalp vessels)
  - B. shave scalp to identify entrance/exit sites, and to save time in the O.R.
- radiographic evaluation
  - A. AP and lateral skull films to localize metal and bone fragments, and to help identify entrance/exit sites (omit if time not available)
  - B. non-contrast CT scan of the brain: identifies bullet track, intracranial hematomas, intraparenchymal location of bone and metal
  - C. angiography is occasionally indicated (*see below*)
- medical treatment (similar to closed head injury)
  - A. assume ICP is elevated:
    1. elevate HOB 30-45° with head midline (avoids kinking jugular veins)
    2. **mannitol** (1 gm/kg bolus) as blood pressure tolerates
    3. **hyperventilate** to  $\text{PaCO}_2 = 30-35$  mm Hg if indications are met (*see Indications for hyperventilation (HPV)*, page 881)
    4. steroids: (unproven efficacy) 10 mg dexamethasone IVP
  - B. prophylaxis against GI ulcers:  $\text{H}_2$  antagonist (e.g. ranitidine 50 mg IVPB q 8 hrs), NG tube to suction
  - C. begin phenytoin (**PHT**) loading: effective in controlling acute seizures, incidence of late seizures are not reduced once PHT is stopped
  - D. antibiotics: generally used although no controlled study demonstrates efficacy in preventing meningitis or abscess. Most organisms are sensitive to penicillinase resistant agents, e.g. nafticillin, recommended for  $\approx 5$  days
  - E. tetanus toxoid administration

### ANGIOGRAPHY IN GSWH

Rarely performed emergently. When done, usually performed on  $\approx$  day 2-3.

Indications<sup>365</sup>:

1. unexpected delayed hemorrhage
2. a trajectory that would likely involve named vessels in a salvageable patient
3. large intraparenchymal hemorrhages in a salvageable patient

### SURGICAL TREATMENT

Indications for surgery are controversial. Patients with minimal neurologic function, e.g. fixed pupils, decorticate or decerebrate posturing... (when not in shock and with good oxygenation) should not be operated upon, because the chance of meaningful recovery is close to zero. Patients with less severe injuries should be considered for urgent op-

eration.

### Goals of surgery

1. debridement of devitalized tissue: less tissue is injured in civilian GSWH, but elevated ICP post-op may imply more vigorous debridement was needed, especially of non-eloquent brain (e.g. temporal tips)
2. evacuation of hematomas: subdural, intraparenchymal...
3. removal of accessible bone fragments<sup>A</sup>
4. retrieval of bullet fragment<sup>A</sup> for forensic purposes (note: everyone who handles the fragments may be subpoenaed to testify as to the "chain of evidence"). Large intact fragments should be sought as they tend to migrate
5. obtaining hemostasis
6. watertight dural closure (usually requires graft)
7. separation of intracranial compartment from air sinuses traversed by bullet
8. identification of entry and exit wounds for forensic purposes (see *Evaluation* above)

### Surgical technique

Some key points of surgical technique<sup>366 (p 2098-104)</sup>:

- positioning and draping should make both entry and exit wounds accessible
- devitalized tissue around the entry & exit wounds should be excised
- fractured bone should be excised by a circumferential craniectomy (craniotomy may be used in some civilian GSWH, the entry site within the craniotomy should be rongeuired or drilled back to clean bone)
- air sinuses that are traversed should have the mucosa exenterated, and are then packed with muscle, and covered with a graft (e.g. periosteum or fascia lata) to separate them from intracranial compartment
- the dura is opened in a stellate fashion
- pulped brain is removed from within using suction and bipolar in an enlarging cone until healthy tissue is encountered (further injury to deep midline structures should be avoided, here, stay within bullet tract)
- contralateral fragments with no exit wound should only be removed if accessible
- intraventricular fragments can present significant risk. Ventriculoscopy (if available) may be well suited for removing these
- dural closure should be watertight; grafts of pericranium, temporalis fascia, or fascia lata grafts may be used; avoid dura substitutes
- cranioplasty should be delayed 6-12 months to reduce risk of infection
- a post-op CSF fistula that persists > 2 weeks should be repaired

### ICP MONITORING

ICP is often elevated after surgical debridement<sup>367</sup> and monitoring may be warranted.

