B.1. Diagnosis and Management of Head Trauma

a. Understand and Assign the Glasgow Coma Scale.

The Glasgow Coma Scale was developed in order to standardize the neurologic assessment of patients with head injury. It was specifically designed to be easily performed based upon clinical data, and to have a low rate of interobserver variability. In addition, the Glasgow Coma Scale score is correlated with outcome in that patients with a higher Glasgow Coma Scale score have a statistically better outcome than patients with a lower Glasgow Coma Scale score.

The Glasgow Coma Scale score is determined by adding the values for eye opening, verbal response, and motor response. Possible values range from 3 to 15. Note that this scale rates the best response only. In patients who are intubated, in whom assessment of best verbal response cannot be performed, notation of this is made in the Glasgow Coma Scale score by adding a "t" to the end of the score. In patients who are intubated, the best possible score would therefore be 11t. Certain numerical values of the Glasgow Coma Scale have particular clinical significance. Patients with a Glasgow Coma Scale of 7 or less are considered to be comatose. Patients with a Glasgow Coma Scale score of 8 or less are considered to have suffered a severe head injury.

b. Recognize the Presentation of Brain Herniation Syndromes in the Setting of Trauma:

Distortion of the midline brain structures secondary to brain trauma may lead to specific combinations of signs and symptoms which are collectively referred to as herniation syndromes. In general, these symptoms result from the distortion of midline brain structures secondary to brain swelling, hydrocephalus, or intracranial mass lesions. While numerous herniation symptoms have been described, the two most commonly seen syndromes in the setting of trauma are uncal herniation and tonsillar herniation.

i. Uncal herniation:

Most often results from a laterally placed mass displacing the brain stem contralaterally and pushing the uncus of the temporal lobe medially over the tentorial edge.

Early Sign:

- Ipsilateral pupillary dilation

Late Signs:

- Complete ipsilateral third nerve palsy
- Loss of consciousness
- Contralateral hemiplegia (secondary to mass)
- Ipsilateral hemiplegia (secondary to compression of contralateral cerebral peduncle against edge of tentorium [Kernohan’s notch])
- Flaccid paralysis

b. Tonsillar herniation:

Results from downward displacement of the cerebellar tonsils through the foramen magnum, causing compression of the cervicomedullary junction. Frequently

secondary to posterior fossa mass. May be precipitated by lumbar puncture in the presence of such a mass.

Signs:

- Head tilt/neck pain
- Respiratory arrest
- Loss of consciousness
- Flaccid paralysis

d. Initiate Management of Elevated Intracranial Pressure in Head Trauma:

Brain injury after acute head trauma can be divided into two categories. The first category is primary injury, which is suffered at the time of impact. The second category is secondary injury, which may occur at any time from that point forward. One of the most important causes of secondary brain injury in head trauma is felt to be elevated intracranial pressure (ICP).

In treatment of the patient with head trauma, the possibility of elevated intracranial pressure should always be considered. Management of the traumatized patient begins with the primary survey and resuscitation.

Steps involved include:

- Airway patency with cervical spine control: It is important to establish the presence of a patent airway. If such an airway is not present, one should be established. This may include the use of chin lift or jaw thrust, clearance of foreign bodies, endotracheal intubation, or creation of a surgical airway. It is important to consider that the cervical spine may be injured and that it should be maintained in a neutral position during any of the above maneuvers.
- Breathing control. The chest should be examined and the rate and depth respirations determined. Inadequacy may indicate the need for mechanical ventilation. High concentrations of oxygen should be administered. Pneumothorax should be treated.
- Circulatory and hemorrhage control. The quality, rate, and regularity of the pulse should be determined. Sights of major hemorrhage should be identified and treated.
- Disability. A brief neurologic examination should be performed. The Glasgow Coma Scale should be determined. Pupils should be assessed for size, equality, and reaction.

During the secondary survey, a more complete neurologic examination should be performed including evaluation of the patient’s strength, sensation, reflexes, and remaining cranial nerves.

Following the secondary survey, appropriate imaging studies should be obtained. In a patient with obvious craniofacial trauma, mechanism of injury sufficient to produce brain injury, or a disturbed level of consciousness, a CT scan of the head without contrast should be performed. The presence of fractures, foreign bodies, space occupying lesions, or hemorrhage should be noted, as well as the ventricular size.

