Traumatic Brain Injury

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7.1 Introduction

7.1.1 Epidemiology

Despite prevention efforts, pediatric head trauma remains the most common cause of serious injury and death in children. Seventy-five percent of children who are hospitalized secondary to trauma sustain a traumatic brain injury (TBI). Most pediatric TBI is mild in severity, although central nervous system (CNS) injury is the most common cause of pediatric traumatic death (Greenberg 2010). While the statistics will vary in other countries, those in the United States are illustrative. In the year 2007, there were a total of 5,500 pediatric TBI-related deaths in the United States. The Centers for Disease Control (CDC) data from 1997 to 2007 shows that the rate of TBI-related deaths has decreased in all pediatric age groups. The rate decreased by 8.2 % from 1997 to 2007 and by 11.4 % in the earlier period from 1989 to 1998. The leading causes of TBI-related deaths in young children (birth to 1½ years) are falls, motor vehicle accidents (MVAs), and abuse, whereas the causes in adolescents shift to MVAs and assaults (Doppenberg and Ward 2008). TBI-related deaths from MVAs are higher in adolescents (12 per 100,000 in 15–24-year-olds) than for any other age group throughout the lifespan (Coronado et al. 2011).

Pediatric TBI is also a substantial contributor to the health-related financial burden in the United States. According to Schneier et al. (2006), 50,658 pediatric (0–17 years) TBI hospitalizations in the year 2000 resulted in over $1 billion dollars in inpatient expenses. In addition, many childhood survivors of severe TBI are left with varying degrees of permanent disability and require ongoing rehabilitative care.

Pediatric TBI-related injuries are often preventable. Prevention efforts have been directed toward car seat, seat belt, and helmet safety laws, as well as injury prevention education programs (Coronado et al. 2011; Ragheh 2008). The reduction in pediatric deaths in the United States referenced above can be attributed in part to such efforts.

7.2 Pediatric Anatomy and Physiology in Head Trauma

The adage "children are not just small adults" holds true when discussing pediatric head trauma. The pediatric craniocerebral anatomy increases the child's vulnerability to head trauma, but also protects against worsened

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severity and outcome. In general, children under the age of 2 years or who are nonverbal require a higher level of suspicion for injury. They lack the ability to communicate what has happened to them and what they are feeling. The exam is often less revealing based on the child's developmental age.

7.2.1 Skull

The physically larger and proportionately heavier pediatric cranium, together with the greater laxity of the cervical spine, creates a fulcrum and predisposes children to traumatic injury of the head and cervical spine. An infant's skull consists of a single layer, with open sutures between eight cranial bones. Open cranial sutures are protective against gradual increases in intracranial pressure, such as from tumors or hydrocephalus. Rapidly expanding mass lesions, however, are not tolerated and result in increased intracranial pressure (ICP). The head circumference of infants should be measured and recorded on admission and daily, as a rapidly increasing head circumference is indicative of increased ICP. Presence of bulging or firm fontanels, with the infant calm and in an upright posture, can also be an indicator of increased ICP. The skull becomes a closed system by 4 years of age (Doppenberg and Ward 2008).

The infant's skull is thinner, softer, and more deformable when fractured, but heals quickly after fracture due to accelerated bone growth. The temporal and parietal regions are the thinnest cranial bones and the most common sites of accidental fracture. The thickest cranial bones are the frontal and occipital. Occipital fractures are related to more serious brain injuries due to the increased force necessary to generate a fracture in the thickest bone of the skull (Greenberg 2010). The pediatric skull can absorb a significant impact with little external evidence of significant intracranial injury. When evaluating the head-injured child, the nurse must consider all external indications, such as bruising, swelling, and lacerations, as well as the mechanism of injury and the degree of neurologic deficit.

7.2.2 Brain

The pediatric brain is softer due to higher water content and less white matter myelinization. The subarachnoid space is wider. The thin pediatric skull, soft brain, and large subarachnoid space allow increased movement of the brain within the skull, which makes the child more susceptible to brain injury, including extraparenchymal hemorrhage, shearing or tearing of neuronal processes, and diffuse axonal injury (Barkovich 2005; Dias 2004; Doppenberg and Ward 2008). Young children are more susceptible to shear-type injury and less susceptible to mass lesions, with the incidence of mass lesions increasing to that of an adult as the child ages (Doppenberg and Ward 2008). The thin cranium, pliable brain, and open sutures allow infants and toddlers to tolerate mass hemorrhage better than older children. The pressure-volume curve is shifted to the left, meaning that children tolerate acute increases in intracranial volume poorly. Children have a smaller intracranial space, in which smaller increases in volume produce exponentially larger increases in ICP.

7.3 Initial Evaluation and Resuscitation

Following traumatic injury involving the head or neck, the child should arrive in an emergency department secured on a backboard and wearing a well-fitted cervical collar. A comprehensive multidisciplinary trauma evaluation should be performed to assess for multiple injuries. Immediate baseline neurologic examination should occur simultaneously with evaluation and resuscitation of airway, breathing, and circulation. Neurologic examination should be performed consistently and be clearly documented on arrival and at frequent intervals. Critical evaluation of trends in the neurologic exam, vital signs, and ICP measurements is the most sensitive method of detecting early neurologic deterioration (Andrews and Hammer 1997).

Table 7.1 Initial history and physical in TBI

Subjective: Witness to mechanism of event (i.e., police, EMT, parent report, photographs), loss of consciousness (LOC), anterograde or retrograde amnesia, witnessed traumatic seizure, abnormal behavior or vomiting, cardiorespiratory compromise/resuscitation, immobilization of cervical spine, improved or worsened exam after initial resuscitation
Objective: General survey for multiple traumatic injuries, including spine. Survey for cranial injury: scalp hematoma; laceration, contusion, or abrasion; open or penetrating intracranial injury. Evidence of basal skull fracture includes: Battle's sign, raccoon eyes, otorrhea, rhinorrhea, and hemotympanum. Facial fractures (LeFort – facial instability or step-off) may indicate serious neurologic injury
Physical examination: General assessment provides suspicion of location and severity of injury
Vital signs, LOC/mental status, GCS, orientation
Cranial nerve exam:
Olfactory nerve (CN I)
Optic nerve (CN II) – assess vision (Snellen card, finger counting, hand motion, light perception). Note: may have temporary cortical blindness 1–2 days after blow to back of head
Oculomotor nerve (CN III) – pupillary size and reaction to light, ptosis, abducted gaze
Oculomotor (CN III), trigeminal (CN IV), and abducens (CN VI) nerves – extraocular eye movements
Trochlear nerve (CN V) – facial sensation, sensory portion of corneal reflex
Facial nerve (VII) – facial movement, motor portion of corneal reflex
Acoustic nerve (CN VIII) – hearing
Glossopharyngeal (CN IX) and vagus (CN X) – intact gag and cough
Motor exam: cooperative – assess strength x4 extremities, uncooperative – movement to noxious stimuli (caution: differentiate seizure from posturing, and avoid mistaking spinal cord reflexive movement as indication of cerebral function)
Sensory exam: cooperative – differentiate tickle and pinch in all extremities, if uncooperative – assess for grimace and vocalization to central painful stimuli
Reflexes: DTRs, Babinski reflex, clonus
History: Any previous head injury – timing, frequency, severity, other PMH such as bleeding dyscrasias, seizures, medications and allergies, NPO status, alcohol or drug use, metabolic abnormality (i.e., IDDM)
Previous developmental or cognitive impairments (Greenberg 2010)

Infants and young children are not able to communicate the circumstances of their injuries. Therefore, the caregiver must rely on the report of parents or other historians regarding previous medical history, baseline neurologic/cognitive/developmental deficits, and vital information to determine the circumstances of the child's injury. Circumstances of the accident or trauma are vital to determine the mechanism of injury and the potential severity of the child's injuries. First responders provide vital information such as a photograph, description of the scene, or a police report. Witnessed loss of consciousness, amnesia before (retrograde) or after (anterograde) the event, posttraumatic seizures, initial clinical evaluation (GCS), cardiorespiratory collapse or hypotension, interventions provided, and patient's response (worsened or improved) should be included in the paramedic's report to the trauma team (Andrews and Hammer 1997; Dias 2004). Table 7.1 includes elements of the traumatic brain injury history and physical examination.

7.3.1 Primary Versus Secondary Mechanism of Injury

Primary injury includes that which is present at impact, to include cortical contusions, lacerations, diffuse axonal injury (DAI), and brainstem injury. Secondary injury is that which develops subsequent to the impact. This includes injury from hemorrhage, edema, hypoxemia, ischemia (increased ICP or shock), and vasospasm. All patient management decisions and interventions are directed at preventing secondary injury (Greenberg 2010).

7.4 Neurologic Assessment and Deterioration in Pediatric Head Trauma

7.4.1 General Assessment

Inspection for external trauma, such as scalp or facial swelling, abrasions, laceration, or ecchymosis, can indicate traumatic brain injury. Palpable step-off or depression indicates skull fracture, which may be associated with contusion of brain, laceration of dura or brain, and CSF leak. Significant scalp swelling in the infant may be indicative of hemorrhage, which can cause anemia with pallor and tachycardia. A basilar skull fracture in the base of the anterior fossa causes "raccoon eyes" or periorbital ecchymoses and can be associated with rhinorrhea (CSF leak from the nares). Fracture in the base of the middle fossa causes "Battle's sign," or postauricular ecchymoses, and can be associated with otorrhea (leak of CSF from the ear). Hemotympanum can indicate temporal or basilar skull fracture. Otorrhea indicates disruption of the tympanic membrane (TM) related to temporal skull fracture. The cervical spine must be immobilized and protected from spinal cord injury until radiographic clearance is accomplished (Dias 2004; Greenberg 2010). The entire spine is immobilized, inspected, and palpated for deformity, swelling, tenderness, and crepitance. Refer to the spine chapter.

7.4.2 Vital Functions

Every patient assessment must begin with evaluation of adequate airway, breathing, and circulation, which are vital to sustain life. A decreased level of consciousness after TBI can interfere with protection of the pediatric airway and adequate ventilation. Inadequate ventilation results in hypercarbia and hypoxia, which cause vasodilation and secondary ischemic brain injury. Vasodilation and resultant ischemia contribute to further increases in ICP. Vital control centers located within the brainstem regulate respiratory and cardiac functions. Brainstem pathophysiology can be identified by changes in the vital signs. The following abnormal respiratory rate and patterns indicate neurologic dysfunction secondary to progressive brainstem compression in increasing ICP (Dias 2004; Greenberg 2010; Hickey 2009):

- Cheyne-Stokes: rhythmic cycles of breaths, which gradually increase in amplitude and then trail off, followed by an expiratory pause; indicates diencephalic injury or bilateral hemispheric dysfunction
- Central neurogenic hyperventilation (rare): increased rate and depth of respirations, indicates pons dysfunction
- Apneustic (rare): a pause at full or prolonged (slow and deep) inspiration, indicates injury to the pons
- Ataxic: no pattern in rate or depth, indicates medulla or lower brainstem dysfunction with impending herniation, indicates injury to the respiratory centers in the medulla (also known as agonal respirations)
- Apnea: respirations cease with herniation

Following loss of autoregulation (the ability of the brain to maintain perfusion despite changes in systemic perfusion), the cerebral blood flow is dependent on the systemic blood pressure. Adequate systemic perfusion is critical following pediatric TBI because hypotension causes secondary injury and is associated with poor outcome (Zebrack et al. 2009; Pigula et al. 1993). Prevention and immediate correction of hypoxia and hypotension are imperative. A study by Zebrack et al. (2009) found that the odds of death and long-term disability were both more than three times higher for children who did not have their hypotension addressed in the field. In children, hypotension is a late sign, which indicates compromised systemic and likely cerebral perfusion. Other earlier indications of poor systemic perfusion include tachycardia, decreased LOC, signs of inadequate skin perfusion (capillary refill > 2 s), and decreased urine output (less than 1 cm^3/kg/h). Hypertension occurs as a compensatory mechanism to maintain cerebral perfusion in the face of increased ICP. The mechanism, known as Cushing's response, is activated by decreased cerebral blood perfusion and includes increased systolic blood pressure, widened pulse pressure, and bradycardia

(Hickey 2009). Cushing's triad is a classic presentation of vital signs, including hypertension, bradycardia, and increasingly abnormal respiratory pattern, which is a late and ominous sign of severe increased ICP and impending herniation (Dias 2004; Greenberg 2010; Hickey 2009).

7.4.3 Level of Consciousness

The child's level of consciousness (LOC) and whether it is worsening or improving are the most important indicators of neurologic status (Dias 2004; Curley and Moloney-Harmon 2001). The neurologically intact child is awake, alert, and responsive to his/her surroundings. Level of responsiveness varies with the developmental age of the child. Infants should respond to feeding and measures to console them (Curley and Moloney-Harmon 2001). Toddlers and older children should recognize and respond to their parents. Older children and adolescents should be able to follow commands. Children of all ages should respond to and withdraw from painful stimulus. After neurologic injury, pediatric head-injured victims may have degradation in LOC as follows: subtle restlessness, disorientation, and agitation; somnolence (arouses to full consciousness and resumes sleep if not stimulated); lethargy (requires vigorous stimulation to arouse to full consciousness); stupor (nearly unconscious, may moan or withdraw from pain); and finally coma (unresponsive) (Hickey 2009; Greenberg 2010). A worsening LOC suggests neurologic deterioration. Caution should be exercised not to mistake neurologic deterioration for pain or anxiety, as treatment of the same with narcotics or antianxiety agents will further blunt the neurologic exam and delay treatment. Any subtle change from documented baseline, including parental concern that child is "not acting right," must be taken seriously and reported to the physician.

7.4.4 Glasgow Coma Scale

The Glasgow Coma Scale (GCS) and the modified pediatric GCS measure the child's level of consciousness (Table 7.2). As pediatric responses are different from those of adults, the GCS was modified to allow for consistent, objective, serial measurements of the child's level of neurologic responsiveness following TBI. The scale considers the child's best response, following adequate central stimulation to eye opening, motor, and verbal responses, with each assigned a score and the three scores totaled (Hickey 2009; Greenberg 2010). The scores range from 3 (lowest score indicating no response) to 15 (highest score indicating intact neurologic status). A worsening GCS and decreased level of responsiveness indicate a rise in ICP (Dias 2004). A change of two or more points on the GCS score is very significant and should be reported to the physician immediately.

It is important, when assessing responsiveness, for the nurse to use an adequately painful, central stimulus to elicit the child's best response. Application of firm pressure to the mandible, sternum, supraorbital area, or sternocleidomastoid muscle provides an adequate central painful stimulation (Marcoux 2005). Peripheral painful stimulation should be avoided, as it can elicit a spinal reflex. The spinal reflex arc is a response to peripheral sensory stimulation in which the sensory afferent fibers carry stimulation to the dorsal root and spinal cord. The signal synapses in the cord with the motor neuron in the anterior horn. Motor efferent fiber signals travel back to the neuromuscular junction, which elicits a muscle contraction (Hickey 2009; Young et al. 2008). The spinal reflex should not be confused as a demonstration of cerebral function.

