

# Nursing care plan

[Health & Medicine](#), [Nursing](#)



## ASSOCIATE DEGREE NURSING NURSING PROCESS FORM: PART I –

ASSESSMENT Student: Date of Care: 3/4/13 Client's Initial: WB Room # 1011

Occupation: Teacher Age: 59 Sex: F Race: Black Religion: Christian Admission

Date: 3/1/13 Primary Language: English Role in family: Widowed from

husband Stage in Life Cycle: Generativity vs. Stagnation Surgery date(s) this

admission: N/A Chief complaint: Brain Dysfunction/Traumatic, closed injury

Admission Diagnosis: Post Blunt Force (Head) Trauma Additional Diagnosis:

Type 2 DM; HTN; DM related Neuropathy Past Medical/Surgical History

(dates) 11/29/10 Dx w/ Pancreatitis & Hep B Current Medical History: 59 y/o

female AA pt w/ PMH of Type 2 DM; HTN; DM related Neuropathy, suffering

from closed injury head trauma caused by basketball hoop and backboard

falling on her head. A CAT scan of her head & cervical spine came back

negative for fractures. She was scheduled for DC from ED but b/c of her ?

vision; gait problems; and persistent headache, she was admitted to West

Tower for inpatient rehab and continued med evaluation.

Psycho-Social History: No Hx of tobacco, alcohol or illicit drug use. Lives in

own one-story home. Widowed for 5 yrs. Has 25 yrs as a school teacher, Has

family support (daughters). NURSING PROCESS FORM: PART I

PATHOPHYSIOLOGY OF CURRENT DIAGNOSIS FROM TEXTBOOK Diagnosis and

Definition of Diagnosis: Dx: Post Blunt Force (Head) Trauma Definition: a

usually serious injury caused by a blunt object or collision with a blunt

surface (as in a vehicle accident or fall from a building) Etiology:

While various mechanisms may cause TBI, the most common causes include

motor vehicle accidents (eg, collisions between vehicles, pedestrians struck

by motor vehicles, bicycle accidents), falls, assaults, sports-related injuries,

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and penetrating trauma. Motor vehicle accidents account for almost half of the TBIs in the United States, and in suburban/rural settings, they account for most TBIs. In cities with populations greater than 100, 000, assaults, falls, and penetrating trauma are more common etiologies of head injury.

The male-to-female ratio for TBI is nearly 2: 1, and TBI is much more common in persons younger than 35 years. Diagnostic Procedures: The physical examination and the history of the exact details of the injury are the first steps in caring for a patient with head injury. The patient's past medical history and medication usage will also be important factors in deciding the next steps. Plain skull X-rays are rarely done for the evaluation of head injury. It is more important to assess brain function than to look at the bones that surround the brain.

Plain X-ray films may be considered in infants to look for a fracture, depending upon the clinical situation. Computerized tomography (CT) scan of the head allows the brain to be imaged and examined for bleeding and swelling in the brain. It can also evaluate bony injuries to the skull and look for bleeding in the sinuses of the face associated with basilar skull fractures. CT does not assess brain function, and patients suffering axonal shear injury may be comatose with a normal CT scan of the head.

Numerous guidelines exist to give direction as to when a CT should be completed in patients who present awake after sustaining a minor head injury. The Ottawa CT head rules apply to patients age 2 to 65. High Risk

- Glasgow Coma Scale less than 15, two hours after injury ?
- Suspect open or depressed skull fracture ?
- Sign of basilar skull fracture ?
- Vomiting more than once ?
- Older than 65 years of age

Medium Risk

- Amnesia before

impact greater than 30 minutes? •Dangerous mechanism of injury Signs, Symptoms, and Course of the Disease/Disorder:

It is important to remember that a head injury can have different symptoms and signs, ranging from a patient experiencing no initial symptoms to coma. A high index of suspicion that a head injury may exist is important, depending upon the mechanism of injury and the initial symptoms displayed by the patient. Being unconscious, even for a short period of time is not normal. Prolonged confusion, seizures, and multiple episodes of vomiting should be signs that prompt medical attention is needed. In some situations, concussion-type symptoms can be missed.