Intracranial pressure monitoring should be considered in the following situations:

Patients with an abnormal admission CT scan and Glasgow Coma Scale score of 3 to 8 after cardiopulmonary resuscitation.

Or

Patients with a normal head CT with a Glasgow Coma Scale score of 3 to 8 and the presence of two or more of the following features: Age over 40 years, unilateral or bilateral motor posturing, systolic blood pressure less than 90 mmHg.

The current preferred modality for monitoring of intracranial pressure is the placement of a ventricular catheter (ventriculostomy). Use of fiberoptic or strain gauge pressure monitors can be considered.

Elevated intracranial pressure has been shown to have definite prognostic implications in a patient with severe brain injury. In addition, it is generally held that treatment of elevated intracranial pressure may improve outcome in the patient with severe brain injury. The currently recommended threshold for treatment of elevated intracranial pressure is 20 to 25 mmHg. Interpretation and treatment of intracranial pressure based on this threshold value should be corroborated by frequent clinical examination and assessment of the cerebral perfusion pressure. Current recommendations suggest that CPP should be a minimum of 60 to 70 mm of mercury. It is important to consider that, while MAP is an important determining factor in the CPP, it has also been shown that low MAP is an independent predictor of poor outcome.

After elevated intracranial pressure has been identified, treatment should be initiated. In general, treatment should proceed in a stepwise fashion, beginning with the least onerous treatment modalities. Escalation of treatment should proceed only after failure of less onerous modalities. The suggested hierarchy of treatment includes the following therapeutic modalities:

- Body positioning. The head should be elevated 30 degrees. The neck should be maintained in a neutral position. Compression of the jugular veins should be avoided.
- Maintenance of homeostasis. Euvolemia should be established. Arterial blood gases should be measured with the goal of maintaining the PO2 in the 90 to 100-mm mercury range and the PCO2 in the 35 to 40-mm mercury range.
- Mild sedation. This is most frequently carried out using a combination of benzodiazepines and/or narcotics.
- External ventricular drainage. At this stage, placement of a ventriculostomy, if not done previously for ICP measurement, should be considered. The reservoir is generally placed 5 to 10 cm above ear level or, alternatively, placed at ear level and opened at regular intervals.
- Use of osmotic diuretics. Mannitol is the most commonly used osmotic diuretic. It is most frequently given in a dose of 0.25 to 1.0 grams/kg I.V. over 15 minutes. If the effect of treatment with Mannitol is transient, the dose may be repeated, so long as the serum osmolality remains less than or equal to 320 mOsm/L and the patient remains euvolemic.
- Moderate hyperventilation. At this stage, moderate hyperventilation to a PCO2 of 30 to 35 mm of mercury can be considered.

 Second tier therapies including barbiturate therapy. At this stage, barbiturate therapy can be considered. Pentobarbital is the most commonly used barbiturate for the treatment of refractory intracranial hypertension. Recommended loading dose is 10 mg/kg over 30 minutes followed by a maintenance dose of 1 mg/kg per hour as a continuous infusion. The dose is then titrated to achieve serum Pentobarbital levels in the range of 3 to 4 mg/dl or an electroencephalographic pattern of burst suppression. Potential complications of this modality of treatment are numerous, with hypotension being the primarily dose-limiting toxicity. Barbiturate treatment of refractory intracranial hypertension has been shown to decrease mortality but has not been shown to improve neurologic outcome.

The above treatment algorithm assumes that all significant cranial space-occupying lesions have been appropriately surgically treated. During implementation of the above algorithm, it is important to consider the possibility that a new intracranial lesion has developed. Because of this, a repeat CT scan of the head should be considered prior to escalation of therapy.

Note that the above treatment algorithm does not include the use of corticosteroids. The use of glucocorticoids is not recommended for improving outcome or reducing intracranial pressure in patients with severe head injury. The routine use of prophylactic anticonvulsant medication is not recommended for the prevention of posttraumatic seizures in the patient without a premorbid seizure disorder. The use of anticonvulsants may be considered, in the first 7 days after injury, to prevent early posttraumatic seizures in patients who are at high risk. Phenytoin and carbamazepine are the most commonly used agents. Finally, nutritional support of brain injured patients should be instituted within 7 days of injury.

e. Recognize and Initiate Management of Concussion Brain Contusion and Diffuse Axonal Injury.