The immediate post-resuscitation modified Glasgow Coma Scale (GCS) for Infants and Children score is used to rate the severity of pediatric head trauma, as well as to predict outcome. The severity of head trauma is determined by the following:

- GCS 14–15 = mild head trauma
- GCS 9–13 = moderate head trauma
- GCS < or equal to 8 = severe head trauma (Greenberg 2010)

Coma is defined as the inability to arouse or interact with the environment. A GCS of 8 or less is an operational definition of coma (Greenberg 2010).

Table 7.2 Modified Glasgow Coma Scale for Infants and Children (Hickey 2009)

Response	Child	Infant	Score
Eye opening	Spontaneous	Spontaneous	4
	Verbal stimuli	Verbal stimuli	3
	Pain only	Pain only	2
	No response	No response	1
Verbal response	Oriented, appropriate	Coos and babbles	5
	Confused	Irritable cry	4
	Inappropriate words	Cries to pain	3
	Incomprehensible words or sounds	Moans to pain	2
	No response	No response	1
Motor response	Obeys commands	Moves spontaneously and purposeful	6
	Localizes painful stimulus	Withdraws to touch	5
	Withdraws to pain	Withdraws to pain	4
	Flexion to pain	Decorticate posture (abnormal flexion) to pain	3
	Extension to pain	Decerebrate posture (abnormal extension) to pain	2
	No response	No response	1

7.5 Cranial Nerve (CN) Evaluation

The cranial nerves originate in the brainstem, with CN I through IV from the midbrain, CN V through VIII from the pons, and CN IX through XII from the medulla. Evaluation of cranial nerve and brainstem function is valuable to locate neurologic injury. Rostral (head)-caudal (tail) deterioration with worsening increased ICP manifests as an anatomic "picking off" (dysfunction) of the cranial nerves in chronologic order as the pressure progresses downward through the brainstem. It is critical for the nurse to recognize this subtle deterioration early so that there is potential to reverse the process before herniation and death occur.

7.5.1 Visual Acuity

Following TBI, it is essential to assess for presence of bilateral vision, which indicates innervation by the optic nerve (CN II). Presence of a squint to light in an infant indicates intact vision. Vision in older children can be assessed on a continuum progressing from abnormal to normal, including blindness, light perception, hand motion, finger counting, to full baseline vision (Wilson-Pauwels et al. 2010). Presence of papilledema on a fundoscopic exam indicates presence of increased ICP. This finding presents 12–24 h after injury, however, and its absence should not delay treatment when other findings are consistent with severe brain injury (Dias 2004). The presence of retinal hemorrhages with subdural hematomas is a classic finding in abusive head trauma (AHT) (Vinchon et al. 2005), but can also be seen with high-impact accidental injuries.

7.5.2 Pupillary Response

Pupillary response represents a balance between sympathetic and parasympathetic systems, wherein dysfunction in one system results in unopposed action of the other (Dias 2004). Pupillary response is innervated by the third cranial nerve. The pupils are normally equal in size, round, and reactive to light and accommodation, thus the acronym PERRLA. When assessing pupillary response, darken the room. Bring the light in from the periphery, and note direct (same side) and consensual (opposite side) response to light; repeat with the other eye. Accommodation, assessed by directing gaze at a distant object, causes the pupils to dilate. Then gaze is directed to a near object (finger), which causes the pupils to constrict and converge on the near object (Young et al. 2008; Hickey 2009).

Abnormal mydriasis (pupillary dilation) is caused by unopposed sympathetic input, whereas miosis (pupillary constriction) is due to unopposed parasympathetic input (Greenberg 2010). Bilateral dilated (mydriatic) and nonreactive pupils, caused by unopposed sympathetic input, indicate an injury to the oculomotor (CN III) nucleus in the midbrain, or CN III injury due to trauma or increased intracranial pressure (Dias 2004). Unilateral mydriasis in TBI suggests either direct orbital trauma, transtentorial (uncal) herniation, or expanding mass hemorrhage on the same (ipsilateral) side as the dilated pupil. A new finding of pupillary inequality, even by only 1 mm, must be taken seriously and reported to the physician. Bilateral mydriasis can also occur following seizure or medications such as atropine that mimic the sympathetic response. A pharmacologically dilated pupil is very large (7–8 mm), whereas mydriasis due to CN III compression is typically 5–6 mm (Greenberg 2010). The nurse should be aware of what medications are given and notify other caregivers of iatrogenic pupillary dilatation. Miosis occurs with injury to the pons or carotid artery and with administration of narcotics or other miotic drugs.

Hippus is a spasmodic, rhythmic pupillary response to light manifested as alternating dilation and constriction (Greenberg 2010). Hippus can be a normal variant or indicate increasing pressure on CN III, with impending transtentorial herniation (Hickey 2009). Anisocoria (inequality of the pupil size) is a variant of normal in approximately 20 % of the population (Greenberg 2010; Hickey 2009). Physiologic anisocoria is a pupillary difference of <1 mm, whereas pathologic anisocoria due to increased ICP will manifest as a pupillary difference of >1 mm. Common causes of pathologic anisocoria include CN III palsy or compression caused by transtentorial herniation, Parinaud's syndrome, and Horner's syndrome. Parinaud's syndrome, caused by a lesion (tumor) or pressure (increased ICP or hydrocephalus) exerted on the tectum of the midbrain, results in impaired upgaze ("sunsetting"), impaired convergence, dilated and fixed pupils, and lid retraction. Horner's syndrome occurs with interruption of sympathetic input to the eye and face, resulting in a unilateral reactive miotic pupil, ptosis, and lack of perspiration on the same side of the face.

7.5.3 Extraocular Eye Movements

Eye position and movement are controlled by cranial nerves III (oculomotor), IV (trochlear), and VI (abducens), as well as the cerebral hemispheres and the brainstem. Extraocular eye movements (EOM) are assessed by having the conscious child follow the examiner's finger in the pattern of an "H" (cardinal fields of gaze). Cranial nerve injury following TBI is manifested as extraocular eye muscle weakness, resulting in abnormal eye position in the conscious or unconscious child. The third CN innervates four of the six ocular muscles, which control all directions of gaze except downward and inward (CN IV) and lateral (CN VI). When control of eye movement in one direction is lost, there is overcompensation of positioning of the eye in the opposite direction. The following (Table 7.3) is a limited review of abnormal eye position and related etiology (localization of injury) (Young et al. 2008; Dias 2004). Saccadic eye movements (rapid, voluntary movements to search a field) are controlled by the frontal gaze centers, where injury causes deviation toward the lesion. Pursuit movements (slow, involuntary movements keeping the eyes fixated on a moving target) are controlled by the occipital gaze centers.

7.5.4 Brainstem Reflex Exam

The trigeminal (CN V) nerve innervates the sensory portion of the corneal reflex, where stimulation of blowing into the child's eye elicits eye closure. The motor response of blinking is innervated by CN VII. A unilateral facial (CN VII) weakness or hearing loss (CN VIII) can occur with a basilar skull fracture. Integrity of the vestibular function (CN VIII) is assessed by performing oculovestibular and oculocephalic testing, which indicates the presence or absence of brainstem function in the comatose patient (Table 7.4) (Greenberg 2010; Wilson-Pauwels et al. 2010). Intact gag and cough reflexes assess

Table 7.3 Etiology of abnormal eye position in pediatric head trauma

Location of lesion	Eye deviation	Hemiparesis	Other
Frontal lobe injury	Toward lesion	Opposite	
Expanding mass hemorrhage	Toward lesion	Opposite	
Occipital injury	Toward lesion		Hemianopsia (contralateral loss of vision)
Seizure	Away from lesion	Same side	
CN III (oculomotor)	Down and out (exotropia). Also causes: ptosis, dilated pupil, unable to accommodate	Uncal herniation: contralateral hemiparesis or motor posturing	Uncal herniation: unilateral fixed, dilated pupil (↑ICP causes pressure on CN III nucleus)
CN IV (trochlear)	Elevates (inability to look down/in); diplopia		CN IV injury (rare)
CN VI (abducens)	Loss of lateral gaze; inability to abduct. Causes double vision with lateral gaze to affected side; squint and head tilt		↑ICP secondary to trauma, skull (clivus) fracture. Originates in pons; longest intracranial course increases risk of injury
Parinaud's syndrome	Convergence and accommodation lost; upward gaze palsy (sunsetting sign); pupils fixed, dilated. Infants unable to fix/follow		Elevated ICP, hydrocephalus; mechanism is pressure on the tectum of brainstem

Table 7.4 Brainstem reflexes assess BS function between the pons and the oculomotor (CN III) nuclei in the midbrain

Brainstem reflex	Awake	Comatose	Brain death
Oculovestibular (cold calorics). *Caution*: *must have intact TM*. Elevate HOB, 60–100 ml ice water instilled into ear	*Awake or obtunded with intact brainstem* – slow ipsilateral gaze; then rapid contralateral nystagmus "COWS" (Cold Opposite, Warm, Same). Refers to direction of nystagmus	*Comatose* – conjugate, tonic eye deviation toward stimulus; no nystagmus	*Brain death* – no eye movement
Oculocephalic (doll's eyes). *Caution*: *Do not perform unless C-spine clearance obtained*	*Awake* – eyes move with or away from (contraversive to) lateral head rotation	*Comatose with intact BS* – contraversive conjugate eye movement (positive doll's eyes)	*Brain death* – no eye movement

continuity of the glossopharyngeal (CN IX) and vagus (CN X) nerves.

7.5.5 Motor Exam

The infant should have dominant flexor tone, but relax to easily perform full range of motion. The child's ability to follow command is assessed by asking them to perform a purposeful and reproducible task, such as holding up two fingers. Note whether the child initiates movement spontaneously or what stimulus is required to elicit movement. Note the symmetry and quality of strength using the following scale: 0 – no muscle contraction; 1 – palpation of trace contraction; 2 – movement without gravity; 3 – movement against gravity, but not resistance; 4 – movement against some resistance; and 5 – movement against full resistance (Hickey 2009). Weakness on the side opposite the lesion with hypertonicity and hyperreflexia indicates cerebral or upper motor neuron injury. Whereas a lower motor neuron injury presents with weakness or paralysis, on the same side as the lesion or bilaterally,

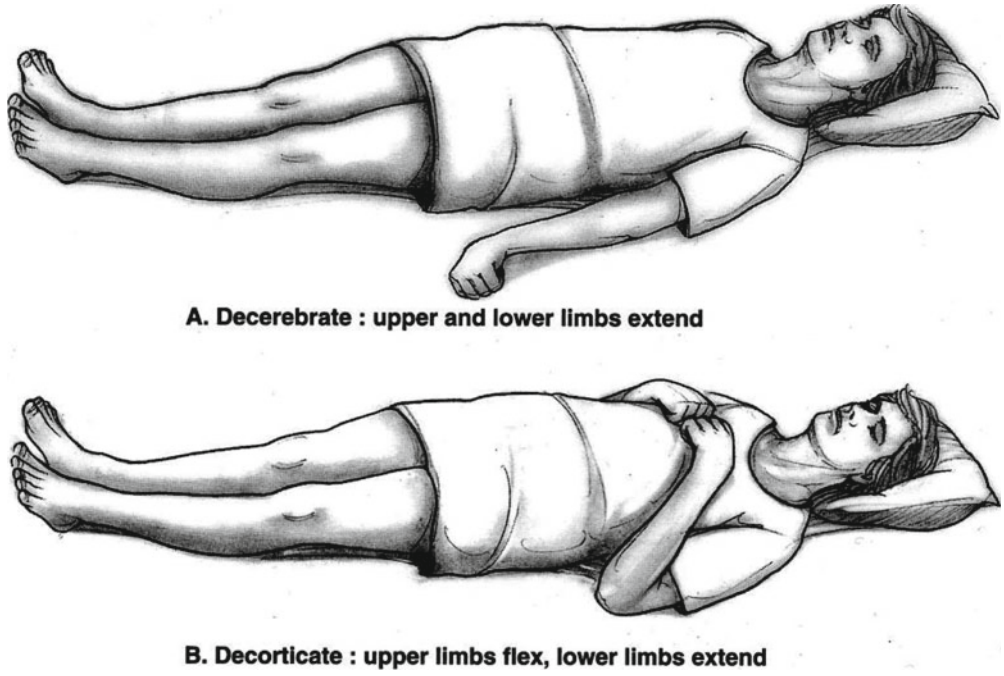

Fig. 7.1 (**a**) Abnormal posturing indicates brainstem compression in the comatose patient. Decerebrate posturing with abnormal upper extremity (*UE*) and lower extremity (*LE*) extension (*late*). (**b**) Decorticate posturing with abnormal UE flexion and LE extension (*early*) (Reprinted with permission from Young et al. (2008))

hypotonia, and areflexia. Cerebellar injury results in hyporeflexia, ataxia, and dysarthria (Young et al. 2008).

Abnormal flexion or extension posturing indicates severe traumatic brain injury. Posturing indicates neurologic activity (or inactivity) secondary to brainstem compression and impending herniation in comatose patients (Young et al. 2010). Deterioration of neurologic status occurs in a rostral (head) to caudal (tail) progression. This is true of cranial nerve and brainstem dysfunction with impending herniation. Decorticate posturing implies a more rostral lesion and a better prognosis (Greenberg 2010). Decorticate posture is abnormal flexion of the upper extremities with extension of the lower extremities and is indicative of disinhibition of the corticospinal pathways above the midbrain. Decerebrate posturing implies further deterioration and impending herniation, as it indicates disinhibition of the pons and medulla. Decerebrate posturing includes abnormal extension of the upper and lower extremities (Fig. 7.1). Posturing may be reversible or may represent impending brain death. Progression from decorticate to decerebrate indicates worsening brainstem function, whereas progression from decerebrate to decorticate indicates improvement. Figure 7.2 illustrates the brainstem centers that are compressed by downward herniation, progressing from decorticate to decerebrate posturing, and finally herniation (brain death) (Young et al. 2008). Posturing may be reversible, but is associated with a more ominous outcome.