Patients may experience difficulty concentrating, increased mood swings, lethargy or aggression, and altered sleep habits among other symptoms. Medical evaluation is always wise even well after the injury has occurred. Treatment and Prognosis: The treatment of head injury may be divided into the treatment of closed head injury and the treatment of penetrating head injury. While significant overlap exists between the treatments of these 2 types of injury, some important differences are discussed. Closed head injury treatment is divided further into the treatment of mild, moderate, and severe head injuries.

Mild head injury Most head injuries are mild head injuries. Most people presenting with mild head injuries will not have any progression of their head injury; however, up to 3% of mild head injuries progress to more serious injuries. Mild head injuries may be separated into low-risk and moderate-risk groups. Patients with mild-to-moderate headaches, dizziness, and nausea are considered to have low-risk injuries. Many of these patients require only

minimal observation after they are assessed carefully, and many do not require radiographic evaluation.

These patients may be discharged if a reliable individual can monitor them. Patients who are discharged after mild head injury should be given an instruction sheet for head injury care. The sheet should explain that the person with the head injury should be awakened every 2 hours and assessed neurologically. Caregivers should be instructed to seek medical attention if patients develop severe headaches, persistent nausea and vomiting, seizures, confusion or unusual behavior, or watery discharge from either the nose or the ear. Patients with mild head injuries typically have concussions.

A concussion is defined as physiologic injury to the brain without any evidence of structural alteration. Concussions are graded on a scale of I-V. A grade I concussion is one in which a person is confused temporarily but does not display any memory changes. In a grade II concussion, brief disorientation and anterograde amnesia of less than 5 minutes' duration are present. In a grade III concussion, retrograde amnesia and loss of consciousness for less than 5 minutes are present, in addition to the 2 criteria for a grade II concussion.

Grade IV and grade V concussions are similar to a grade III, except that in a grade IV concussion, the duration of loss of consciousness is 5-10 minutes, and in a grade V concussion, the loss of consciousness is longer than 10 minutes. As many as 30% of patients who experience a concussion develop postconcussive syndrome (PCS). PCS consists of a persistence of any combination of the following after a head injury: headache, nausea, emesis,

memory loss, dizziness, diplopia, blurred vision, emotional lability, or sleep disturbances.

Fixed neurologic deficits are not part of PCS, and any patient with a fixed deficit requires careful evaluation. PCS usually lasts 2-4 months. Typically, the symptoms peak 4-6 weeks following the injury. On occasion, the symptoms of PCS last for a year or longer. Approximately 20% of adults with PCS will not have returned to full-time work 1 year after the initial injury, and some are disabled permanently by PCS. PCS tends to be more severe in children than in adults. When PCS is severe or persistent, a multidisciplinary approach to treatment may be necessary.

This includes social services, mental health services, occupational therapy, and pharmaceutical therapy. After a mild head injury, those displaying persistent emesis, severe headache, anterograde amnesia, loss of consciousness, or signs of intoxication by drugs or alcohol are considered to have a moderate-risk head injury. These patients should be evaluated with a head CT scan. Patients with moderate-risk mild head injuries can be discharged if their CT scan findings reveal no pathology, their intoxication is cleared, and they have been observed for at least 8 hours.

**Moderate and severe head injury** The treatment of moderate and severe head injuries begins with initial cardiopulmonary stabilization by ATLS guidelines. The initial resuscitation of a patient with a head injury is of critical importance to prevent hypoxia and hypotension. In the Traumatic Coma Data Bank study, patients with head injury who presented to the hospital with hypotension had twice the mortality rate of patients who did not present

with hypotension. The combination of hypoxia and hypotension resulted in a mortality rate 2.5 times greater than if neither of these factors was present.

Once a patient has been stabilized from the cardiopulmonary standpoint, evaluation of their neurologic status may begin. The initial GCS score provides a classification system for patients with head injuries but does not substitute for a neurologic examination. After assessment of the coma score, a neurologic examination should be performed. If a patient has received muscle relaxants, the only neurologic response that may be evaluated is the pupillary response. After a thorough neurologic assessment has been performed, a CT scan of the head is obtained.