## OUTCOME

### Prognostic factors

1. level of consciousness is the most important prognostic factor: ~ 94% of patients who are comatose with inappropriate or absent response to noxious stimulus on admission die, and half the survivors are severely disabled<sup>368</sup>
2. path of the bullet. Especially poor prognosis is associated with:
  - A. bullets that cross the midline
  - B. bullets that pass through the geographic center of the brain
  - C. bullets that enter or traverse the ventricles
  - D. the more lobes traversed by the bullet
3. hematomas seen on CT are poor prognostic findings
4. suicide attempts are more likely to be fatal

A. risk of infection and seizures due to retained bullet fragments is not high in civilian GSWH, therefore only accessible fragments should be sought and removed



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## 27.13. Non-missile penetrating trauma

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This section deals with penetrating injuries to the brain (and to some extent to the spinal cord) excluding missile injuries (i.e. gunshot wounds - see page 912). Includes trauma from: knives, arrows, lawn darts... Injury to neural tissue tends to be more limited than with missiles because many of the associated injurious aspects of the missile are absent (see page 912).

### Arrow injuries

As a result of the lower velocity (e.g. 58 m/s) compared to firearms and the sharp tip, injury is usually limited to tissue directly incised by the arrowhead<sup>369</sup>.

### Cases with foreign body still embedded

In penetrating trauma, it is usually not appropriate to remove any protruding part of the foreign body until the patient is in the operating room, unless it cannot be avoided. If possible, it is helpful to have another identical object for comparison in planning extrication of the embedded object<sup>370</sup>. To minimize extending the trauma to the CNS, the protruding object should be stabilized in some way during transportation and evaluation. Intraoperatively, devices such as the Greenberg retractor may be used to stabilize the object during preparation and the initial approach.

### Indications for pre-op angiography

1. object passes in region of large named artery
2. object passes near dural sinuses
3. visible evidence of arterial bleeding: angiography is not appropriate if hemorrhage cannot be controlled

### Surgical techniques

It is impossible to give details to cover every situation. Some guidelines:

1. empiric antibiotic coverage is appropriate (see *Meningitis post craniospinal trauma*, page 343). Take cultures from the wound and the foreign body to guide later antibiotic therapy
2. optimal control can usually be gained by performing a craniotomy up to and if possible around the object, such that removing the bone flap will not disturb the object. The last remnants of bone may then be removed with a rongeur
3. if at all possible open the dura before removing the object, since removal with the dura closed does not allow adequate control of any bleeding from the brain
4. removal of the object ideally should follow the entry trajectory if possible
5. although gunshot wounds are not sterile as once thought, they are probably less contaminated than penetrating wounds. One should debride any easily accessible impacted bone and other extracranial tissue and material along the track

### Post-op care

1. a course of antibiotics are usually appropriate since infection is common
2. consider a post-op arteriogram to rule-out traumatic aneurysm

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## 27.14. High altitude cerebral edema

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Acute high-altitude sickness (AHAS) is a systemic disorder that affects individuals usually within 6-48 hrs after ascent to high altitudes. **Acute mountain sickness (AMS)** is the most common form of AHAS, with symptoms of nausea, headache, anorexia, dyspnea, insomnia and fatigue<sup>371</sup> and is often assessed using the Lake Louise system<sup>372</sup>. The incidence is ~ 25% at 7,000 feet, and ~ 50% at 15,000 feet. Other symptoms of AHAS include edema of feet and hands, and pulmonary edema (HAPE = high altitude pulmonary edema). Ocular findings include retinal hemorrhages<sup>373</sup>, nerve fiber layer infarction, papilledema and vitreous hemorrhage<sup>374</sup>. Cerebral edema (HACE = high altitude cerebral edema), usually associated with pulmonary edema, may occur in severe cases of AHAS. Symptoms of HACE include: severe headache, mental dysfunction (hallucinations, inappropriate behavior, reduced mental status), and neurologic abnormalities (ataxia, paralysis, cerebellar findings).

The unproven "tight-fit" hypothesis postulated that individuals with less compliant CSF systems (smaller ventricles and CSF spaces) were more vulnerable to AMS<sup>375</sup>. A small study of 10 volunteers<sup>376</sup> analyzing CT scans before ascent and symptoms showed a trend that supports the hypothesis.