7.5.6 Reflexes

A reflex is an autonomic nervous system motor response to stimulation (O'Toole 1992). The stimulus (striking tendon) travels via sensory (afferent) fibers to the dorsal ganglion and anterior horn of the spinal cord. The ventral horn relays the motor (efferent) signal back to the muscle, causing a reflexive contraction. This chain of events is referred to as the reflex arc. Deep tendon reflexes (DTR) or muscle stretch reflexes are

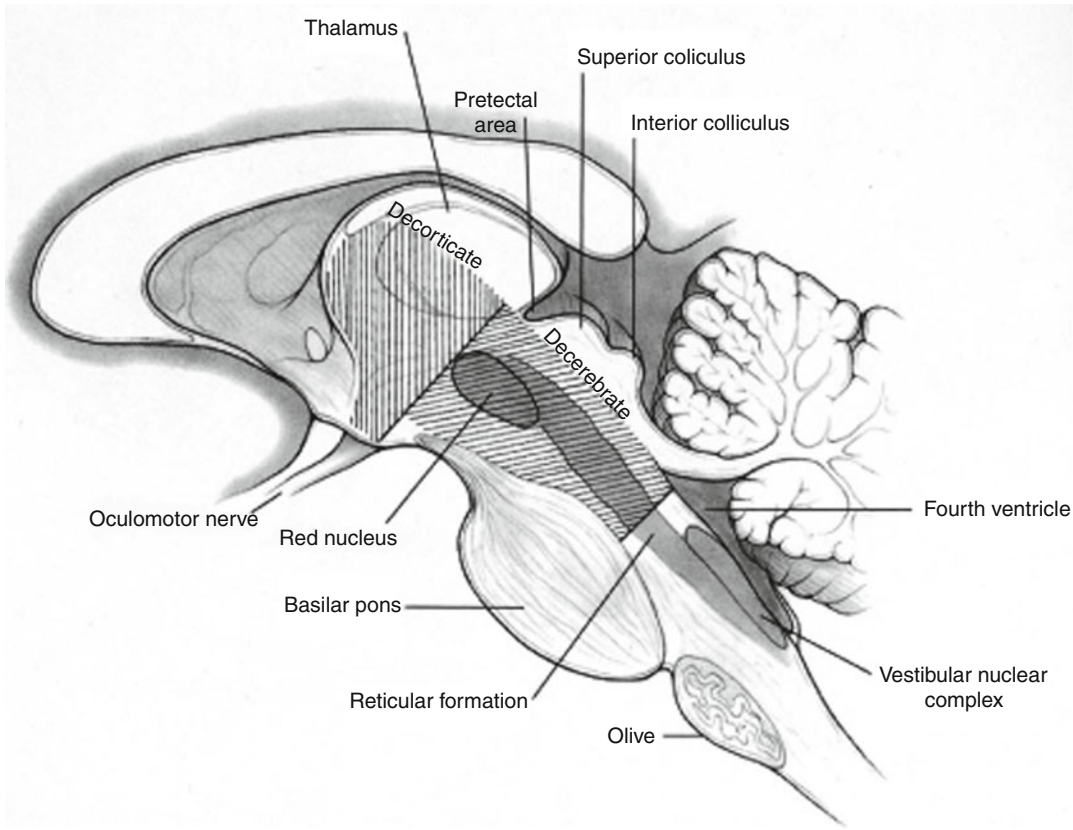

Fig. 7.2 Brainstem compression occurs in a rostral (head) to caudal (toe) progression. Median view of brainstem showing levels of impairment associated with abnormal posturing: Decorticate indicates a more rostral lesion (above red nucleus); decerebrate indicates a more caudal lesion (midbrain or pons) (Reprinted with permission from Young et al. (2008))

assessed to determine the presence and location of nervous system dysfunction in both conscious and unconscious children. Injury can occur to the central nervous system – brain and spinal cord (upper motor neurons) or the peripheral nervous system (lower motor neurons).

With upper motor neuron (UMN) injury, signals (both excitatory and inhibitory) from the cortex are diminished or cut off, causing the spinal cord to become hyperreflexic. Hyperreflexia indicates injury to the CNS corticospinal tract with resultant irritability in the spinal cord. UMN injury is associated with increased tone, spasticity, clonus (muscle spasm with forceful dorsal flexion of the ankle), and a present Babinski. Unilateral hyperreflexia indicates a CNS injury, such as an expanding mass hemorrhage on the opposite side of the brainstem or cerebral cortex, resulting in increased ICP. Injury to the peripheral nervous system (PNS), or lower motor neurons (LMN), is associated with hyporeflexia or areflexia (loss of efferent motor fibers), as well as muscle weakness, flaccidity, and atrophy (Greenberg 2010; Hickey 2009; Young et al. 2008). Hypotonia and atrophy occur due to the loss of LMNs, which innervate muscles and maintain normal tone (Young et al. 2008). Preserved reflexes in a flaccid limb indicate CNS (UMN) injury, not a PNS (LMN) injury.

Babinski sign is present when stroking the plantar surface of the foot, resulting in dorsiflexion of the great toe and fanning of the other toes. This is a primitive reflex seen normally in infants and usually disappears by 10 months of age (range 6–12 months) (Greenberg 2010). Presence of a Babinski sign after age 6 months in TBI is pathologic and indicates injury to the corticospinal tract at any level (Hickey 2009).

7.5.7 Supratentorial Versus Infratentorial Injury

The tentorium cerebelli is a fold of the dura mater, separating the cerebral hemispheres from the cerebellum and brainstem. The "tent" is an important landmark, as assessment for deterioration of neurologic status differs based on whether the injury is above the tentorium (supratentorial) or below the tentorium (infratentorial). The tentorium also contains the tentorial notch through which uncal or brainstem herniation occurs with increased ICP. Impending herniation must be recognized early to prevent brain death. Unilateral supratentorial mass lesions cause uncal herniation, evidenced initially by ipsilateral sluggish pupillary response, progressing to ipsilateral pupillary dilation, contralateral hemiparesis, trochlear and abducens cranial nerve paralysis, and a decreased level of responsiveness with eventual rostral-caudal deterioration. The mass effect causes lateral displacement of the midbrain, forcing the opposite cerebral peduncle against the tentorium, producing Kernohan's notch. This is important to remember when attempting to identify the location of a lesion, as it results in hemiparesis ipsilateral to the expanding mass lesion (Hickey 2009). Bilateral supratentorial lesions, which cause mass effect and increased ICP, progress in a rostral (head) to caudal (tail) progression with impending herniation of the brainstem through the tentorial notch (see Sects. 7.5, 7.5.4, and 7.9.4).

An expanding mass lesion in the posterior fossa (rare in children) results in direct compression on the brainstem and cerebellum, upward herniation, or downward herniation. A primary infratentorial injury affects the reticular activating system (consciousness) directly. Downward compression on the vital cardiac and respiratory centers in the medulla oblongata results in pathologic alteration in vital signs, with respiratory and cardiac arrest (Hickey 2009). More often, posterior fossa lesions in children show progressive deterioration, with or without development of obstructive hydrocephalus, and present with rostral-caudal deterioration or lower cranial nerve deficits. Ataxia (decreased muscle coordination) and dysarthria (discoordinated speech) also occur with cerebellar injury (see Chap. 1).

7.6 Radiographic Imaging in Pediatric Head Trauma

For the purpose of this chapter, traumatic intracranial injuries are discussed individually, but in reality any combination of lesions can and does occur. The non-contrast head computerized tomography (CT) is the initial study of choice in pediatric head trauma. Obtaining CT imaging is fast and allows for ease of monitoring of the unstable child with moderate to severe TBI. CT scan is sensitive to hemorrhage, mass effect, and skull fractures. Magnetic resonance imaging (MRI) is more useful in the subacute or chronic stage of injury and should also be considered if CT scan findings do not fully explain the extent of neurologic deficit. Be aware that CT scans do expose the child to radiation, and order them wisely. See Table 7.5 for a comparison of modalities for neuroimaging. Skull radiographs are minimally useful. When obtained in the presence of scalp swelling or other injury, x-rays can reveal skull fractures or intracranial air, which may indicate more serious intracranial injuries. Ultrasound can be useful in neonates and infants with open fontanels as a screening tool for hemorrhage, although it is limited to allow imaging of the full periphery of the brain. Cerebral ultrasound is useful in identifying the presence of intracerebral hemorrhage (ICH) and intraventricular hemorrhage (IVH), as well as assessment of ventricular size with hydrocephalus. The neuroimaging modality of choice will be discussed in greater detail with each classification of traumatic intracranial injury (Barkovich 2005).

7.7 Types of Traumatic Brain Injury

7.7.1 Birth-Related Traumatic Brain Injury

Traumatic injury to the brain may occur during the birth process. Infants with greater risk for birth-related injuries include those above the 90th percentile for weight. The rate of birth injury is higher in infants weighing more than 3,500 g (Blackburn and Ditzenberger 2007). Birth injuries may also be related to the infant's

Table 7.5 Comparison of neuroimaging modalities in pediatric traumatic brain injury

	X-ray	Ultrasound	CT scan	MRI
Timing	Early, especially if scalp swelling, trauma is present	Useful with open fontanel; portable	Fast, immediate posttrauma imaging	Subacute or chronic imaging
Type of injury identified	Skull fracture	Hemorrhage	Scalp swelling	Nonhemorrhagic contusion
	Pneumocephalus	Ventricular size: (hydrocephalus or small, obliterated ventricles with IICP)	Skull fracture	
	Foreign body	Cranial Doppler for vasospasm secondary to SAH	Pneumocephalus	Diffuse axonal injury
	Split cranial sutures with increased ICP		Extraparenchymal hemorrhage: (EDH, SDH, SAH, IVH)	Early ischemic injury (cerebral infarct)
			Intraparenchymal hemorrhage: (ICH, hemorrhagic contusion)	CT scan does not explain neurologic deficit
			Mass effect: (obliteration of ventricles and cisterns, poor gray-white differentiation, splitting cranial sutures)	Injury dating in child abuse
			Hydrocephalus	MRA (posttraumatic aneurysm)

position during labor and delivery (e.g., breech presentation), as well as cephalopelvic disproportion, where the mother's pelvis size or shape is not adequate for vaginal birth; difficult labor or delivery; prolonged labor; fetal anomalies; and very low birth weight or extremely premature infants. Some of the more common birth injuries to the neonatal head and brain include extracranial hemorrhage (caput succedaneum, subgaleal hemorrhage, or cephalohematoma), skull fracture, and intracranial hemorrhage (epidural, subarachnoid, subdural, or intracerebellar hemorrhage).

Caput succedaneum, a common finding in the newborn, involves soft tissue swelling of the presenting part of the head in a vertex (head first) delivery. The scalp edema consists of serum or blood or both and may have ecchymosis (bruising), petechiae, or purpura. Caput succedaneum may occur after spontaneous delivery due to pressure of the fetal head against the uterine wall, the cervix, or the vaginal wall, or after use of a vacuum extractor. The scalp edema may cross over suture lines and does not continue to increase in size after delivery. It heals in hours to days and rarely has complications. Nursing care involves parent education about the cause of the tissue swelling and/or discoloration (Hernandez and Glass 2005; Sansoucie and Cavaliere 2003).

Cephalohematoma is a subperiosteal collection of blood secondary to the rupture of blood vessels between the skull and the periosteum. It is typically over the parietal bone, and is usually unilateral, but can occur bilaterally. Cephalohematoma is seen most often in male infants after a prolonged, difficult, or forceps-assisted delivery. The characteristic finding is a firm, tense mass that does not cross the suture lines. It may enlarge slightly by 2–3 days of age and takes weeks to months to resolve, occasionally with residual calcification. The calcified

"lump" gradually subsides as bones grow and reshape. Approximately 10–25 % of cephalohematomas have an underlying linear skull fracture (Volpe 2008). Rarely the cephalohematoma may contain enough blood to affect hematocrit and bilirubin levels. Nursing care involves monitoring and parent teaching about hyperbilirubinemia. Anemic infants should also be evaluated for symptoms of intracranial hemorrhage. Generally, there are no long-term sequelae from a cephalohematoma.

Subgaleal hemorrhage is the most serious extracranial hemorrhage in newborns, though it occurs much less frequently than caput succedaneum and cephalohematoma. Blood collects below the epicranial aponeurosis (connective tissue over the brain) and may spread beneath the entire scalp and down the subcutaneous tissue in the neck. There is a strong association between vacuum extraction and subgaleal hemorrhage. The hemorrhage may be from suture diastasis (separation), linear skull fracture, or fragmentation of the superior margin of the parietal bone. Subgaleal hemorrhage presents as a firm fluctuant mass, crosses suture lines, and may increase in size after birth. Blood loss can be significant, up to 260 ml, exceeding the total blood volume of a full-term infant (Reid 2007). This volume loss into the large potential space between the galea aponeurotica and the periosteum of the skull can necessitate urgent blood transfusion and may contribute to hyperbilirubinemia (Blackburn and Ditzenberger 2007; Volpe 2008). Early detection of this clinical emergency is vital. Nursing interventions include serial measurements of head circumference, inspection of the back of the head and neck for increasing edema, and observation of the ears being pushed forward and lateral. The nurse should also monitor for changes in LOC and decrease in hematocrit along with signs of hypovolemic shock (Barkovich 2005; Schierholz and Walker 2010). Parent teaching includes preparing them for the swelling and discoloration of the face, head, and neck. Lesser lesions resolve in 2–3 weeks (Volpe 2008). Moderate to severe lesions may require intensive care, and up to 25 % of these babies may die (Reid 2007).

7.7.2 Neonatal Skull Fracture

Skull fractures, both depressed and linear, are occasionally seen in the newborn. The fetal skull is flexible, malleable, and poorly ossified, when compared to the adult skull, and thus is often able to tolerate mechanical stressors relatively well. Skull fractures can occur in utero, during labor, with forceps delivery (Fig. 7.3), or during a prolonged or difficult labor. The fetal skull can be compressed against the maternal ischial spines, sacral promontory, or symphysis pubis. Cerebral injury should be suspected when neurologic signs are apparent and there is a history of a difficult delivery. Skull x-ray or CT scan is used to confirm the fracture and identify cerebral contusion or hemorrhage. CT is preferred as it identifies space-occupying hematomas and injury to the underlying brain (Fig. 7.3).

Depressed skull fractures (which are not really fractures, but an indentation of bone) are usually seen after forceps delivery, but occasionally are seen after a spontaneous vaginal or cesarean delivery (Parker 2005). A depressed skull fracture is a visible and palpable dent in the skull, usually over the right parietal bone, and does not cross suture lines. There may be no other symptoms unless there is an underlying cerebral contusion or hemorrhage. A depressed skull fracture may be referred to as a "ping-pong" lesion, as it resembles a dent in a ping-pong ball. The uncomplicated depressed fracture can be manually elevated if it does not occur spontaneously in the first few days (Verklan and Lopez 2011). Manual elevation becomes more difficult later on. Methods to elevate the fracture include gentle pressure, use of a breast pump, or use of a vacuum extractor. Surgical intervention is necessary when the depressed fracture cannot be elevated manually, when bone fragments are in the cerebrum, if neurologic deficits exist, or if intracranial pressure is increased. If there is CSF leakage, antibiotics may be prescribed for prophylaxis. Some infants will require treatment for shock and hemorrhage.