The results of the CT scan help determine the next step. If a surgical lesion is present, arrangements are made for immediate transport to the operating room. Fewer than 10% of patients with TBI have an initial surgical lesion. Although no strict guidelines exist for defining surgical lesions in persons with head injury, most neurosurgeons consider any of the following to represent indications for surgery in patients with head injuries: extra-axial hematoma with midline shift greater than 5 mm, intra-axial hematoma with volume greater than 30 mL, an open skull fracture, or a depressed skull fracture with more than 1 cm of inward displacement. In addition, any temporal or cerebellar hematoma that is larger than 3 cm in diameter is considered a high-risk hematoma because these regions of the brain are smaller and do not tolerate additional mass as well as the frontal, parietal, and occipital lobes. These high-risk temporal and cerebellar hematomas are usually evacuated immediately. If no surgical lesion is present on the CT scan

image, or following surgery if one is present, treatment of the head injury begins.

The first phase of treatment is to institute general measures. Once appropriate fluid resuscitation has been completed and the volume status is determined to be normal, intravenous fluids are administered to maintain the patient in a state of euvolemia or mild hypervolemia. A previous tenet of head injury treatment was fluid restriction, which was believed to limit the development of cerebral edema and increased ICP. Fluid restriction decreases intravascular volume and, therefore, decreases cardiac output.

A decrease in cardiac output often results in decreased cerebral flow, which results in decreased brain perfusion and may cause an increase in cerebral edema and ICP. Thus, fluid restriction is contraindicated in patients with TBI. Another supportive measure used to treat patients with head injuries is elevation of the head. When the head of the bed is elevated to 20-30°, the venous outflow from the brain is improved, thus helping to reduce ICP. If a patient is hypovolemic, elevation of the head may cause a drop in cardiac output and CBF; therefore, the head of the bed is not elevated in hypovolemic patients.

In addition, the head should not be elevated (1) in patients in whom a spine injury is a possibility or (2) until an unstable spine has been stabilized. Sedation is often necessary in patients with traumatic injury. Some patients with moderate head injuries have significant agitation and require sedation. In addition, patients with multisystem trauma often have painful systemic injuries that require pain medication, and many intubated patients require sedation. Short-acting sedatives and analgesics should be used to



accomplish proper sedation without eliminating the ability to perform periodic neurologic assessments.

This requires careful titration of medication doses and periodic weaning or withholding of sedation to allow periodic neurologic assessment. Intravenous lidocaine administered along with rapid sequence induction before endotracheal intubation is not associated with significant hemodynamic changes in traumatic brain injury patients. [9] The use of anticonvulsants in patients with TBI is a controversial issue. No evidence exists that the use of anticonvulsants decreases the incidence of late-onset seizures in patients with either closed head injury or TBI.

Temkin et al demonstrated that the routine use of Dilantin in the first week following TBI decreases the incidence of early-onset (within 7 d of injury) seizures but does not change the incidence of late-onset seizures. [10] In addition, the prevention of early posttraumatic seizures does not improve the outcome following TBI. Therefore, the prophylactic use of anticonvulsants is not recommended for more than 7 days following TBI and is considered optional in the first week following TBI.

After instituting general supportive measures, the issue of ICP monitoring is addressed. ICP monitoring has consistently been shown to improve outcome in patients with head injuries. ICP monitoring is indicated for any patient with a GCS score less than 9, any patient with a head injury who requires prolonged deep sedation or pharmacologic relaxants for a systemic condition, or any patient with an acute head injury who is undergoing extended general anesthesia for a nonneurosurgical procedure. ICP monitoring involves placement of an invasive probe to measure the ICP.

Unfortunately, noninvasive means of monitoring ICP do not exist, although they are under development. ICP may be monitored by means of an intraparenchymal monitor, an intraventricular monitor (ventriculostomy), or an epidural monitor. These devices measure ICP by fluid manometry, strain-gauge technology, or fiberoptic technology. Intraparenchymal ICP monitors are devices that are placed into the brain parenchyma to measure ICP by means of fiberoptic, strain-gauge, or other technologies. The intraparenchymal monitors are very accurate; however, they do not allow for drainage of CSF.