Prevention: gradual ascent, 2-4 day acclimatization at intermediate altitudes (especially to include sleeping at these levels), avoidance of alcohol or hypnotics.

Treatment of cerebral edema: immediate descent and oxygen (6-12 L/min by NC or face-mask) are recommended. Dexamethasone 8 mg PO or IV followed by 4 mg q 6 hrs may help temporize.

## 27.15. Pediatric head injury

75% of children hospitalized for trauma have a head injury. Although most pediatric head injuries are mild and involve only evaluation or brief hospital stays, CNS injuries are the most common cause of pediatric traumatic death<sup>377</sup>. The overall mortality for all pediatric head injuries requiring hospitalization has been reported between 10-13%<sup>378</sup>, whereas the mortality associated with severe pediatric head injury presenting with decerebrate posturing has been reported as high as 71%<sup>379</sup>.

Differences between adult and pediatric head injury:

1. epidemiology:
  - A. children often have milder injuries than adults
  - B. lower chance of a surgical lesion in a comatose child<sup>380</sup>
2. types of injury: injuries peculiar to pediatrics
  - A. birth injuries: skull fractures, cephalhematoma (*see below*), subdural or epidural hematomas, brachial plexus injuries (*see page 802*)
  - B. perambulator/walker injuries
  - C. child abuse (*see below*): shaken baby syndrome...
  - D. injuries from skateboarding, scooters...
  - E. lawn darts
  - F. cephalhematoma: *see below*
  - G. leptomeningeal cysts, AKA "growing skull fractures": *see page 892*
3. response to injury
  - A. responses to head injury of older adolescent are very similar to adults
  - B. "malignant cerebral edema": acute onset of severe cerebral swelling (probably due to hyperemia<sup>17,381</sup>) following some head injuries, especially in young children (may not be as common as previously thought<sup>382</sup>)
  - C. posttraumatic seizures: more likely to occur within the 1st 24 hrs in children than in adults<sup>383</sup> (*see page 398*)

### Imaging studies

#### PRACTICE GUIDELINE 27-31 IMAGING IN MINOR PEDIATRIC HEAD INJURY\*

**Recommendations**<sup>†384</sup>: CT scan for children with neurologic or cognitive dysfunction, or suspicion of a depressed or basilar skull fracture

**Recommendations**<sup>† 384</sup>: when a CT scan is not done in a child  $\leq 1$  year age meeting the above criteria (e.g. because of sedation concerns), a skull film may be considered

\* Definitions: minor head injury: GCS  $\geq 13$ ; pediatrics = ages 1 month - 17 years of age. Excludes: suspicion or proof of child abuse, patients requiring hospitalization for other reasons

† based mostly prospective trials (not randomized) or large case series

### Home observation

#### PRACTICE GUIDELINE 27-32

##### HOME OBSERVATION IN MINOR PEDIATRIC HEAD INJURY\*

**Recommendations**<sup>†384</sup>: a child with GCS = 14-15 and normal CT scan<sup>‡</sup> can be considered for home observation if neurologically stable

\* Definitions: same as in PRACTICE GUIDELINE 27-31

† mostly prospective trials (not randomized) or large case series

‡ these patients are at near zero risk of having an occult brain injury

~ 22% of those with a history of loss of consciousness (LOC) > 5 mins have a brain injury, whereas 92% without LOC > 5 mins will have no brain injury<sup>384</sup>.

## Outcome

As a group, children fare better than adults with head injury<sup>385</sup>. However, very young children do not do as well as the school-age child<sup>386</sup>.

All aspects of neuropsychological dysfunction following head injury may not always be related to the trauma, as children who get injured may have pre-existing problems that increase their propensity to get hurt<sup>387</sup> (this is controversial<sup>388</sup>).

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### 27.15.1. Cephalhematoma

Accumulation of blood under the scalp. Occur almost exclusively in children.