Linear skull fractures are usually seen in the frontal and parietal bones and are often associated with extracranial hemorrhage, such as

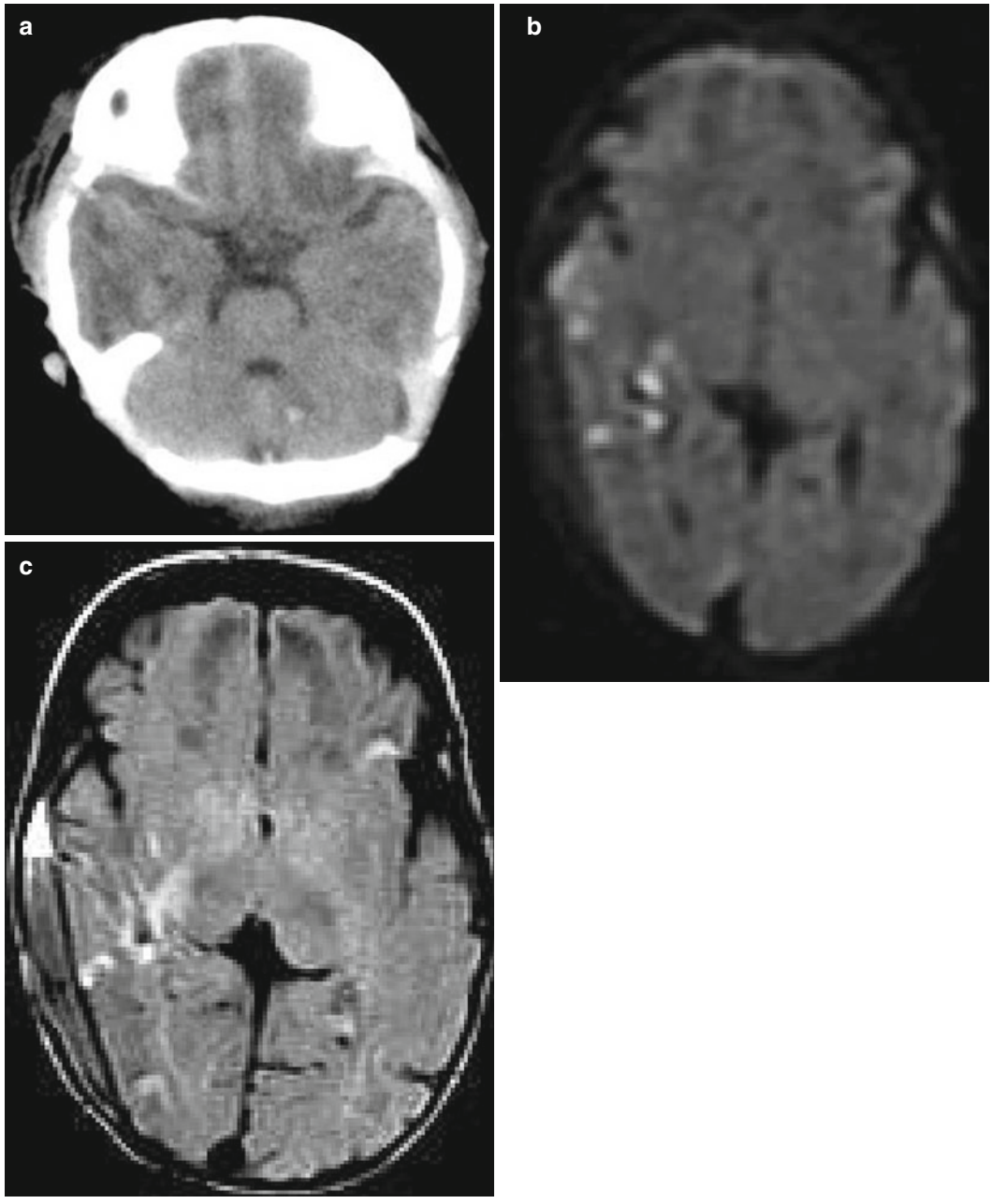

Fig. 7.3 (**a**–**c**) Neonate with significant birth trauma after vaginal delivery with forceps. (**a**) Infant sustained a right parietal depressed skull fracture, scalp swelling (caput succedaneum), bilateral extra-axial hematomas (*SDH* subdural hemorrhage), right temporal and left cerebellar hemorrhage as seen on computed tomography (*CT*) images. Subarachnoid hemorrhage was also seen on the tentorium and at the vertex (not shown). (**b**) Diffusion-weighted magnetic resonance imaging on DOL 7 shows brain contusion with injury in the right temporal lobe and corpus callosum (not shown). (**c**) Fast fluid-attenuated inversion-recovery (*FLAIR*) MRI reveals right temporoparietal SDH and scattered white matter hemorrhage bilaterally (right temporal, bilateral occipital, and left frontal). The infant was treated with observation only; the lesions resolved, and the child did well, without development of hydrocephalus

cephalohematoma. These fractures are diagnosed with skull x-rays and are usually asymptomatic. The exact incidence is unknown as routine x-rays in otherwise healthy newborns are uncommon. Linear fractures are rarely complicated by intracranial hemorrhage (Blackburn 2003). Linear skull fractures in infants may heal in 6 months (Barkovich 2005). They heal spontaneously with no sequelae, unless a dural tear allows the leptomeninges to protrude into the fracture site (i.e., growing fracture of childhood). A cyst may form and grow, causing the fracture to enlarge. Leptomeningeal cyst is rare, occurring in less than 1 % of linear fractures in children under age 3 years (Greenberg 2010). Depressed fractures that are small or treated early have a good prognosis. Larger fractures have a greater risk of sequelae, especially if treatment is delayed. Sequelae are related to the cerebral injury, from either dural hemorrhage or hypoxic event, or both, not from the fracture itself (Blackburn 2003).

Nursing assessment involves supportive care and monitoring of the infant for signs of neurologic dysfunction (increased ICP from hemorrhage, seizures, apnea, and meningitis). Parents may be concerned about brain damage and their infant's appearance if there is a depressed fracture. Parents should be educated to observe their infant for and report signs of increased ICP (irritability, poor feeding, vomiting, hypersomnolence) and growing fracture (growing bulge at fracture site) to the practitioner. They should be instructed to have the fracture site examined at each newborn visit.

7.7.3 Intracranial Hemorrhage

Intracranial hemorrhage may occur in the neonate secondary to trauma or hypoxia in the perinatal period (epidural hemorrhage – EDH, primary subarachnoid hemorrhage – SAH, subdural hemorrhage – SDH, intracerebellar hemorrhage) or due to immature structures and hemodynamics in the premature infant (periventricular/intraventricular hemorrhage – P/IVH), especially those under 32-week gestational age at birth. The pathophysiology of P/IVH involves disruption to the autoregulation of CBF, which is affected by hypoxia and acidosis, leaving the germinal matrix area vulnerable to systemic blood pressure changes. Systemic blood pressure changes may be caused by handling, suctioning, positive-pressure ventilation, hypercapnia, and rapid volume expansion (Blackburn 2003).

7.7.4 Epidural Hemorrhage

EDH is a rare occurrence and may be associated with cephalohematoma. It refers to blood collection above the dura mater and below the periosteum (inner surface of the skull). Most cases are associated with a linear skull fracture. Nearly all affected infants have a history of difficult delivery. Signs of increased ICP, including a bulging fontanel, may be apparent in the first hours of life. An emergent CT scan should be performed. Surgical evacuation may be required. Aspiration of the accompanying cephalohematoma has been reported as a means of reducing the epidural lesion (Smets and Vanhauwaert 2010). Untreated lesions may result in death within 48 h.

Nursing care involves prompt recognition and reporting, timely preparation and transport for CT scan, transfer to the appropriate facility, and preparation for surgery. Postoperative nursing care includes supportive care for oxygenation, ventilation, thermoregulation, fluids and nutrition, pain management, and monitoring of neurologic signs. Parents will need support and teaching to understand their infant's condition and participate in the treatment plan. Complications range from none to permanent neurologic deficits and/or seizures.

7.7.5 Subarachnoid Hemorrhage

Primary SAH is the most common intracranial hemorrhage in the neonate. SAH occurs in full- and preterm infants, but is more common in the premature infant. Primary SAH consists of venous bleeding into the subarachnoid space (arterial bleeding is usually the cause of SAH in

older children and adults.) The usual site in the neonate is over the cerebral convexities, especially in the posterior fossa (Volpe 2008).

Trauma causing increased intravascular pressure and capillary rupture is associated with SAH in the full-term infant. Asphyxia may cause SAH in the premature infant. Risk factors for SAH include birth trauma, prolonged labor, difficult delivery, fetal distress, and perinatal asphyxia.

The most common presentation of SAH is the asymptomatic premature infant with a minor SAH. The SAH is discovered accidentally with a bloody lumbar puncture during a sepsis work-up or cerebral ultrasound to rule out intraventricular hemorrhage. Another presentation of SAH occurs in a full-term or preterm infant who presents with seizures or apnea at 2–3 days of age. Between seizures, the infant appears healthy. Infants with a massive SAH (quite rare) associated with birth trauma and severe asphyxia have a rapid and fatal course (Blackburn 2003; Volpe 2008).

Ultrasonography or CT is useful to confirm the diagnosis of SAH. If the infant has seizures, other causes of seizures must be eliminated. Blood in the CSF on lumbar puncture may be from SAH or from a bloody tap. Rarely a severe, acute SAH may require a craniotomy. Infants with minor or asymptomatic SAH survive and generally have good developmental outcomes. Up to half of infants with symptomatic SAH, with sustained traumatic and hypoxic injury, have neurologic sequelae. Occasionally, SAH results in hydrocephalus due to CSF obstruction at the level of the arachnoid villi. Periodic cerebral ultrasound evaluation for ventricular size may be indicated. Nursing care involves assessment for seizures and other neurologic signs. Parents will need support and teaching about SAH, so they can understand the needs of their infant.

7.7.6 Subdural Hemorrhage

SDH is not unusual after vaginal delivery. Small posterior fossa subdural hematomas are common after uncomplicated vaginal deliveries (Barkovich 2005). The most likely site for hemorrhage is over the cerebral hemispheres. Significant bleeding over the posterior fossa causes compression of the brainstem, as do dural tears near the great vein of Galen. SDH affects full-term infants more often than preterm infants, usually as a result of precipitous, prolonged, or difficult delivery; use of forceps; cephalopelvic disproportion; breech delivery; or large infant (Reichard 2008; Volpe 2008).

Excessive head molding results in stretching of the falx (folds of dura mater that separate the two cerebral hemispheres and the two cerebellar hemispheres) and tentorium (dura mater between the cerebrum and cerebellum), and venous sinuses, with tearing of the vein of Galen or cerebral or cerebellar veins (Lynam and Verklan 2010). As with SAH, SDH diagnosis depends upon the history and presentation of the infant. If seizures are present, other causes must be excluded. SDH can occur along with SAH; cephalohematoma; subgaleal, subconjunctival, and retinal hemorrhages; skull fractures; and brachial plexus and facial palsies. An MRI or CT will help to confirm the diagnosis. Ultrasound is less reliable. Clinical signs are related to the site and severity of the bleeding. There are three patterns of presentation in infants with bleeding over the cerebral hemispheres (Vinchon et al. 2005). The most common presentation is seen in infants with a minor hemorrhage. They are asymptomatic or have minor signs such as irritability and hyperalertness. The second presentation pattern involves seizures in the first 2–3 days of life. The seizures are usually focal, and other neurologic signs may or may not be present, such as hemiparesis, unequal or sluggish pupils, full or tense fontanel, bradycardia, and irregular respirations. The third pattern of presentation is seen in infants who had no or nonspecific signs in the neonatal period, but then present at 4 weeks to 6 months of age with increasing head size as a result of continued hematoma formation, poor feeding, failure to thrive, altered LOC, and, occasionally, with seizures due to chronic subdural effusion (Volpe 2008).

If the posterior fossa SDH is small, there may be no signs for 3–4 days. As the subdural clot enlarges, signs of increased ICP appear and the infant's condition deteriorates. Infants with significant posterior fossa SDH have abnormal neurologic signs from birth, including stupor or

coma, eye deviation, asymmetric pupil size, altered pupillary reaction to light, tachypnea, bradycardia, and opisthotonos (prolonged, sustained posture with leg extension, trunk arching, and variable arm posture, often extended). As the clot enlarges, there is rapid deterioration with signs of shock in minutes to hours. The infant becomes comatose, with fixed, dilated pupils, altered respirations and heart rate, and finally respiratory arrest.

Care is primarily supportive, including oxygenation and perfusion, thermal management, and fluids and nutrition. Surgical evacuation of bleeding over the temporal convexity with increased ICP may be necessary for infants unable to be stabilized neurologically. Massive posterior fossa hemorrhage requires neurosurgical intervention. Infants at risk for SDH should be monitored for 4–6 months for head size, growth, feeding, activity, LOC, and seizure activity. Aside from supportive nursing care, nurses provide parents education about the cause and prognosis for their infant. Referral to early intervention services is recommended at discharge.

Prognosis varies with the size and severity of the hemorrhage. Infants with SDH, who are asymptomatic or have transient seizures in the neonatal period, do well if there is no associated cerebral injury. Minor posterior fossa hemorrhages rarely have clinical significance (Barkovich 2005). Early diagnosis of large posterior fossa hemorrhage with MRI and CT has improved the outcome for those infants. Most infants with massive bleeding over the tentorium or falx cerebri (near the great vein of Galen) die. Those who survive usually have hydrocephalus and neurologic sequelae.

7.7.7 Intracerebellar Hemorrhage

Intracerebellar hemorrhage is more common in preterm than full-term infants. Although rare, it is generally associated with hypoxia in the preterm infant and associated with trauma in the full-term infant.

Intracerebellar hemorrhage may be caused by intravascular factors (vitamin K deficiency, thrombocytopenia), vascular factors (damage due to hypoxia, followed by hypertensive spikes, e.g., from too rapid intravenous colloid infusion), and extravascular factors (mechanical deformation of the occiput during forceps or breech delivery in the full-term infant, compression of the compliant skull during caregiving, or the use of constrictive bands around the head, especially in the preterm infant) (Lynam and Verklan 2010; Volpe 2008). Intracerebellar hemorrhage may be a primary bleed or extension of a hemorrhage into the cerebellum.

Infants with intracerebellar hemorrhage either present critically ill from birth, with apnea, a declining hematocrit, and death within 24–36 h, or present less ill with symptoms developing at up to 2–3 weeks of age. Clinical signs include apnea, bradycardia, hoarse or high-pitched cry, eye deviations, facial paralysis, opisthotonos or intermittent tonic extension of the limbs, seizures, vomiting, hypotonia, diminished or absent Moro reflex, and hydrocephalus (Blackburn 2003; Volpe 2008).

Cranial ultrasound and/or CT scan is used for diagnosis. Lack of echogenicity of the cerebellum may be an important finding (Lynam and Verklan 2010). Intracerebellar hemorrhage is frequently diagnosed at autopsy. Treatment is primarily supportive. Surgery may be indicated, including hematoma evacuation or ventriculoperitoneal shunt for hydrocephalus. Nursing care involves supportive care for the infant and care and comfort for the parents/family, including referral for early intervention services after discharge. Prognosis is poor in preterm infant survivors. Full-term infants have more favorable outcomes, but generally with subsequent neurologic deficits, especially motor and variable involvement of intellect. About half of the infants have hydrocephalus (Volpe 2008).

7.7.8 Pediatric Traumatic Brain Injury

7.7.8.1 Concussion

Historically, concussion was defined as loss of consciousness or amnesia following a blow to the head.

Concussion, as newly defined by the Zurich Consensus Statement on Concussion in Sport, is a complex pathophysiologic process (biochemically

mediated neuronal dysfunction), induced by a direct or indirect traumatic force to the head. Other common features that aid in defining concussion include rapid onset of impaired neurologic function with spontaneous recovery; neuropathologic changes with symptoms reflecting functional rather than structural injury; graded set of symptoms, with or without loss of consciousness; normal neuroimaging; and a sequential course of recovery, with a small number having prolonged post-concussive symptoms (McCrory et al. 2009). Concussion is also frequently referred to as mild traumatic brain injury (MTBI).