Epidural devices measure ICP via a strain-gauge device placed through the skull into the epidural space. This is an older form of ICP measurement and is rarely used today because the other technologies available are more accurate and more reliable. A ventriculostomy is a catheter placed through a small twist drill hole into the lateral ventricle. The ICP is measured by transducing the pressure in a fluid column. Ventriculostomies allow for drainage of CSF, which can be effective in decreasing the ICP. A risk of symptomatic hemorrhage exists with ventriculostomy placement, and Bauer et al report from a retrospective study that an international normalized ratio (INR) of 1.2-1.6 is an acceptable range for emergent ventriculostomy placement in patients with TBI. [11] Once an ICP monitor has been placed, ICP is monitored continuously. No absolute value of ICP exists for which treatment is implemented automatically. In adults, the reference range of ICP is 0-15 mm Hg. The normal ICP waveform is a triphasic wave, in which the first peak is the largest peak and the second and third peaks are progressively smaller.

When intracranial compliance is abnormal, the second and third peaks are usually larger than the first peak. In addition, when intracranial compliance is abnormal and ICP is elevated, pathologic waves may appear. Lundberg described 3 types of abnormal ICP waves, A, B, and C waves. [12] Lundberg A waves, known as plateau waves, have a duration of 5-20 minutes and an amplitude of 50 mm Hg over the baseline ICP. After an episode of A waves dissipates, the ICP is reset to a baseline level that is higher than when the waves began.

Lundberg A waves are a sign of severely compromised intracranial compliance. The rapid increase in ICP caused by these waves can result in a significant decrease in CPP and may lead to herniation. Lundberg B waves have a duration of less than 2 minutes, and they have an amplitude of 10-20 mm Hg above the baseline ICP. B waves are also related to abnormal intracranial compliance. Because of their smaller amplitude and shorter duration, B waves are not as deleterious as A waves. C waves, known as Hering-Traube waves, are low-amplitude waves that may be superimposed on other waves.

They may be related to increased ICP; however, C waves can also occur in the setting of normal ICP and compliance. When treating elevated ICP, remember that the goal of treatment is to optimize conditions within the brain to prevent secondary injury and to allow the brain to recover from the initial insult. Maintaining ICP within the reference range is part of an approach designed to optimize both CBF and the metabolic state of the brain. Treatment of elevated ICP is a complex process that should be tailored

to each particular patient's situation and should not be approached in a "cookbook" manner.

Many potential interventions are used to lower ICP, and each of these is designed to improve intracranial compliance, which results in improved CBF and decreased ICP. Acute treatment of increased intracranial pressure The Monro-Kellie doctrine provides the framework for understanding and organizing the various treatments of elevated ICP. In patients with head injuries, the total intracranial volume is composed of the total volume of the brain, the CSF, intravascular blood volume, and any intracranial mass lesions. The volume of one of these components must be reduced to improve intracranial compliance and to decrease ICP.

The discussion of the different treatments of elevated ICP is organized according to which component of intracranial volume they affect. The first component of total intracranial volume to consider is the blood component. This includes all intravascular blood, both venous and arterial, and comprises approximately 10% of total intracranial volume. Elevation of the head increases venous outflow and decreases the volume of venous blood within the brain. This results in a small improvement in intracranial compliance and, therefore, has only a modest effect on ICP.

The second component of intracranial vascular volume is the arterial blood volume. Hypocapnia is capable of reducing cerebral blood flow 4% for each mm Hg change in PaCO<sub>2</sub>. The control mechanism is probably extravascular pH changes in fluid bathing cerebral resistor vessels, which alter smooth muscle intracellular calcium concentrations. This may be reduced by mild-to-moderate hyperventilation, in which the PCO<sub>2</sub> is reduced to 30-35 mm Hg.

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This decrease in PCO<sub>2</sub> causes vasoconstriction at the level of the arteriole, which decreases blood volume enough to reduce ICP.

The effects of hyperventilation have a duration of action of approximately 48-72 hours, at which point the brain resets to the reduced level of PCO<sub>2</sub>. This is an important point because once hyperventilation is used, the PCO<sub>2</sub> should not be returned to normal rapidly. This may cause rebound vasodilatation, which can result in increased ICP. Below a PaCO<sub>2</sub> of 25-30 Torr, CBF falls much less rapidly, presumably because of severe enough vasoconstriction to induce hypoxemia in brain tissues, limiting oxygen delivery. PaCO<sub>2</sub> tensions less than 25 Torr are sufficient to change brain metabolism into anaerobic, which increases acidosis.