Two types:

1. **subgaleal hematoma**: may occur without bony trauma, or may be associated with linear nondisplaced skull fracture (especially in age < 1 yr). Bleeding into loose connective tissue separates galea from periosteum. May cross sutures. Usually starts as a small localized hematoma, and may become huge (with significant loss of circulating blood volume in age < 1 year, transfusion may be necessary). Inexperienced clinicians may suspect CSF collection under the scalp which does not occur. Usually presents as a soft, fluctuant mass. These do not calcify
2. **subperiosteal hematoma** (some refer to *this* as cephalhematoma): most commonly seen in the newborn (associated with parturition, may also be associated with neonatal scalp monitor<sup>389, 390</sup>). Bleeding elevates periosteum, extent is limited by sutures. Firmer and less ballotable than subgaleal hematoma<sup>391 (p 312)</sup>; scalp moves freely over the mass. 80% reabsorb, usually within 2-3 weeks. Occasionally may calcify

Infants may develop jaundice (hyperbilirubinemia) as blood is resorbed, occasionally as late as 10 days after onset.

## Treatment

Treatment beyond analgesics is almost never required, and most usually resolve within 2-4 weeks. Avoid the temptation of percutaneously aspirating these as the risk of infection exceeds the risk of following them expectantly, and in the newborn removal of the blood may make them anemic. Follow serial hemoglobin and hematocrit in large lesions. If a subperiosteal hematoma persists > 6 weeks, obtain a skull film. If the lesion is calcified, surgical removal may be indicated for cosmetic reasons (although with most of these the skull will return to normal contour in 3-6 months<sup>391 (p 315)</sup>).

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### 27.15.2. Child abuse

At least 10% of children < 10 yrs age that are brought to E/R with alleged accidents are victims of child abuse<sup>392</sup>. The incidence of accidental head trauma of significant consequence below age 3 is low, whereas this is the age group in which battering is highest<sup>393</sup>.

There are no findings that are pathognomonic for child abuse. Factors which raise the index of suspicion include:

1. retinal hemorrhage (*see below*)
2. bilateral chronic subdural hematomas in a child < 2 yrs age (*see page 905*)
3. skull fractures that are multiple or associated with intracranial injury
4. significant neurological injury with minimal signs of external trauma

## SHAKEN BABY SYNDROME

Vigorous shaking of a child produces violent whiplash-like angular acceleration-decelerations of the head (the infant head is relatively large in proportion to the body, and the neck muscles are comparatively weak)<sup>394</sup> which may lead to significant brain injury. Some researchers believe that impact is often also involved<sup>273</sup>.

Characteristic findings include retinal hemorrhages (*see below*), subdural hematomas (bilateral in 80%) and/or subarachnoid hemorrhage (SAH). There are usually few or no external signs of trauma (including cases with impact, although findings may be ap-

parent at autopsy). In some cases there may be finger marks on the chest, multiple rib fractures and/or pulmonary compression  $\pm$  parenchymal lung hemorrhage. Deaths in these cases are almost all due to uncontrollable intracranial hypertension. There may also be injury to the cervicomedullary junction<sup>395</sup>.

## RETINAL HEMORRHAGE (RH) IN CHILD ABUSE

"In a traumatized child with multiple injuries and an inconsistent history, the presence of RH is pathognomonic of battering"<sup>393</sup>. However, RH may also occur in the absence of any evidence of child abuse. 16/26 battered children < 3 yrs age had RH on funduscopy, whereas 1/32 non-battered traumatized children with head injury had RH (the single false positive: traumatic parturition, where the incidence of RH is 15-30%).

Differential diagnosis of etiologies of retinal hemorrhage:

1. child abuse (including "shaken baby syndrome", see above)
2. benign subdural effusion in infants (see page 904)
3. acute high altitude sickness (see *High altitude cerebral edema*, page 916)
4. acute increase in ICP: e.g. with a severe seizure (may be similar to Purtschers retinopathy - see below)
5. Purtschers retinopathy<sup>396</sup>: loss of vision following major trauma (chest crush injuries, airbag deployment<sup>397</sup>...), pancreatitis, childbirth or renal failure, among others. Posterior pole ischemia with cotton-wool exudates and hemorrhages around the optic disc due to microemboli of possibly fat, air, fibrin clots, complement-mediated aggregates or platelet clumps. No known treatment

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## NOTES