The force of a blow to the head is dissipated over the skull and dura mater and then centrifically to the brain. The brain is a fluid medium with limited compliance, making it deformable and susceptible to neuronal injury. Mechanical forces induce depolarization of an action potential and synchronous cortical neurotransmitter release. On a cellular level, potassium gates open, allowing potassium to move out of the cells. The Na-K-ATPase pump attempts to restore potassium into the cells, but with insufficient energy. Anaerobic metabolism ensues with hyperglycolysis and accumulation of lactate. Calcium flows in to the mitochondria, the power house of the cell, leading to further dysfunction. Enzymes are activated causing cellular dysfunction and apoptosis (microtubules in the axon break down leading to axonal swelling and axotomy). Excitatory neurotransmitters (glutamate, NAA, inflammatory cytokines, free radicals, lactic acid, electrolytes) result in disrupted ion transport, leaky cell membranes, and a hypermetabolic state in the brain. The physiologic result of this complex biochemical cascade is global cerebral depression secondary to energy exhaustion, edema, alteration of the blood–brain barrier, mitochondrial dysfunction, neuropraxia (appears structurally normal on imaging, but with obvious functional abnormality), potential cell alteration, and potential cell death.

Concussion produces brief, non-focal clinical findings such as headache, vomiting, and altered memory or level of consciousness. Early and late concussion symptoms are listed in Table 7.6. The majority (80–90 %) of concussions resolve within a short period of time (7–10 days) (McCrory et al. 2009). Generally, 50 % remain symptomatic after 1 week, 25 % after 1 month, and 5–10 % after 3–6 months. Children and adolescents may remain symptomatic for a longer period of time (McCrory et al. 2009). Children are often admitted for symptomatic treatment, especially with associated vomiting which is more common in children after minor TBI and concussion (Ragheb 2008). Treatment is focused on management of symptoms (headache, vomiting) with analgesics, antiemetics, and parenteral fluid administration, as well as monitoring for neurologic deterioration.

Table 7.6 Signs and symptoms of concussion

Symptoms of concussion (early)	Symptoms of concussion (late)
Headache	*Somatic*: Headache, dizziness, blurred vision, balance disturbance
Nausea or vomiting	*Cognitive*: Poor concentration, dementia (cognitive decline, memory dysfunction), impaired judgment
Confusion Amnesia LOC (not a requirement) Vacant stare or expression Delayed verbal or motor response Unable to focus or perform Altered speech Dizziness or vertigo Incoordination	*Psychosocial*: Emotional lability, depression, personality or behavioral change, easily fatigued, insomnia

Concussion is generally associated with normal neuroimaging (Ragheh 2008; Greenberg 2010). Opinion differs on absolute indications to obtain a head CT for MTBI. Indications for imaging suggested in the literature include prolonged loss of consciousness (>1 min), depressed mental status, post-concussive seizures, focal neurologic deficit, persistent or worsening symptoms (headache and vomiting), physical evidence of skull fracture, and high-risk mechanism of injury (McCrory et al. 2009; Greenberg 2010; Kamerling et al. 2003; Simon et al. 2001). Holmes et al. (2011), in a multicenter prospective observational

Table 7.7 Graduated Return to Play Protocol: applied to children 10 years of age and older; 24 h per step, recurrence of symptoms at any stage requires return to previous step after additional 24-h rest period (McCrory et al. 2009)

Rehabilitation stage	Functional exercise at each stage of rehabilitation	Objective
No activity	Complete physical and cognitive rest	Recovery
Light aerobic exercise	Walking, swimming, or stationary cycling (<70 % maximal predicted heart rate (MPHR))	Increase heart rate
Sport-specific exercise	Skating drills in ice hockey, running drills in football (soccer)	Add movement
Noncontact training drills	Progression to more complex training drills with resistance	Exercise, coordination, cognitive
Full-contact practice	After medical clearance, participate in normal training activities	Restore confidence, assess skills
Return to play	Normal game play	

cohort study ($N = 13{,}500$), found that children with blunt head trauma, initial ED GCS of 14 or 15, and normal head CT scan are at very low risk for subsequent positive findings on neuroimaging and extremely low risk of needing neurosurgical intervention. They concluded therefore that hospitalization for children with mild TBI and normal CT scan is generally unnecessary.

The cornerstone of treatment for MTBI or concussion is complete (physical and cognitive) rest until asymptomatic. Complete physical rest means no training, playing, exercise, or exertion with normal daily activities. Cognitive rest means limited scholastic work, television, extensive reading, video games, or text messaging. Most have gradual resolution of symptoms within 7–10 days, although we know that children can remain symptomatic for a longer period. There is debate regarding the length of the required rest period, with suggestions including a 1-week period after completely symptom-free or requirement of a symptom-free period that is the same length as the symptomatic period.

There is no consensus on grading the severity of concussion or guidelines for when an athlete should return to sports participation following concussion. Concussion-grading scales such as the Cantu, Colorado, and American Academy of Neurology (AAN) Guidelines assign severity of concussion based on loss of consciousness. It is now understood that concussion can occur with and without loss of consciousness. Anterograde amnesia may be a more sensitive indicator of severity. Regarding return to play, the most current recommendation is that no child be allowed to return to play on the same day in which the concussion occurs (McCrory et al. 2009). In addition, there is agreement that the child and adolescent athlete be symptom-free with exertion, and without medication, prior to returning to the game (Greenberg 2010; Kelly and Rosenberg 1997; Vagnozzi et al. 2005). A gradual return to play protocol offers a structure in which the athlete advances from no activity through graded levels of activity and then returns to full play (Table 7.7). The stepwise rehabilitation takes place over a 1-week period with advancement to the next step every 24 h. If the athlete becomes symptomatic, then they must return to the previous step and try to progress again after a 24-h rest period (McCrory et al. 2009). This author postulates that similar objectives could also be applied to a stepwise return to normal activity and scholastic activities in the nonathlete and children younger than 10 years of age with concussion. Absolute contraindications to play include symptomatic and permanent CNS sequelae such as developmental delay, visual impairment, and posttraumatic hydrocephalus (Greenberg 2010).

Guidelines for return to play are crucial to prevent more severe injury or sudden death caused by second impact syndrome (SIS). Second impact syndrome is a rare condition that occurs when the individual sustains a second TBI while still in recovery phase from an earlier one. Classically, the athlete walks off the field, collapses into coma, and deteriorates rapidly. Cerebral dysautoregulation occurs with vascular

engorgement, cerebral swelling, and malignant cerebral edema, most often resulting in death (50–100 % mortality) (Greenberg 2010). Adolescents and children are far more susceptible to SIS than adults. It has only been seen in athletes less than 20 years of age. Vagnozzi et al. (2005) utilized a rat model where ATP (adenosine triphosphate) and NAA (N-acetylaspartate) levels were measured following repetitive MTBI at varying intervals. Two consecutive mild TBIs occurring within 3 days produced the same biochemical damage as a severe TBI. After 5 days, however, the two mild TBIs acted as two independent events. The conclusion was that the interval of time between injuries is important because the brain is metabolically vulnerable to repeat injury. Multiple concussions within the same season or similar time period deserve special consideration. Medical evaluation and clearance, with imaging, are recommended after the second and third consecutive concussions in one season. It is also recommended that athletes with three concussions in one season, or abnormality on cerebral imaging, terminate participation for the remainder of the season.

Although most have resolution of symptoms within days to weeks following injury, some have persistent cognitive impairment, behavioral issues, or emotional lability, whether the original symptoms have cleared or not (Table 7.6). This phenomenon is known as post-concussive syndrome. Children who remain symptomatic greater than 3 months benefit from formal neuropsychological testing and often require individualized education plans (IEP), either on a temporary or permanent basis to assist them with school performance. Brain MRI is recommended for symptoms persisting greater than 3 months or if symptoms worsen. EEG should be considered if there is suspicion of seizures. Long-term mental health conditions (such as depression) have been reported following MTBI and concussion. Children and particularly adolescents should be evaluated for depression and referred or treated appropriately. Preexisting conditions (mental health disorder, depression, chronic headaches, ADHD, learning, or sleep disorders) must be considered in management of concussion and in some cases may predict the potential for prolonged or persistent symptoms (McCrory et al. 2009). The psychosocial impact and loss to the child and family can be significant. Education, reassurance, counseling, and a follow-up plan are important to assist families through the recovery period.

Chronic traumatic encephalopathy (CTE) is a neurodegenerative process believed to result from multiple head injuries. It was initially discovered in boxers and more recently has gained recognition with other causes of repeated head trauma (organized sports and repeated abusive head trauma). The pathologic changes include cerebral atrophy, tau-positive inclusion bodies, and neurofibrillary tangles. Disordered memory and executive functioning, as well as behavioral and personality disorders, have been reported. Levels of depression in retired professional athletes, aged 30–39 years, are 19 times higher than the rate of depression in age-controlled counterparts. Multiple suicides in high-profile athletes with diagnosed CTE have brought this condition to the forefront, and additional research is needed to identify measures for prevention, diagnosis, and treatment of CTE (Gavett et al. 2011; Chin et al. 2011).

7.7.8.2 Skull Fractures (Pediatric)

The pediatric skull provides a protective box, which houses the brain. Forces exerted on the skull are absorbed initially in a centrifical configuration and then directed inward toward the brain. Fractures occur when the skull cannot withstand the force of impact. As mentioned previously, the pediatric skull is thinner and more flexible when compared to the adult skull, which predisposes the child to significant traumatic brain injury with or without the presence of a skull fracture. A higher degree of suspicion should be maintained in deciding whether to obtain radiographic imaging based on the reported mechanism of injury (Simon et al. 2001). Twenty percent of children presenting with head trauma will have skull fracture. Of these, most do not require surgical intervention. Initial focus in management of skull fractures is identification of any serious underlying acute hemorrhage or brain injury. While skull fractures are readily visible on skull radiographs as thin, dark lines, CT is preferred

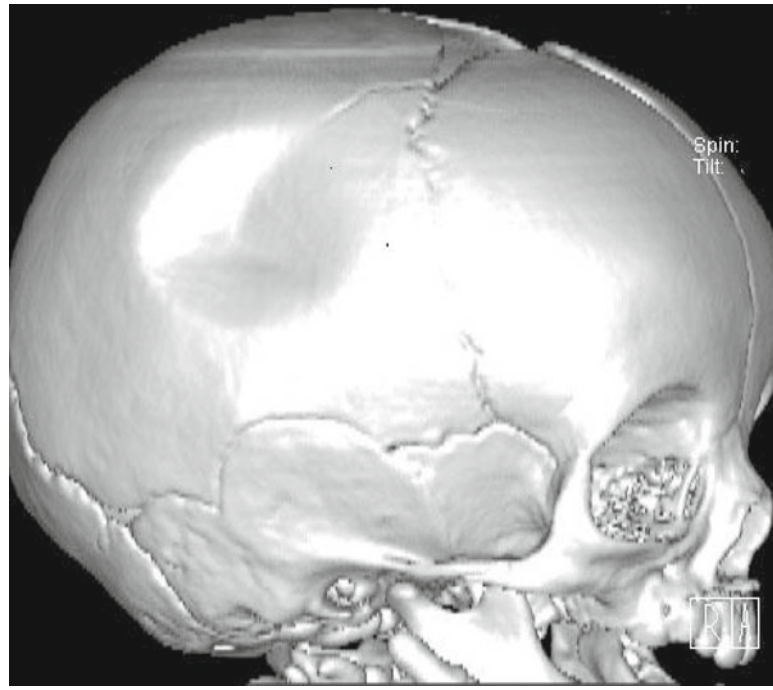

Fig. 7.4 Ping-pong skull fractures occur in newborns and young infants due to the thin, pliable skull and consist of a greenstick fracture and skull depression which resembles a depression in a ping-pong ball

due to the relationship between skull fractures and underlying brain injury in children. Skull fractures are classified by their location (calvarial or basilar), type (linear, closed, open, or depressed), and associated complications (Greenberg 2010; Ragheh 2008).

Linear nondepressed skull fractures comprise 90 % of pediatric skull fractures, occur most frequently in the calvaria (upper portion of the frontal, parietal, and occipital bones), and heal rapidly without intervention (Ragheh 2008). The most common site for linear skull fractures is the parietal bone, which is thinnest of the cranial bones and is most frequently the site of impact in pediatric falls. The frontal and occipital, which are the thickest, require a more severe impact to cause fracture and, therefore, are associated with a higher degree of brain injury. Diastatic fractures occur along a cranial suture line. They are more common in infants and typically result in a widening of the suture.

Young children with a skull fracture and underlying dural laceration may develop a leptomeningeal cyst or growing fracture of childhood. This occurs less than 1 % of the time in children less than 3 years of age. The opening in the dura allows CSF and brain to pulse outwardly into the area of the fracture, preventing healing and causing outward eversion or growth of the fracture margins. A new soft, pulsatile swelling on the scalp is suspicious for a growing skull fracture, requires imaging with a head CT, and may require surgical repair of the dura and cranioplasty (Ragheh 2008).

Most closed (simple), depressed skull fractures, without underlying brain injury, do not require surgery. Nonsurgical management in this case is not associated with increased risk for seizures or neurologic impairment (Ragheh 2008). Due to rapid brain growth, these fractures tend to remodel to a cosmetically pleasing appearance in young children. Closed and open (compound) depressed skull fractures with evidence of dural tear, parenchymal injury, CSF leak, brain extruding through laceration, and focal neurologic deficit require surgical elevation, debridement of pulped brain, evacuation of hemorrhage, and dural repair. Ping-pong skull fractures occur in newborns and young infants due to the thin, pliable skull and consist of a greenstick fracture and skull depression which resembles a depression in a ping-pong ball (Fig. 7.4). Due to rapid skull growth, most ping-pong fractures heal well

without surgery and mold to become cosmetically acceptable. Elevation is required for underlying brain injury, parenchymal bone fragments, and neurologic deficit. Underlying brain contusion secondary to skull fracture can cause posttraumatic seizures.

Basilar skull fractures occur in the anterior, middle, or posterior fossa at the base of the skull. Structures at the skull base are susceptible to injury and include the carotid artery, venous sinus, cranial nerves, and the middle ear (Ragheb 2008). Basilar skull fractures may be difficult to see on CT, but findings of pneumocephalus and opacification of the mastoid air cells are suggestive. Plain films and clinical findings (CSF otorrhea or rhinorrhea, hemotympanum, Battle's sign, raccoon eyes, cranial nerve injuries) are more sensitive (Greenberg 2010). Temporal bone fractures are classified as transverse (extending across the petrous portion) or longitudinal (extending lateral to medial). Complications associated with transverse temporal bone fracture include sensorineural hearing loss (CN VIII) and facial nerve dysfunction (CN VII), whereas longitudinal temporal fracture can cause hemotympanum, torn tympanic membrane, CSF leak, and conductive hearing loss secondary to bony disruption. In most cases, the hearing loss resolves, but it can be permanent. The middle meningeal artery is housed in a groove of the temporal bone. Laceration by the sharp bony edge of the fracture causes serious life-threatening epidural hematoma formation and need for emergent surgical intervention following temporal bone fracture (Greenberg 2010).