Low arterial O<sub>2</sub> tensions influence CBF but to a lesser degree than PaCO<sub>2</sub>. No measurable changes in CBF occur during hypoxemia until the PaO<sub>2</sub> drops below 50 Torr, at which time CBF gradually increases. In addition to reducing CBF, the resultant respiratory alkalosis may reverse local tissue acidosis, which develops in cerebral edema, benefiting cellular respiration and restoring autoregulation. Within 48-72 hours, renal mechanisms for handling bicarbonate excretion compensate for altered PaCO<sub>2</sub> tensions, thereby normalizing cerebral pH and returning CBF to baseline values.

There are 3 paradoxes to hyperventilation therapy for the control of ICP.

- Since cerebral vasospasm is a serious concern in subarachnoid hemorrhage (SAH), attempts to create further vessel constriction by hyperventilation in order to decrease concomitant cerebral edema are rarely indicated unless the amount of edema is clinically emergent.
- Vessels in the damaged area of the brain have lost their autoregulatory control. While unaffected brain

regions would vasoconstrict normally to the stimulus of decreased PaCO<sub>2</sub>, damaged areas might vasodilate in response to diminished cerebral blood flow.

This can create a “reverse steal” phenomenon, where blood and nutrients are diverted away from “normal” areas of the brain and into “damaged” areas. This diversion would feed the increased metabolic requirement of damaged tissues, but the sum total effect may cause more harm to the rest of the brain. In addition, the increased hydrostatic pressure combined with the capillary permeability damage might, in some cases, paradoxically increase ICP in damaged areas. •Sudden increases in PaCO<sub>2</sub>, as a result of ventilator changes, often result in dramatic increases in CBF, and rapid deteriorations in the patient’s condition.

During hyperventilation, the cerebral bicarbonate level gradually adjusts to offset the lower level of CO<sub>2</sub>, maintaining normal pH. If the pCO<sub>2</sub> is allowed to rise suddenly, the excess CO<sub>2</sub> rapidly crosses the blood-brain barrier, but the bicarbonate level in the brain increases much less rapidly. The result is cerebral acidosis, with attendant cerebral vascular dilatation, increased cerebral blood volume, and elevated ICP, usually resistant to further hyperventilation. Unfortunately, little objective evidence exists that treatment by hypocapnia has significantly improved mortality or survival.

At best, it seems to be a temporary stop-gap measure until some other curative measure, such as surgery, might be attempted. Patients with the most prompt response to hyperventilation generally have the best prognosis for recovery. No evidence exists that hyperventilation therapy produces benefit in hypoxemic-anoxic encephalopathy. CSF represents the third

component of total intracranial volume and accounts for 2-3% of total intracranial volume. In adults, total CSF production is approximately 20 mL/h or 500 mL/d. In many patients with TBI who have elevated ICP, a ventriculostomy may be placed and CSF may be drained.

Removal of small amounts of CSF hourly can result in improvements in compliance that result in significant improvements in ICP. The fourth and largest component of total intracranial volume is the brain or tissue component, which comprises 85-90% of the total intracranial volume. When significant brain edema is present, it causes an increase in the tissue component of the total intracranial volume and results in decreased compliance and increased ICP. Treatments of elevated ICP that reduce total brain volume include diuretics, perfusion augmentation (CPP strategies), metabolic suppression, and decompressive procedures.

Diuresis and brain edema Diuretics are powerful in their ability to decrease brain volume and, therefore, to decrease ICP. Mannitol, an osmotic diuretic, is the most common diuretic used. Mannitol is a sugar alcohol that draws water out from the brain into the intravascular compartment. It has a rapid onset of action and a duration of action of 2-8 hours. Mannitol is usually administered as a bolus because it is much more effective when given in intermittent boluses than when used as a continuous infusion. The standard dose ranges from 0.25-1 g/kg, administered every 4-6 hours.

Because mannitol causes significant diuresis, electrolytes and serum osmolality must be monitored carefully during its use. In addition, careful attention must be given to providing sufficient hydration to maintain euvoemia. The limit for mannitol is 4 g/kg/d. At daily doses higher than this,

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mannitol can cause renal toxicity. Mannitol should not be given if the patient's serum sodium level is greater than 145 or serum osmolality is greater than 315 mOsm. Other diuretics that sometimes are used in patients with TBI include furosemide, glycerol, and urea.