. Concussion.

Cerebral concussion is a diffuse brain injury thought to be caused by acceleration-deceleration injury to the brain. Cerebral concussion is a spectrum of injuries, ranging from mild to severe.

 Mild concussion is defined as no loss of consciousness with transient neurologic disturbance.

 Moderate concussion is defined as loss of consciousness with complete recovery occurring in less than 5 minutes.

 Severe cerebral concussion is defined as unconsciousness lasting greater than 5 minutes.

Evaluation and treatment of patients with cerebral concussion remains controversial. Workup includes complete history and physical examination, with neurological examination. Other tests include cervical spine x-rays and other radiographs as indicated, blood alcohol level and urine drug screen, and CT scan of the head in all patients except those who are completely asymptomatic and neurologically normal.

Treatment of patients with cerebral concussion who have a Glasgow Coma Scale score of 14 or 15 is usually expectant. Most patients should undergo hospital admission with frequent neurological examinations. These include all patients with

an abnormal CT scan, history of loss of consciousness, decreased or decreasing level of consciousness, severe headache, under the influence of alcohol or drugs, have physical examination evidence of CSF rhinorrhea or otorrhea, significant associated injuries, no reliable companion at home, unable to return promptly, or are amnestic for the injury. Only those patients who do not manifest any of the prior criteria should be considered for discharge from the hospital. If hospital discharge is considered, any of the previously listed signs or symptoms should prompt a return to the hospital. A written head injury "warning sheet" should be issued. Neurosurgical follow-up should be scheduled.

In addition to frequent neurologic examinations, patients admitted after cerebral concussion may be treated with Tylenol or very mild doses of narcotic pain medication for headache. Nausea and vomiting, which are frequently present after mild or severe concussion, should be treated with non-phenothiazine antiemetic medication. Discharge may be considered when the patient is neurologically normal, nausea and vomiting has ceased, and headache has ceased or is adequately controlled.

All patients who have suffered a cerebral concussion should be counseled regarding the possible occurrence of "post concussive syndrome". Prominent symptoms include headache, mild impairment of memory, dysequilibrium, and alteration of mood. These symptoms usually regress spontaneously but may persist for weeks to months.

i. Diffuse axonal injury:

Diffuse axonal injury is the most severe form of diffuse brain injury. It is felt to be the most common cause of prolonged posttraumatic coma that is not due to mass lesion or ischemia. Diffuse axonal injury is characterized by focal hemorrhagic lesions involving the corpus callosum, rostral mid brain, superior cerebellar peduncles combined with microscopic evidence of widespread axonal damage. Patients with diffuse axonal injury frequently manifest decorticate or decerebrate posture and autonomic dysfunction in addition to their prolonged coma. Elevated intracranial pressure is frequently absent. Care is primarily supportive. In patients with prolonged coma, the prognosis is generally poor, with a 50% mortality and with an approximately 25% incidence of favorable outcome.

ii. Cerebral Contusion.

A cerebral contusion is a focal brain injury caused primarily by impact of the brain surface and the bony ridges of the calvarium. Cerebral contusions are frequently found in the region of the frontal poles, anterior skull base, adjacent the sphenoid ridge, and at the temporal poles. Other locations include the cerebellar hemispheres and the occipital poles. A characteristic pattern of cerebral contusion called the "coup and contrecoup" injury is frequently seen. The coup contusion occurs at the sight of impact and the contrecoup contusion occurs in the brain at the point diametrically opposite the point of impact.

Treatment of cerebral contusion is guided by the neurologic examination. The patient should be admitted to the hospital for observation and frequent neurologic examinations. Intracranial pressure monitoring and treatment should be instituted for the comatose patient. While surgical treatment, consisting of debridement of

the contused brain tissue, is not routinely recommended, it can be considered in patients with refractory intracranial hypertension.

f. Recognize and Initiate Management of Acute Subdural and Epidural Hematoma, Including Surgical Indications.

. Subdural hematoma.