The nursing assessment should include inspection and gentle palpation of the scalp to check for findings consistent with a skull fracture. External evidence of skull fracture includes swelling, hematoma, depression of the scalp, laceration with or without fluid leak, or extruding brain. Basilar skull fractures are identified by external clinical findings. Basilar fracture of the temporal bone results in "Battle's sign," which is postauricular ecchymoses and can be associated with CSF leak from the ear (otorrhea). A frontal basilar fracture results in "raccoon eyes," which is periorbital ecchymoses. CSF leak from the nares (rhinorrhea) can result secondary to frontal basilar fracture. The majority of cerebrospinal fluid leaks resolve within 7 days without surgical intervention. Nursing care of the patient with CSF leak includes elevation of the head of the bed, restriction of nose blowing, and reporting of fever or other signs of meningitis. The neurosurgeon may need to place a lumbar drain, so the leak can seal. Check with the neurosurgeon before placement of a nasogastric tube, as a frontal fracture through the cribiform plate can allow placement of the catheter into the brain.

7.7.8.3 Extraparenchymal Hemorrhage

Extraparenchymal or extra-axial hemorrhages are those occurring outside the brain itself and are defined by the location in which they occur in relationship to the meninges. They include epidural, subdural, and subarachnoid hemorrhages. The presentation and acuity level varies based on the child's age, as well as location and size of the hemorrhage. Small hemorrhages with minimal or no clinical deterioration may be observed with close monitoring and radiographic follow-up, whereas hemorrhages with significant mass effect and a deteriorating or comatose patient require emergent craniotomy and surgical decompression (Doppenberg and Ward 2008). There is a lower incidence of mass hemorrhage in infants and children than in adults. The anatomy of the pediatric brain and skull dissipates the impact of traumatic injury in this population. The protective features include the thin deformable skull, wider CSF spaces, and softer brain. These features not only protect children from mass hemorrhage but also enable children to tolerate hematomas better up to 4 years of age, at which time the child's skull is a closed, rigid box likened to that of an adult. Interestingly, the anatomical differences that are protective against mass hemorrhage also predispose the pediatric patient to shearing injuries and subarachnoid hemorrhage.

7.7.8.4 Epidural Hemorrhage

Epidural hematomas (EDH) are less common in infancy and steadily increase in incidence with age. Outcome following EDH is improved in

Table 7.8 Surgical or nonsurgical management of epidural hematomas (Doppenberg and Ward 2008)

Surgical indications	Nonsurgical management of EDH
Focal neurologic exam, third nerve palsy, increasing drowsiness	Neurologically intact, with only headache, nausea, vomiting, and irritability (sixth nerve palsy without posterior fossa clot may be excepted)
Focal, significant cortical compression, seen with clots >15 mm diameter	Clot in frontal, parietal, or occipital region
Brainstem herniation	Small posterior fossa clot without compression of cortex, fourth ventricle, or brainstem
Epidural clot volume >30 cc	
Midline shift or uncal herniation, with temporal lobe clot	
Concomitant intraparenchymal or subdural hematoma with mass effect	
Associated fracture transversing major dural vessel with neurologic impairment	

children when compared to adults. Epidural hematomas can be either venous or arterial in origin. The frontal, temporal, and parietal regions are typical locations for EDH. In neonates and infants, EDH is usually venous, secondary to tearing of the dural veins (see Sect. 7.7.1). More commonly, especially in older children, EDH results from a tear of the middle meningeal artery, which is housed within a groove of the temporal and parietal bones. This artery is lacerated by the sharp bone edge when the skull fracture is sustained. Infratentorial (or posterior fossa) epidural hemorrhages occur, but are less common in older children (Doppenberg and Ward 2008). The posterior fossa is a common location for an EDH due to the presence of the dural venous sinuses, and EDH is usually associated with fracture. Epidural hematoma in the posterior fossa is more dangerous due to the smaller anatomic space and potential for direct mass effect on the brainstem, and surgery is recommended (Greenberg 2010).

Regardless of the origin, the bleeding creates a space between the dura mater and the periosteum (or inside) of the skull. Clinical presentation of EDH in pediatrics can be delayed due to the plasticity of the child's skull. The hallmark "lucid interval" following EDH may differ in that the child only appears stunned, while the older child or adult will have loss of consciousness. The volume of a rapidly expanding mass lesion (or hemorrhage), however, is not well tolerated even in the more plastic pediatric cranium. The increased intracranial volume results in general increased intracranial pressure. Nursing care and assessment are dependent on the location of the hemorrhage, being either supratentorial or infratentorial, (above or below the tentorium cerebelli), as the clinical presentation and indications of neurologic deterioration with expanding mass lesions vary based on this important landmark (see Sect. 7.5.7). Indications for surgical versus nonsurgical management of EDH are presented in Table 7.8.

Radiographic evaluation of EDH is best accomplished with a CT scan, which reveals a lentiform, hyperdense (bright white), extraparenchymal fluid collection that is contained within the cranial suture lines (Fig. 7.5). The blood is contained within the sutures because of the attachment of the dura to the periosteum. Epidural hematomas are often also associated with CT scan findings of scalp swelling and the presence of a skull fracture in the frontal, temporal, or parietal regions (Barkovich 2005). Common practice is to repeat the radiographic imaging within 6 h for small, nonsurgical EDH.

7.7.8.5 Subdural Hemorrhage

The incidence of subdural hematomas (SDH) is opposite that of EDH in that they are more common in infants and less common in older children. There is more often underlying brain injury associated with SDH than with EDH. Common etiologies for SDH in children include birth trauma, accidental falls, and child abuse. This type of hemorrhage is due to stretching and tearing of the bridging veins in the subdural space or hemorrhage around severe primary

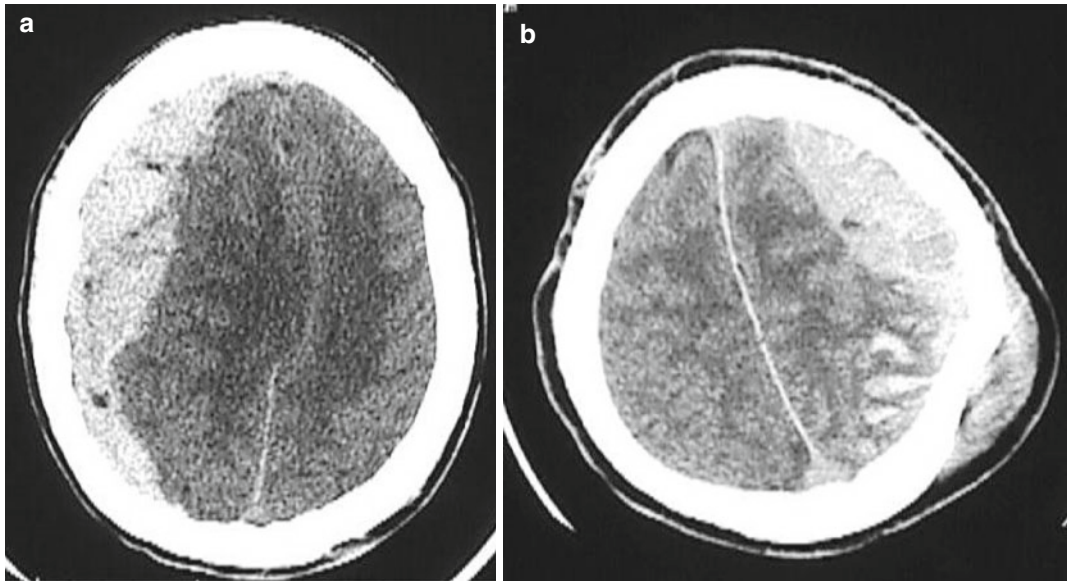

Fig. 7.5 (**a**, **b**) Extraparenchymal hemorrhage. (**a**) Subdural hematoma shown on CT scan as an acute, crescent-shaped blood collection that crosses suture lines. (**b**) Epidural hematoma seen on CT scan as a hyperdense, lentiform collection, contained within the suture lines. Also note significant scalp swelling

brain injury with laceration (Greenberg 2010). The subdural space is located below the dura mater and above the arachnoid membrane. Hemorrhage in this space is not limited to the suture lines and, therefore, can result in large, bilateral blood collections over the entire convexity (Fig. 7.5). Most occur over the convexities but may also be interhemispheric, along the tentorium, or in the posterior fossa (Greenberg 2010; Ragheh 2008).

Large expanding mass lesions of subdural origin significantly increase intracranial volume and thereby intracranial pressure. Infants with SDH present with seizures, irritability, lethargy, vomiting, and increased head circumference. Older children have decreased level of consciousness, pupil asymmetry, and hemiparesis. Severe increased intracranial pressure results in an irregular respiratory pattern, hypertension, and bradycardia, also known as "Cushing's triad." (See Sect. 7.10 Presenting symptoms may be due to the mass effect exerted by the subdural hemorrhage or by the underlying brain injury and possibly cerebral edema. Expansile subdural collections require emergent craniotomy and evacuation to prevent herniation and death. Needle aspirations can be performed in infants with an open fontanel to temporarily relieve pressure. In contrast, a small SDH in a child with minimal neurologic deficits can be observed closely with follow-up imaging within 8–12 h.

Presence of blood in the subdural space is well visualized on CT scan. Subdural hematomas appear as an acute, crescent-shaped, extraparenchymal blood collection, which crosses the suture lines (Fig. 7.6). Severity of a SDH is based on the size, location, and presence of mass effect. Mass effect with any intracranial lesion is manifested as a right or left shift of the cerebral hemispheres (away from the lesion), effacement (compression) of the ventricles, displacement of the brainstem, and obliteration of the sulcal pattern and is indicative of increased ICP. There may also be underlying brain injury, which is less visible until the clot is surgically decompressed. MRI can be useful to determine the timing (acute versus chronic) of subdural hemorrhages, which can be helpful in an investigation for child abuse (see Sect. 7.7.11) (Barkovich 2005).

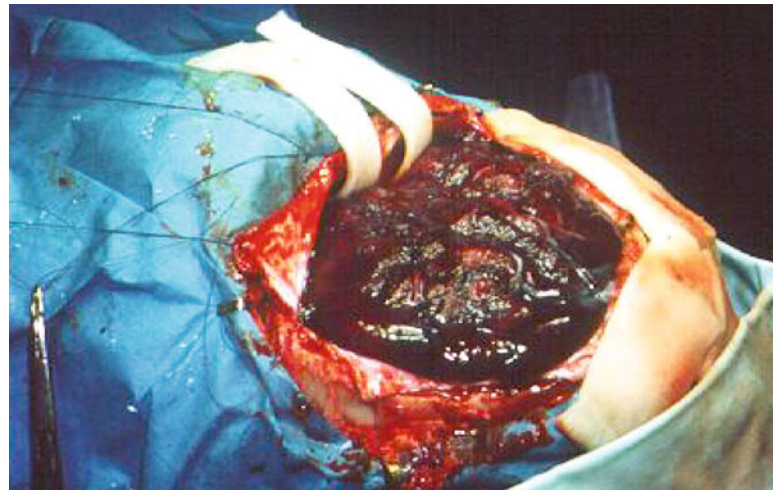

Fig. 7.6 Intraoperative photograph demonstrating a large open craniotomy after severe head trauma. Note the large SDH clot on the surface of the brain

7.7.8.6 Subarachnoid Hemorrhage

Subarachnoid hemorrhage (SAH) is common in children with significant head trauma due to the large, vascular subarachnoid space and the soft calvarium (Doppenberg and Ward 2008). Intraparenchymal injury is often associated with the presence of hemorrhage in the subarachnoid space (Barkovich 2005). The subarachnoid space is located between the arachnoid and the pia mater, which is a thin membrane that is adhered to the brain's surface. Cerebrospinal fluid (CSF) is made primarily in the ventricles and then circulates around the brain within the subarachnoid space. Circulation of CSF allows delivery of metabolic substrates, cushions the brain from trauma, and removes waste products. On CT scan images, subarachnoid hemorrhage (SAH) appears bright white and is seen within the gyral and sulcal pattern on the brain's surface, in the interhemispheric fissure, along the tentorium, and within the cisterns. Clearance of subarachnoid blood is fairly rapid as the blood is "washed out" with the circulation of CSF. MRI is not useful to identify SAH, but should be considered in a child with SAH and severe neurologic deficits. MRI can be useful to identify nonhemorrhagic intraparenchymal lesions, which are often associated with SAH.

The child with SAH should be observed for irritability, headache, stiff neck, and other signs of irritation to the meninges, similar to the presentation of meningitis. Children can develop posttraumatic hydrocephalus, especially when SAH or intraventricular hemorrhage (IVH) is present. The presentation of hydrocephalus is identical to that of increased intracranial pressure. Development of hydrocephalus can be seen on CT scan and, when present, requires CSF diversion to prevent increased ICP and possible herniation. Temporary CSF diversion techniques include drainage via a ventriculostomy, and transfontanel tap in an infant with an open fontanel. Permanent CSF diversion requires placement of a shunt, consisting of a ventricular catheter, a one-way pressure-regulated valve, and a distal catheter to divert CSF to the peritoneum, pleural space, or right atrium.

7.7.8.7 Parenchymal Injury

Following severe head trauma, children have generalized brain swelling. This is thought to occur due to edema and a process of dysautoregulation, whereby the cerebrovascular resistance decreases, resulting in vasodilation and increased cerebral blood volume. Neuroimaging with CT scan completed 12 h after the injury often appears normal, whereas repeat imaging at 24 h post injury reveals poor gray-white matter differentiation with compressed or absent ventricles, cisterns, and sulcal pattern (Barkovich 2005) (Fig. 7.7).