Mannitol is preferred over furosemide because it tends to cause less severe electrolyte imbalances than a loop diuretic. Interestingly, mannitol and furosemide have a synergistic effect when combined; however, this combination tends to cause severe electrolyte disturbances. Urea and glycerol have also been used as osmotic diuretics. Both of these compounds are smaller molecules than mannitol and, as a result, tend to equilibrate within the brain sooner than mannitol; therefore, they have a shorter duration of action than mannitol. Urea has the additional problem that it can cause severe skin sloughing if it infiltrates into the skin.

Hypertonic saline (3%) has generated some interest in the treatment of intracranial hypertension secondary to brain edema because it is thought to be less disruptive to fluid and electrolyte balance than other diuretic agents. Boluses of mannitol can generate a dramatic diuresis, resulting in rapid intravascular depletion and potential kidney damage. Mannitol can cause as much as 1500 cc of fluid to diurese in the space of 2 hours, as intravascular fluid depletion occurs, hematocrit can rise, blood viscosity can increase, and clotting is enhanced. This makes the area of brain irritation much more amenable to stroke.

Saline 3% or 7.5% administered in continuous infusion generates a more predictable and gentle osmotic flow of brain intracellular water into the interstitial space. The maximum effect occurs after the end of infusion and is



visible over 4 hours. Hypertonic saline hydroxyethyl starch (HS-HES) seems to effectively lower ICP but does not increase CPP as much as does mannitol. Therapeutically, the limits of serum sodium and osmolality are in the range of 155-320. More research is needed to elucidate the exact method of action of hypertonic saline and the contraindications. Other supportive treatments

While awaiting possible operative therapy, other supportive treatments are as follows:

- Early extraventricular drainage of CSF is sometimes of value in controlling brain edema if there is a suspicion that the ventricles will progressively diminish in size because edema cannot be cannulated from a burr hole.
- Coughing and straining increase venous pressure, restricting drainage and backing up blood into the head, thereby increasing ICP.

Neuromuscular paralysis may decrease ICP by preventing sudden changes related to coughing or straining and by promoting systemic venous pooling that increases venous drainage from the head.

Any other restrictions to jugular blood drainage, such as a kinked neck from positioning in bed, increase ICP by retarding jugular drainage, transmitting pressure back into the brain.

- Trying to differentiate a drug-induced coma from an increased ICP-induced coma with a trial of naloxone (Narcan) is contraindicated, as it invariably induces agitation if the stupor is narcotic induced. Agitation increases catecholamine response, increases cardiac output, and increases blood flow to the head, thereby increasing hydrostatic pressure and ICP.

Decreased serum protein (albumin) from malnutrition causes a decreased serum osmolality compared to the osmolality in the surrounding tissues. This allows intravascular water to flow along the increased osmotic gradient into the tissues, increasing edema.

Hyperalimentation should be initiated as soon as possible if the course is likely to be protracted. •Boutique intravenous stabilizing cocktails have been said to maintain homeostasis of intravascular and extravascular fluid compartments, avoiding rapid fluid shifts that might adversely affect cerebral metabolism and edema.

Composed of an albumin, bicarbonate, and Lasix solution, the albumin increases intravascular colloid content, resisting fluid flow into the brain substance, the bicarbonate buffers pH changes, and the Lasix tends to promote a stable, consistent urine output, resisting intravascular fluid changes from renal compensations. This may be useful in diffuse brain edema to protect against further damage from vascular compartment shifts, but body physiology probably adapts to it rapidly, thereby limiting its effect.

These cocktails have not been proven to be effective as a treatment of SAH since they tend to promote diuresis and intravascular depletion. •Use of positive end-expiratory pressure (PEEP) for mechanical ventilation is controversial in TBI patients with acute lung injury/acute respiratory distress syndrome. Zhang et al found that PEEP can have a varied impact on blood, intracranial, and cerebral perfusion pressure in patients with cerebral injury. When applying this technique, mean arterial and intracranial pressure monitoring appears beneficial. [13] Management of cerebral perfusion pressure

CPP management involves artificially elevating the blood pressure to increase the MAP and the CPP. Because autoregulation is impaired in the injured brain, pressure-passive CBF develops within these injured areas. As a result, these injured areas of the brain often have insufficient blood flow, and

tissue acidosis and lactate accumulation occur. This causes vasodilation, which increases cerebral edema and ICP. When the CPP is raised to greater than 65-70 mm Hg, the ICP is often lowered because increased blood flow to injured areas of the brain decreases the tissue acidosis. This often results in a significant decrease in ICP.