In the acute traumatic subdural hematoma, blood collects between the dura mater and surface of the brain. Most commonly, the bleeding results from tearing of bridging veins located over the convexity of the brain surface. Bleeding originating from a small cortical artery represents the second most common source. Associated intracranial lesions, particularly cerebral contusions, are found in at least 50% of patients with acute traumatic subdural hematoma.

Patients presenting with acute traumatic subdural hematoma may range from normal to deeply comatose. Unlike the epidural hematoma, to be described later, the most common presentation of the patient with an acute traumatic subdural hematoma is that of a patient rendered unconscious at the time of injury without regaining consciousness prior to presentation.

Neurologic findings may be secondary to mass effect, elevated intracranial pressure, and/or associated brain injuries. Elevated intracranial pressure is a common finding and evaluation and treatment of elevated intracranial pressure, as outlined above, should be instituted immediately. The acute traumatic subdural hematoma is most commonly treated surgically. Observation should only be considered in those patients with small (less than 10 mm thick) subdural hematomas who are neurologically intact. Because the acute traumatic subdural hematoma consists of solid blood clot, burr hole drainage is inadequate for relief of mass effect. A craniotomy should be performed and all easily assessable blood clots should be removed. Postoperatively, patients frequently manifest intracranial hypertension, and this should be treated as outlined above.

Outcome after treatment of acute traumatic subdural hematoma is related to a number of factors, particularly preoperative neurologic status. Mortality ranges from greater than 75% in those patients who present with a GCS of 3 to 5 to minimal in patients who present with GCS of 12 to 15. Time from injury to surgical decompression, elevated intracranial pressure, and associated brain lesions also have a detrimental effect on outcome.

i. Epidural hematoma.

In the acute epidural hematoma, blood collects between the inner surface of the calvarium and the dura mater. Most commonly, the acute epidural hematoma results from fracture of the skull, stripping the dura mater from the inner table of the skull, and causing laceration of meningeal vessels or dural sinuses. Bleeding from the middle meningeal artery is responsible for many supratentorial epidural hematomas. In contrast to the subdural hematoma, the patient harboring an acute epidural hematoma may have an initial loss of consciousness, followed by a brief "lucid" interval, followed by progressive neurologic decline. This presentation is seen in approximately 1/3 of patients having an acute epidural hematoma.

Treatment of acute epidural hematoma is generally surgical. Patients with small epidural hematomas not traversing a meningeal artery vein or major sinus, who

present greater than 6 hours after injury may be considered for a nonoperative therapy. However, admission with frequent neurologic examination and a low threshold for repeat CT scanning is mandatory. For all other patients, operative treatment is recommended. As with the acute subdural hematoma, the acute epidural hematoma is comprised of a solid blood clot. Therefore, burr hole drainage is inadequate for removing the intracranial mass. A craniotomy should be performed and the entire blood clot evacuated. After evacuation, the dura mater should be opened to inspect the subdural space. Frequently, a subdural hematoma may be encountered which was not obvious on the preoperative CT scan. Postoperatively, the patient is monitored for signs of elevated intracranial pressure. Elevated intracranial pressure, if detected, is treated as outlined above.

As in the acute traumatic subdural hematoma, outcome after treatment of acute epidural hematoma is related to a variety of factors, the most important being preoperative neurologic status. Mortality ranges from less than 15% in those patients who present with GCS of less than 8 to very low in patients who present with GCS 8 to 15.

g. Recognize and Initiate Management of Penetrating Trauma Including Gunshot Wounds.

Penetrating injuries include all injuries where the scalp and skull are violated by foreign objects including knives, sticks, pencils, arrows, and bullets. Low velocity injuries, such as those produced by a knife blade, produce brain injury along the tract of the knife. High velocity injuries, such as those produced by a bullet, produce both local injury along the tract of the bullet, as well as remote injury in the cavity produced in the wake of the bullet.

The size of the cavity produced by a bullet is related to its kinetic energy, as well as its shape. The kinetic energy is proportional to the mass of the bullet as well as the square of its velocity. The shape of the bullet not only affects the velocity, but also its ability to transfer its kinetic energy to the brain tissue. Bullets that tumble or deform on impact transfer a greater proportion of their kinetic energy to brain tissue and thus produce a more severe injury.