Mass effect following parenchymal injury can be compartmentalized or global. Mass effect from hemorrhage and surrounding cerebral edema,

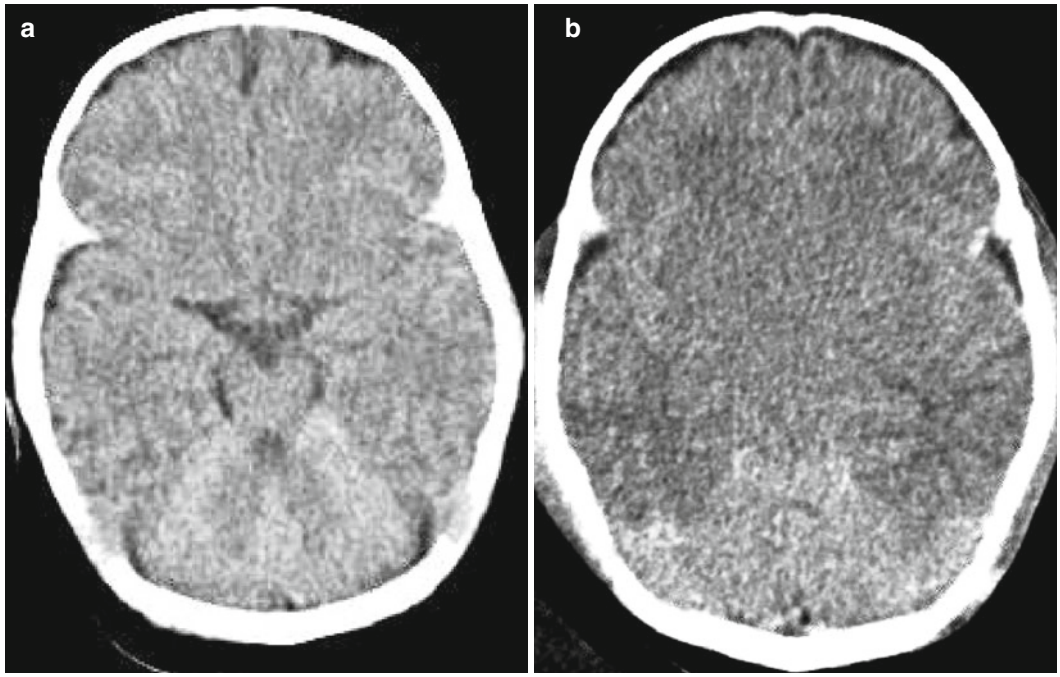

Fig. 7.7 (**a**) Severe traumatic brain injury (*TBI*). Early CT scan without evidence of injury, cisterns, and normal extra-axial fluid is maintained. (**b**) Follow-up CT scan on day 2 showing what is referred to as a "tight" brain, with generalized edema, poor gray-white matter differentiation, and obliteration of the ventricles, cisterns, and sulcal pattern

when confined to the temporal (middle) cranial fossa, is concerning and can lead to transtentorial (or uncal) herniation. Surgical intervention becomes necessary to prevent impending uncal herniation or other neurologic deterioration. The child with an expanding contusion or mass lesion on the right side and pending uncal herniation will present with a right unilateral dilated pupil and contralateral motor weakness, hyperreflexia, present Babinski, clonus, or posturing. The goal of neurosurgical intervention is to remove the hemorrhage, as well as the injured portion of the brain, to decompress the compartment and reduce related volume and, therefore, pressure (Greenberg 2010). It is paramount for the nurse to carry out interventions to prevent secondary injury (fluid overload, hyponatremia, hypercapnia, hypotension) and prevent life-threatening edema or herniation. Any deterioration in the child's level of consciousness indicates increasing ICP and should be reported to the physician immediately and documented (see ICP Management section).

7.7.8.8 Contusion

A contusion is a focal bruise (nonhemorrhagic or hemorrhagic) to the surface of the brain, which, based on size and location, can cause neurologic deficits, seizures, local mass effect, and increased intracranial pressure resulting in herniation. Contusion occurs when the skull impacts a stationary object with sudden deceleration, causing the brain to collide with the bony prominences of the frontal, temporal, or occipital skull. The point of initial impact of the brain on the internal skull is referred to as the "coup" injury. The brain, being suspended in fluid, then strikes the opposite side of the skull, which can cause a "contrecoup" injury. The French meaning of the word contrecoup is "counter blow" (Greenberg 2010). Contrecoup injuries are less common in young children, occurring with a frequency of less than 10 % in ages 0–3 years, and 25 % in ages 3–4 years.

Nonhemorrhagic contusions appear as an area of low attenuation on head CT representing

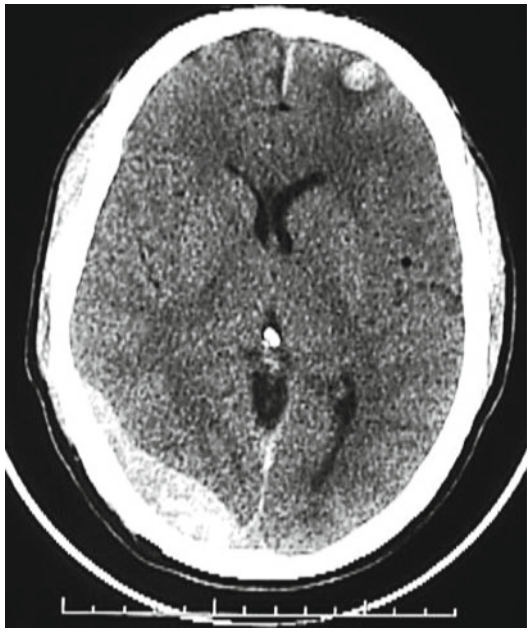

Fig. 7.8 CT scan demonstrating a typical surface contusion in the left frontal lobe, which was likely a contrecoup injury. Note right posterior SDH. The ventricles are asymmetric secondary to the mass effect of the SDH

associated edema, whereas hemorrhagic contusions appear as areas of high attenuation (Fig. 7.8) (Greenberg 2010). The CT may also reveal an accompanying extra-axial hemorrhage or intracranial air, also known as pneumocephalus. MRI is more sensitive to identify nonhemorrhagic contusions (Barkovich 2005). It is the hallmark for contusions to enlarge subacutely and, therefore, require repeat CT imaging within 12–24 h of the injury (Dias 2004; Greenberg 2010). Close observation in an intensive care unit and repeat imaging is required, but contusions typically coalesce and resolve without surgical intervention. Surgical removal is required for neurologic deterioration. (See Sect. 7.10.) Following resolution of a contusion, encephalomalacia (or "dropout") of the brain can occur, with the space then being filled with cerebrospinal fluid (Barkovich 2005).

The child with a focal contusion in an eloquent area of brain, such as speech or motor centers, will likely have worsening of their neurologic function specific to the area of injury as the contusion enlarges. Nurses should be aware of the location of injury and anticipate what deficits may develop. Any change in neurologic function should be reported to the physician immediately. The family should also be warned that the deterioration may occur.

Posttraumatic seizures (PTS) are much more common in young children than in adults (Kochanek et al. 2012; Dias 2004; Carney et al. 2003; Holmes et al. 2004) and occur in approximately 10 % of children following head trauma. The majority of PTS are considered early, occurring within 1 week. Fifty percent occur in the first 24 h post injury (Doppenberg and Ward 2008; Ragheh 2008) and are referred to as *impact* seizures. Studies have indicated that children who experienced PTS after blunt head trauma, with a non-focal neurologic exam and a negative CT scan, can safely be discharged to home. Holmes et al. (2004) prospectively observed a cohort study of 63 children under 18 years with blunt head trauma. Head CT was obtained on all of the children. Ten children had findings on CT scan and were admitted to the hospital, three underwent craniotomy, and two had further seizures. The remaining 52 with negative CT imaging were either observed in the hospital or discharged to home. Follow-up revealed that none of the 52 patients with normal CT scan results had further seizures or required neurosurgical intervention.

Seizures result in increased cerebral metabolism and increased ICP and can cause secondary brain injury. The Guidelines from the Society of Pediatric Critical Care Medicine (Kochanek et al. 2012) recommend prophylactic anticonvulsant therapy with phenytoin as a consideration to reduce the incidence of early posttraumatic seizures in pediatric patients with severe TBI. No data has shown that use of anticonvulsants for early PTS in severe TBI reduces the long-term risk of PTS or improves long-term neurologic outcome. Risk factors associated with the occurrence of PTS include location of the lesion, cerebral contusions, retained bone and metal fragments, depressed skull fracture, focal neurologic deficits, loss of consciousness, Glasgow Coma Scale (GCS) score <10, severity of injury, length of posttraumatic amnesia, subdural or epidural hematoma, penetrating injury, chronic alcoholism, and

age. Prolonged anticonvulsant therapy for 6–12 months may be required (Greenberg 2010; Doppenberg and Ward 2008; Dias 2004).

7.7.9 Diffuse Axonal Injury

Decreased level of consciousness and generalized increased ICP are more likely with diffuse axonal injury (DAI). DAI occurs when the pediatric skull is subjected to rotational forces during high-velocity acceleration or deceleration injuries. The mechanisms of DAI in children vary by age, with older children and adolescents involved in motor vehicle accidents (MVA) and bicycles versus MVA. Younger children are often pedestrians versus MVA. DAI is rare in infants. The softer, more plastic pediatric brain moves within the skull. This movement is further facilitated by the wider subarachnoid space found in children. Neuronal injury occurs in the softer, unmyelinated pediatric brain, when the axons are stretched until there is sufficient strain to cause the axons to fracture (Barkovich 2005). This tearing may be associated with tearing of blood vessels and tiny petechial hemorrhages (Hickey 2009).

DAI typically occurs at the junctions of gray and white matter, the corpus callosum, the internal capsule, the basal ganglia, and the brainstem. The hallmark presentation for DAI is immediate loss of consciousness (LOC) that often lasts greater than 6 h. Abnormal flexion (decorticate) posturing or extension (decerebrate) posturing can accompany LOC, as well as a variation in the GCS score on serial assessments. Children may also have pupillary and other cranial nerve dysfunction and brainstem abnormalities, which will be reflected in assessment findings. The typical triad of pediatric DAI is hypertension, hyperhidrosis (perspiration), and brainstem abnormalities.

Computerized tomography (CT) is not sensitive enough to diagnose DAI. Magnetic resonance imaging (MRI) is most sensitive to shearing injury and should be considered if the CT scan does not reveal injury sufficient to explain the degree of neurologic deficit (Fig. 7.9). T2 and gradient echo-weighted MRI sequences are very sensitive to DAI.

There is no definitive treatment for DAI other than supportive care. Children with severe TBI (GCS 3–8) should be monitored and treated for increased intracranial pressure. The presentation and management of increased ICP are discussed elsewhere in this chapter. Pure DAI is associated with mild increased ICP (Ragheh 2008). Greatly increased ICP in children with DAI requires repeat imaging with CT scan for suspected expanding mass lesion (Barkovich 2005). Recovery from DAI is a slow, gradual, and often incomplete process and can continue for weeks or months following DAI (Ragheh 2008). Outcomes depending on the severity of DAI can vary from cognitive and neurologic deficits to severe disability and death (Hickey 2009).

7.7.10 Penetrating Craniocerebral Injury

Penetrating craniocerebral injuries (PCI) are less common but more often fatal in children. Poor prognostic factors associated with PCI include presentation in coma and the penetrating object crossing midline through the center of the brain or through the ventricles (Greenberg 2010). Concomitant anoxia results in a grave prognosis. Child survivors of PCI often have compromised quality of life. Causes of PCI include accidental impalement with a random sharp object or firearm injury, suicide, and homicide (Krieger et al. 2008). According to Krieger et al. (2008), two key factors with impalements are penetration through the thin calvarium with high risk for vascular and cranial nerve injuries and infection risk with the foreign body (Fig. 7.10). Gunshot wounds are classified by the caliber or velocity at which the missile (bullet) enters the brain. Low-velocity injuries occur with most handguns and cause tissue injury along a path slightly wider than the bullet. High-velocity PCI occurs with military weapons and hunting rifles and causes additional damage due to a shock wave which pushes the brain tissue away from the bullet. Secondary brain injury occurs with cerebral edema,

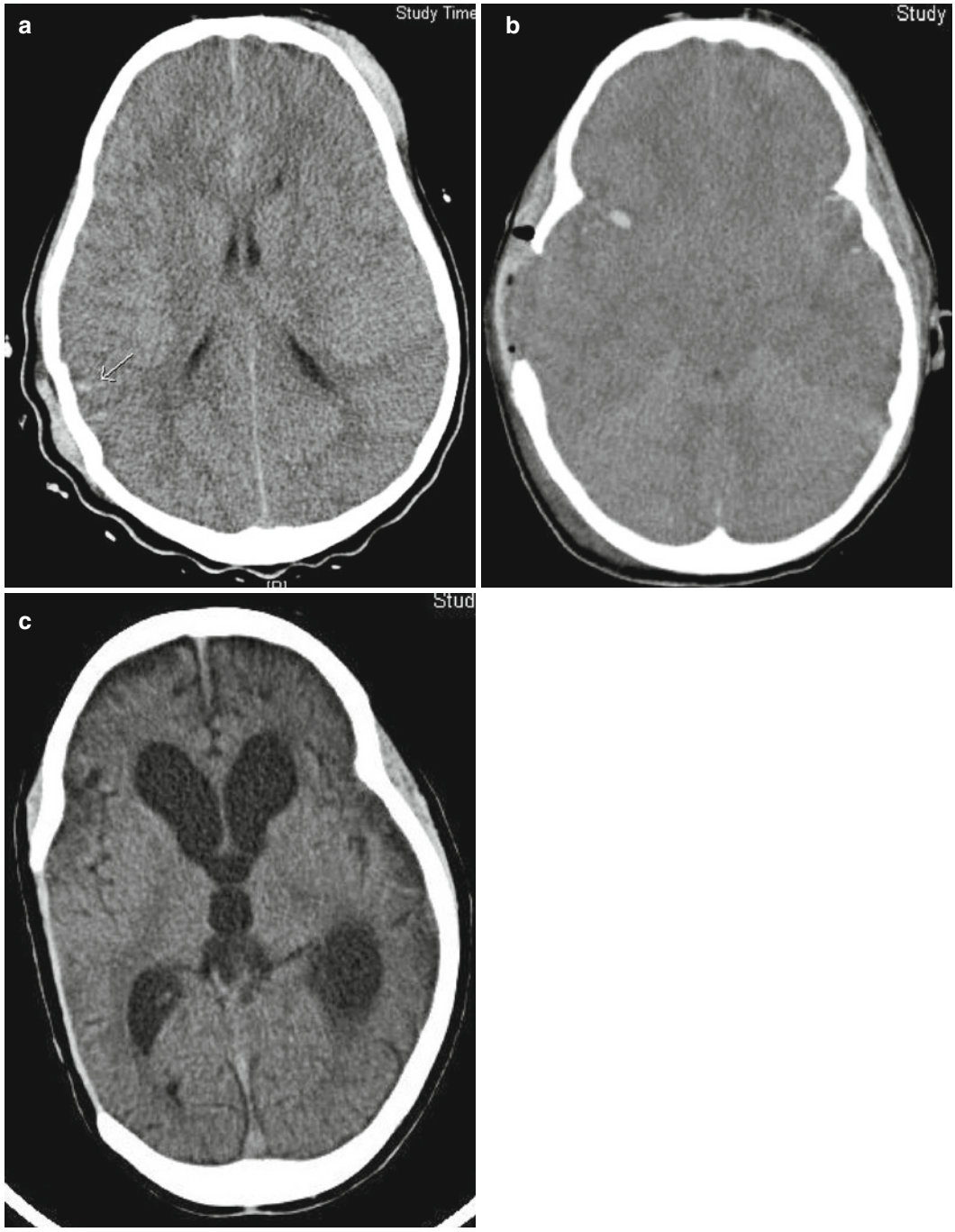

Fig. 7.9 (**a**–**c**) Diffuse axonal injury (*DAI*). This 5-year-old was struck by a van, sustaining severe closed head injury. (**a**) CT scan on the day of the injury shows a depressed right parietal skull fracture, with overlying soft tissue swelling. The child also sustained right temporal and orbital fractures. Multiple punctuate hemorrhages consistent with DAI are seen adjacent to the fracture (*arrow*). (**b**) An intracranial pressure (*ICP*) monitor was placed. Due to uncontrollable elevated ICP, an emergent decompressive craniectomy was performed (see absent bone flap). Multiple small parenchymal hemorrhages (DAI) at the gray-white matter junction can be seen on the CT scan. Also note that the brain is "tight" with poor gray-white matter differentiation and complete effacement of the ventricles and sulci. (**c**) Cerebral atrophy and ventriculomegaly are seen on the CT scan performed 6 months after injury. The child survived his injury but is functionally wheelchair bound with severe morbidity. Films compare the CT scan, where the DAI is not apparent, and MRI performed due to neurologic impairment, which was not explained on the CT scan

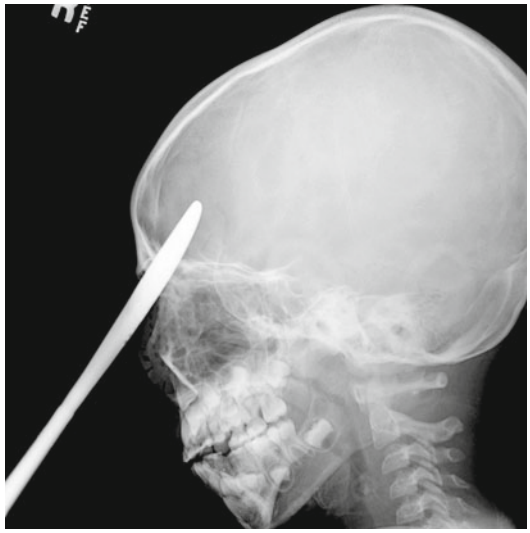

Fig. 7.10 Skull x-ray reveals a penetrating cerebral injury, after a 3-year-old fell and was impaled onto a butter knife

low cardiac output, DIC, and intracranial hemorrhage (Greenberg 2010).