Metabolic therapies are designed to decrease the cerebral metabolic rate, which decreases ICP. Metabolic therapies are powerful means of reducing ICP, but they are reserved for situations in which other therapies have failed to control ICP. This is because metabolic therapies have diffuse systemic effects and often result in severe adverse effects, including hypotension, immunosuppression, coagulopathies, arrhythmias, and myocardial suppression. Metabolic suppression may be achieved through drug therapies or induced hypothermia. Barbiturates are the most common class of drugs used to suppress cerebral metabolism.

Barbiturate coma is typically induced with pentobarbital. A loading dose of 10 mg/kg is administered over 30 minutes, and then 5 mg/kg/h is administered for 3 hours. A maintenance infusion of 1-2 mg/kg/h is begun after loading is completed. The infusion is titrated to provide burst suppression on continuous electroencephalogram monitoring and a serum level of 3-4 mg/dL. Typically, the barbiturate infusion is continued for 48 hours, and then the patient is weaned off the barbiturates. If the ICP again escapes control, the patient may be reloaded with pentobarbital and weaned again in several days.

Hypothermia may also be used to suppress cerebral metabolism. The use of mild hypothermia involves decreasing the core temperature to 34-35°C for

24-48 hours and then slowly rewarming the patient over 2-3 days. Patients with hypothermia are also at risk for hypotension and systemic infections. Another treatment that may be used in patients with TBI with refractory ICP elevation is decompressive craniectomy. In this surgical procedure, a large section of the skull is removed and the dura is expanded. This increases the total intracranial volume and, therefore, decreases ICP.

Which patients benefit from decompressive craniectomy has not been established. Some believe that patients with refractory ICP elevation who have diffuse injury but do not have significant contusions or infarctions will benefit from decompressive craniectomy. Management of elevated ICP involves using a combination of treatments. Each patient represents a slightly different set of circumstances, and treatment must be tailored to each patient. Although no rigid protocols have been established for the treatment of head injury, many published algorithms provide treatment schemas.

The American Association of Neurologic Surgeons published a comprehensive evidence-based review of the treatment of TBI, called the Guidelines for the Management of Severe Head Injury. In these guidelines, 3 different categories of treatments, standards, guidelines, and options are outlined. Standards are the accepted principles of management that reflect a high degree of clinical certainty. Guidelines are a particular strategy or a range of management options that reflect a high degree of clinical certainty. Options are strategies for patient management for which clinical certainty is unclear.

Prognosis: The outcome of TBI is related to the initial level of injury. While the initial GCS score provides a description of the initial neurologic condition, it does not correlate tightly with outcome. Various methods have been used in an attempt to predict the outcome of TBI, and these are beyond the scope of this discussion. However, one simplified model uses 3 factors, that is, age, motor score of the GCS, and pupillary response (ie, normal, unilateral unresponsive pupil, bilateral unresponsive pupils), to provide a probability of outcome.

The Traumatic Coma Data Bank analyzed 780 patients with head injuries and identified 5 factors that correlated with a poor outcome, as follows: (1) age older than 60 years, (2) initial GCS score of less than 5, (3) presence of a fixed dilated pupil, (4) prolonged hypotension or hypoxia early after injury, and (5) presence of a surgical intracranial mass lesion. An examination of the Crash Injury Research Engineering Network database found a significantly higher mortality rate among elderly motor vehicle accident victims (age > 60 years) compared to their younger counterparts. 15] Many methods exist for evaluating the outcome of TBI. A simple and commonly used method is the Glasgow outcome scale. This divides outcome into 5 categories, as follows: (1) good, (2) moderate disability, (3) severe disability, (4) vegetative, and (5) dead. The scale can be divided further into good outcomes (eg, good plus moderate disability) and poor outcomes (eg, severe disability, vegetative, dead). Reference (with page numbers, should be in the last 5 years and must be from a med-surgical textbook): Crippen, D. W. (2011, September 26). Head trauma treatment & management.