After resuscitation and primary survey, an evaluation of the patient with a penetrating injury includes a thorough physical examination and neurological examination. The location of entry and exit wounds should be well documented. Extent of tissue loss should likewise be documented. All patients who have had penetrating brain injury should undergo a CT scan of the head. Coronal CT scan of the head should be considered in patients with involvement of the anterior skull base. Cerebral angiography should be considered in patients where vascular injury is suspected. Cerebral angiography should be given particular consideration where the tract of the injury passes close to a major vascular structure or in patients with a significant subarachnoid hemorrhage or delayed hematoma.

As in patients with severe non-penetrating brain injury, elevated intracranial pressure should be suspected. The algorithm for this is described above. In addition, in patients with penetrating brain injury, intracranial pressure monitoring is indicated when it is not possible to assess the neurological examination accurately or when the need to evacuate a mass lesion is unclear.

As with the open depressed skull fracture, most penetrating brain injuries require surgical treatment.

- Entrance and exit wounds. Treatment of small bullet entrance wounds where the scalp is not devitalized and the patient has no significant intracranial pathology may be treated with local wound care and closure. More extensive wounds with nonviable scalp, bone, or dura may require operative debridement before primary closure and dural grafting. Watertight dural closure is recommended. This includes patients with significant fragmentation of the skull.
- Intraparenchymal lesions. Intraparenchymal lesions resulting in significant mass effect may require debridement of necrotic brain tissue and removal of easily accessible bone fragments. This includes patients with significant intracranial hematomas.
- Injuries resulting in communication between an open-air sinus and the intracranial space should be closed in a watertight fashion.

Routine removal of bone fragments or missile fragments remote from the entry site is not recommended. Vascular injuries detected on arteriography may require surgical or endovascular treatment as indicated. Cerebrospinal fluid leaks which do not close spontaneously or which do not resolve after the primary surgery should be treated with temporary CSF diversion. Those which do not respond to temporary CSF diversion may require surgical treatment. Patients with penetrating brain injury should be administered broad-spectrum antibiotics prophylactically. The duration of this treatment is somewhat controversial. Finally, the use of anti-seizure prophylaxis in the first week of penetrating brain injury is recommended to prevent early posttraumatic seizures. As with severe non-penetrating brain injury, long-term prophylactic treatment with anticonvulsants to prevent late posttraumatic seizures is not recommended.

h. Recognize and Understand the Principles of Management of Open, Closed, and Basilar Skull Fractures Including Cerebrospinal Fluid Leak in Chronic Subdural Hematoma in Children and Adults.

. Open skull fractures.

The treatment of skull fractures with overlying laceration is primarily based on whether the fracture is depressed or non-depressed. Open non-depressed fractures may be treated with inspection, cleansing, and scalp suturing with an acceptably low rate of infection. Open depressed skull fractures present a significant risk of infection. They are generally treated with operative irrigation, debridement, and removal of the depressed fragments. Frequently, this will necessitate further surgical procedures to correct the resulting cosmetic deformity.

i. Closed fractures.

Closed skull fractures require no specific treatment. As noted above, the patient harboring a skull fracture is at increased risk of formation of epidural hematoma. Patients harboring this injury should be treated as for those patients with severe concussion, unless symptoms of elevated intracranial pressure develop. Traditionally, closed depressed skull fractures were treated surgically to elevate the depressed fragments. It has since been shown that this practice does not result in a decreased incidence of posttraumatic epilepsy and has therefore been abandoned.

Treatment of patients with depressed, closed fractures, except where there is a significant cosmetic deformity, should be treated as outlined above for closed, non-depressed fractures.

ii. Basilar skull fractures.

A basilar skull fracture involves the cranial base. The most common sites are the floor of the anterior cranial fossa and the temporal bone. Evidence that a patient may have suffered a basilar skull fracture includes "raccoon eyes" and "battle sign" (bruising posterior to the ear, possibly including the mastoid process). Most basilar skull fractures do not require treatment in the absence of associated brain injury. They are a sign that a significant blow has been delivered to the cranium. Most patients with significant basilar skull fracture should be observed with frequent neurological examinations for 24 hours after the injury. Occasionally, complications of basilar skull fracture may result. These include posttraumatic cerebrospinal fluid leakage, optic nerve injury, and facial nerve injury. The incidence of posttraumatic cerebrospinal fluid leak after a closed head injury is approximately 2%. The most common sites of cerebrospinal fluid egress are the nose (CSF rhinorrhea) and ear (CSF otorrhea). Most traumatic CSF leaks resolve with nonoperative treatment including head elevation. It is important to insure that the CSF leakage does resolve, as persistent CSF rhinorrhea carries with it an approximately 25% risk of meningitis, as well as a risk of tension pneumocephalus.

iii. Posttraumatic CSF leak.

Treatment of the posttraumatic CSF leak which does not resolve with head elevation includes repeated high volume lumbar punctures, as well as continuous catheter CSF drainage. There is a finite risk of inducing tension pneumocephalus using those modalities. For leaks which persist despite these measures, surgical treatment should be considered.

iv. Chronic subdural hematoma.

As described above, the chronic subdural hematoma is a collection of blood and blood products between the inner surface of the dura mater and the outer surface of the brain. Unlike the acute traumatic subdural hematoma, onset of symptoms and detection of the subdural hematoma occurs much later in the course. The hallmark of the chronic subdural hematoma is blood products, visualized on the CT scan, which are isodense or hypointense with respect to brain tissue. It is theorized that, like the acute traumatic subdural hematoma, the source of the blood products is lacerated bridging veins resulting from acceleration-deceleration forces applied to the skull. While this is usually the result of trauma, trauma may be mild, remote, and not remembered by the patient or family. Many patients who present with a chromic subdural hematoma have one or more risk factors. These risk factors include:

- Advanced age and cerebral atrophy.
- Male sex.
- Coagulopathy.
- Intracranial hypotension secondary to CSF shunting procedures
- Chronic alcoholism.

Chronic subdural hematomas tend to enlarge slowly over time. The most common mechanism of enlargement appears to be multiple episodes of re-bleeding, however, other mechanisms may also be important.

Symptoms at presentation include symptoms of increased intracranial pressure (headache, papilledema, decreased level of consciousness), as well as those of hemispheric mass effect (hemiparesis, dysphasia, tremors, and dystonia. Seizures occur occasionally.

Treatment of chronic subdural hematoma is generally surgical. While treatment with prolonged bedrest, head elevation, and osmotic diuretics has produced acceptable results, the risks of prolonged immobilization in these generally debilitated patients frequently outweigh the benefits.

There are multiple surgical options for the treatment of chronic subdural hematoma including:

- Twist drill hole and drainage.
- Multiple burr holes and drainage.
- Craniotomy and drainage with stripping of membranes.

Hematomas that are not fully liquefied may require craniotomy. In addition, recurrence rate after evacuation of chronic subdural hematoma is substantial.

Postoperatively, elevated intracranial pressure is unusual. Patients are generally maintained supine with minimal elevation of the head for at least 24 hours to allow some re-expansion of the brain. Because of the substantial rate of recurrence, neurosurgical follow-up is essential.

v. Chronic subdural hematoma in children.

Like the adult chronic subdural hematoma, blood and blood products, in the childhood chronic subdural hematoma, accumulate between the inner surface of the dura and the brain surface. In children, it is particularly important, and sometimes difficult, to distinguish between the chronic subdural hematoma and the subdural hygroma, in which cerebrospinal fluid collects between the dura and arachnoid membrane. Furthermore, low-density fluid may collect between the arachnoid and pia mater secondary to communicating hydrocephalus.

While trauma is the most common cause of chronic subdural hematoma in children, the possibility of non-accidental trauma (child abuse) must always be considered, particularly in children less than 2 years of age. Coagulopathy remains a major risk factor.

In children, presenting symptoms are usually those of elevated intracranial pressure including vomiting, lethargy, irritability, or increase in the head size. Seizures are also seen.

The treatment of chronic subdural hematoma in children is different than that of adults. Unlike adult patients, very young patients may have an open fontanel, facilitating percutaneous aspiration of the subdural hematoma. Failure of this treatment modality generally requires placement of a subdural to peritoneal shunt.