Retrieved from <http://emedicine.medscape.com/article/433855-treatment>

WORKSHEET INFORMATION Description \*Vital signs frequency: 3/3/13, 1220: pain 6/10; BP 136/67; HR 63; Temp 97. 4 F/36. 3 C; Resp 18bpm; SpO2 97% 3/4/13, 0900: pain 0/10; BP 142/70; HR 62; Temp 97. 2 F/36. 2 C; Resp 17bpm; SpO2 97%; pre-brkfst glucose 103 Weight 118 Kg/BMI 40. 9 \*I&O (not charted) Intake this shift 3/4/13: 3-6 oz. cups of water = 540mL; 2-4 oz. cups of tea = 240mL; 1-6 oz. cup of juice = 180mL; Total of 960mL; Avg. 120mL/hr \*Urinary Elimination Continent, (not charted) \*Bowel Elimination Continent, LBM was 3/3/13 NG tube N/A \*Diet Lo Cal 1800, Diabetic \*Positioning or Turn self w/ bedrails and mod assist \*Bath Type self w/ mod assist of bed transfer and gaiting \*Activity Level gaiting 30 ft w mod assist Dressing Change/Site N/A Orthopedic Device/Care N/A Scheduled Procedures CT Scan (head) 3/3/13 O2 at L/Min via: Room Air I. V. Fluid Type N/A I. V. Site/rate (Hep Lock IV) N/A \*Allergies NKA Code Status Full Isolation Status None Standard Precaution \*Required Critical Information NURSING PROCESS FORM: PART I – REVIEW OF SYSTEMS/NURSING HEALTH Subjective and Objective Data

General Appearance (Posture, expression, first impressions) Pt is obese (BMI 40. 9) AA female, grooming appropriate, sitting up in wheelchair, is responsive and has a pleasant manner, getting ready to eat brkfst, I feel the pt has some anxiety as she stated “ I am worried about my injury and if I can go back to work. I think I may have to retire. ” Neurological (pain and its perception, last hearing or visual check, LOC, papillary response, signs of conduction deficits, speech pattern, orientation): Pain 0/10 but pt states “ I have a headache whenever my pain medication wears off. Pt can’t

remember last visual but she is wearing an ordered eye patch for her diplopia & blurred vision; Pt can't remember last hearing check but no sign of hearing impairment while talking to pt; GCS (15) E-4, V-5, M-6; Orientation positive to person, place, & day; PERRLA; Speech pattern normal; Musculoskeletal (mobility, safety, range of motion, atrophy or edema, prosthetic): Mobility w/ mod assist; Transfers w/ mod assist; Gaiting w/ mod assist from two attendants; ?

ROM and muscle weakness R-side extremities; Edema R ankle +3 pitting; no atrophy; no prosthetics; Cardiovascular (rate, rhythm, quality of pulses, PMI, capillary refill, bedside monitoring device, 12 Lead ECG report): HR 67; Rhythm regular; Radial pulse +3, Dorsalis +1; BP 124/74 L upper arm; Cap refill < 3 sec. ; no bedside monitoring; Pulmonary (tobacco history, shortness of breath, breath sounds, rate, rhythm, cough): No tobacco, mild SOB; Breath sounds clear; Rate 17 bpm; Rhythm even; No cough;

Gastrointestinal (bowel sounds, appetite, weight gained or lost, elimination pattern, nutritional supplements, date of last bowel movement): Normal bowel sounds x4 quads; appetite good (intake 100% of brkfst & lunch); elimination pattern continent LBM 3/3/13; Lo Cal 1800 diet & diabetic diet; Reproductive (last breast or testicular self-examination, mammogram, pelvic or prostate exam, LMP, pain): N/A Endocrine (diagnosed at what age, last HgbA1c or associated lab work): DM type 2 Dx'ed at 41 y/o; glucose finger-stick pre-brkfst 103, pre-lunch 113, no glucose required.

EENT (eyes, nose or sinus problems, swallowing difficulties, dental or oral problems) Blurred vision L&R eyes & diplopia; hearing slight ringing x2; had some epistaxis upon admission to ED on 3/1/13 but not in last 24 hrs; no

difficulty swallowing; no dental or oral problems; Renal (current urinalysis, urinary devices, voiding pattern, urgency, burning, pain, etc. ): No urinalysis; no urinary devices; voiding pattern continent, no urinary retention; no urgency, burning, or pain; I&O's not charted; Integumentary (skin undertone, turgor, edema, temperature, etc. ): Skin warm & dry; no lesions; turgor good