Early aggressive hemodynamic stabilization and ICP management are vital. (See Sect. 7.10) Head CT will reveal the gamut of intracranial injuries as well as help distinguish the entry (small, in-driven fragments and beveled edges) and exit wounds (larger and irregular). Skull films may be helpful to identify foreign objects, fractures, and skull or bullet fragments. In impalement, the protruding object must be secured and left in place by the nurse until evaluation and surgical removal by the neurosurgeon. Surgical intervention is recommended for patients who are considered salvageable. Patients with post-resuscitative GCS 3–5 and fixed pupils have mortality rates greater than 80 % and very poor outcomes. Goals of surgery include:

- Removal of foreign object (prevent infection, seizures, aneurysm)
- Removal of necrotic brain (prevent hemorrhage, edema, scar)
- Elimination of mass effect
- Evacuation of hematomas
- Repair of vascular injury
- Closure of dura and scalp
- Placement of ICP monitor (Krieger et al. 2008)

Complications of PCI include disseminated intravascular coagulation (DIC), infection (with or without abscess), and seizures (Krieger et al. 2008). Disseminated intravascular coagulation is an abnormal coagulation/thrombolytic cascade secondary to thrombin release from injured brain and leads to uncontrolled cerebral hemorrhage. Treatment includes replacement of clotting factors such as fresh frozen plasma, cryoprecipitate, and platelets. Infection is common after PCI, especially in low-velocity GSW and impalement, where the rate of infection approaches 43 %. Prophylactic antibiotics are most often used. Repeat cerebral imaging is important to assess for abscess, in the face of fever, new focal neurologic deficits, and change in mental status. Incidence of posttraumatic seizures following PCI has been found to be as high as 50 % (Greenberg 2010); therefore, treatment with antiseizure medications is recommended (see Sect. 7.7.8.8). MRI is contraindicated if none or a portion of the foreign body can be removed (i.e., bullet fragment).

7.7.11 Abusive Head Trauma

7.7.11.1 Introduction

In 2009, child maltreatment (including abuse and neglect) resulted in 1,770 deaths or 2.34/100,000 children in the United States. Abusive head trauma (AHT) is the leading cause of death from child abuse and the most common cause of severe TBI in infants (Barlow et al. 2001; Keenan et al. 2003; Duhaime 2008). Data from the Kids' Inpatient Database, including coding in hospital discharge databases, was examined to estimate the rate of AHT in the United States at 32.2 cases per 100,000 infants per year (Ellingson et al. 2008). Risk factors for inflicted injury include young parents, low socioeconomic status, single parents, prematurity, and substance abuse (Duhaime 2008). Perpetrators include fathers (37 %), boyfriends (20.5 %), female babysitters (17.3 %), and mothers (12.6 %) (Starling et al. 1995). Retrospective studies have shown that children with AHT are frequently misdiagnosed, resulting in reinjury and deaths (Jenny et al. 1999).

AHT is also known as non-accidental trauma. Common clinical findings include scalp or skull injury, subdural hematoma (SDH), retinal hemorrhage (RH), and other fractures or injuries. The acute and long-term clinical outcome of AHT ranges from mild to severe, with a varied spectrum from full recovery to death or permanent disability. The neuroscience nurse must be knowledgeable of, and suspicious for, indicators of AHT. As healthcare professionals, nurses are mandated to report any suspicion of child maltreatment. A multidisciplinary plan for treatment and investigation is paramount in treating the child with AHT.

7.7.11.2 Pathophysiology

Pathophysiology of AHT is complex and may involve more than one occurrence or more than one mechanism of injury (direct trauma, shaking with or without impact, strangulation). The mechanism of the shaken shaken-impact syndrome is well described, including violent shaking, which is often associated with an impact causing sudden deceleration of the head and brain. Shaking an infant is believed to cause an angular deceleration of the child's head sufficient to cause tearing of subdural veins and hemorrhage (Caffey 1974). Injury severity and outcomes worsen when the shaking injury is further compounded by an acute life-threatening event, seizure, or unresponsive presentation, with apnea or hypoxia (Duhaime 2008). Other common injuries associated with AHT include retinal hemorrhages, acute or healing skeletal trauma, external signs of trauma, and signs of physical neglect.

Acute subdural hemorrhage is the most common head injury seen with AHT and typically is extensive collection over the convexities as well as in the posterior hemispheric fissure (Duhaime 2008; Vinchon et al. 2005). Vinchon et al. (2005) found that SDH was present in 81 % of child abuse cases, with AHT accounting for 64 % of all traumatic SDH in infants. Subdurals with mixed density on CT imaging are more frequent in cases of AHT, whereas homogeneous hyperdense subdural is more frequent with accidental head trauma (Tung et al. 2006). Hemorrhages of different density (age) indicate repetitive AHT. MRI is a more sensitive tool to estimate the age of the hemorrhages and to assess for presence of membranes, indicative of chronic subdurals (Barkovich 2005). Axonal injuries are now believed to be due to diffuse hypoxia and edema (Barkovich 2005). Treatment of subdurals and other types of traumatic brain injuries is the same as for accidental mechanisms and was discussed earlier in this chapter.

Retinal hemorrhage (RH) occurs in both accidental and AHT. Vinchon et al. (2005) found that severe RH (grade 2 or 3) was 100 % specific for the diagnosis of child abuse. It is requisite that grading of RH be determined by a trained ophthalmologist to assure a correct diagnosis and prevent misdiagnosis. Skeletal survey is necessary to assess for skeletal fractures. Skull fractures, spiral fractures of the long bones, metaphyseal fractures, and posterior rib fractures are common fractures associated with child abuse.

7.7.11.3 Clinical Presentation

Most infants present with nonspecific clinical findings and without external signs of trauma. Vague presenting symptoms include poor feeding, vomiting, irritability, lethargy, seizure, apnea and unresponsiveness, external ecchymosis, or other marks indicating trauma. Pallor, tachycardia, or poor perfusion may indicate anemia secondary to intracranial hemorrhage. Children may present with minimal or no external signs of AHT, despite significant neurologic sequelae.

The true history of the injury is withheld (Duhaime 2008), leaving the medical team to piece important history and a constellation of clinical findings together to make a diagnosis of AHT. Little or no history of trauma may be given. Or the reported trauma may not match the severity of the child's injury. It is essential to obtain a detailed history of the child's condition from the parent or guardian. The family should be made aware of the need to find out what happened to the child for the purpose of managing the injuries and anticipating complications. The interviewer needs to document a specific account of exactly what happened and the time that it occurred. Determine who was responsible for the child's

care at or around those times. Inquire specifically about any history of trauma, which may have caused the injuries, and if there were witnesses. Inconsistencies in the history between caregivers, or from the same caregiver on separate occasions, are concerning for AHT (Hettler and Greenes 2003). Multidisciplinary team members (i.e., child abuse physician, social worker, children's service board, and law enforcement) will interview the family as part of a detailed investigation for AHT. Input from other physicians, the neurosurgeon, the ophthalmologist, the radiologist, and the nurse is critical to the investigation.

7.7.11.4 Management

Medical care begins with the EMT/paramedics arrival at the scene or upon the child's arrival to the ED. It is important to stabilize the patient's cardiorespiratory status rapidly to prevent further injury. Once the ED physician has suspicion of AHT, the remainder of the multidisciplinary team should be involved. The neurosurgeon must evaluate the child to determine if urgent surgical intervention is warranted.

7.7.11.5 Case Study

A 5-month-old female was brought to ED by EMS for decreased responsiveness. The mother stated the child fell from a changing table onto a hardwood floor a week ago, cried, and vomited. The mother allegedly called 911 for advice. The baby was not brought in for evaluation. She had continued vomiting, fever, and diarrhea and was seen and diagnosed by the PCP with "stomach flu." The infant is now brought to ED by squad for parental concern of decreased responsiveness. The mother denies trauma, other than the fall the week before, but did state that the sibling (age 4) was "rough" with the child. The child is otherwise healthy; there is no family history of abnormal bleeding. The mother is the primary caregiver, and the father works.

7.7.11.6 Exam Findings

General: Irritable cry, pale, bulging fontanel

HEENT: Pupils sluggish reaction, right gaze preference

Lungs: RR 28–32

CV: HR 135, ST, blocked PACs, cap refill <2 s

Abdomen: Soft, non-tender, no organomegaly, or palpable masses

Extremities: Limbs normal, painless to palpation; symmetric, full ROM

Neurologic: Child has frequent thrusting movements of the tongue, stiff neck, and global hypotonia; no tonic-clonic movements

Skin: Faint blue bruise to left inferior periorbital area; mucus membranes moist

7.7.11.7 Diagnostic Findings

MRI: Bilateral frontal extra-axial fluid collections (chronic SDH) and an acute interhemispheric bleed. Figure 7.11 shows bilateral subdural hematomas with formed membranes secondary to AHT.

Skeletal survey: Split sutures, no fractures.

Ophthalmology: Bilateral RH.

Lumbar puncture was "bloody"; RBC = 1,580.

7.7.11.8 Discussion

A multidisciplinary team investigated for possible AHT. She was admitted to the PICU for observation. Bilateral subdural drains were placed with resolution of SDHs. The infant was discharged to a foster home. Investigation failed to prove AHT, and custody was returned to the biological mother. She presented 1 year later with inflicted trauma (2 months after placement with biological parents). Injuries in this admission included:

Left distal radius fracture

Left proximal tibial fracture

Right distal radius fracture

Left distal ulnar fracture

Bilateral optic edema, with retinal hemorrhages

Increased subdural hemorrhages, with mass effect

Multiple ecchymosis throughout the body, bite marks throughout integument

Increased radionuclide bone scan activity, ribs (10 and 11), and left scapula, indicative of fracture

The child is currently doing well after adoption by a caring foster family. She has mild

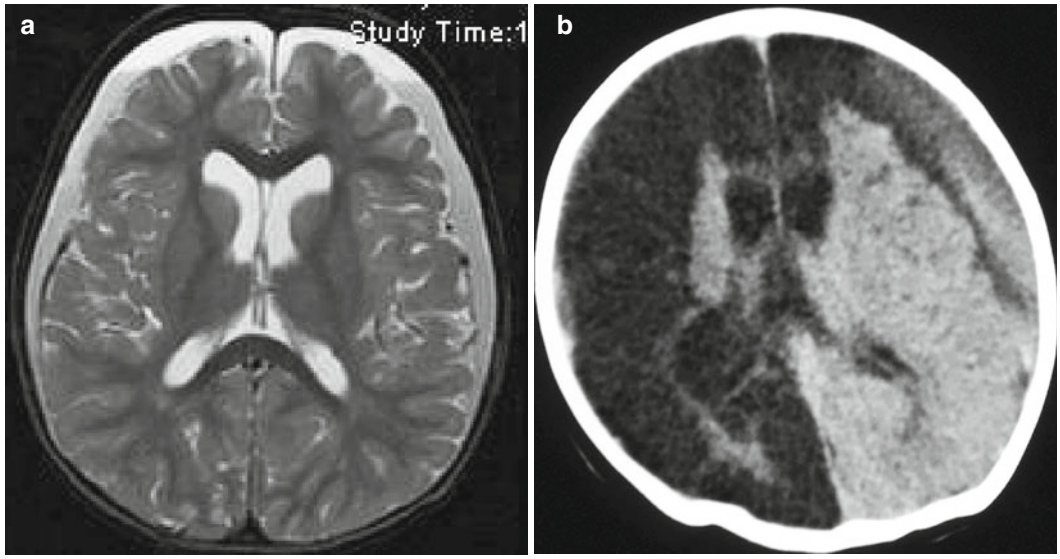

Fig. 7.11 (**a**, **b**) Abusive head trauma. (**a**) Bilateral SDH seen on MRI, which reveals two different densities, implying acute and chronic injury. Subdural membranes are present. (**b**) Severe cerebral atrophy 3 months after infant was shaken

developmental delay and behavioral issues, requiring developmental intervention and rehabilitation therapies.

7.7.11.9 Outcomes

In the acute stage of AHT, the full extent of brain injury on the child's development may not be visible. Imaging 1 year post injury may show extensive atrophy to the brain tissue despite improvement in the patient's neurologic deficits due to the ability of the brain to reassign functions to healthy brain tissue (Barkovich 2005). Research shows that chronic changes, such as cerebral atrophy and ex-vacuo (brain dropout), are present in 40–45 % of AHT (Fig. 7.11) (Cobbs-Ewing et al. 1998).

Information is limited regarding long-term outcome of AHT. Barlow et al. (2001) studied survivors for 59 months, proving that 68 % of the sample had developmental delays at the initial follow-up visit (16 % with mild delays, 16 % with moderate delays, 36 % with severe delays). Study results indicated 60 % had motor deficits, 48 % visual deficits, 20 % seizures, 64 % speech and language deficits, and 52 % behavioral difficulties. Outcomes of AHT, in addition to developmental or cognitive delays, may include seizure disorder, blindness, cerebral palsy, hydrocephalus, and emotional or behavioral problems. Forty percent have deficits severe enough to impair their life-long ability to live independently (Barlow et al. 2001).

7.8 Concepts of Cerebral Physiology

7.8.1 Intracranial Dynamics

The skull forms a protective vault, which houses the brain and other cerebral structures. The protection offered by the cranial vault is not infinite. Severe TBI results in cerebral edema, increased intracranial volume, and eventual increased ICP, leading to decreased perfusion, decreased oxygen delivery, and cell death.

The Monroe-Kellie Doctrine recognizes that the skull is a rigid structure and that the sum of the intracranial volumes is constant. Intracranial components consist of brain (80 %), cerebrospinal fluid (CSF) (10 %), and blood within the cerebral vasculature (10 %). The intracranial pressure is determined by the total intracranial volume and intracranial compliance (the change in pressure

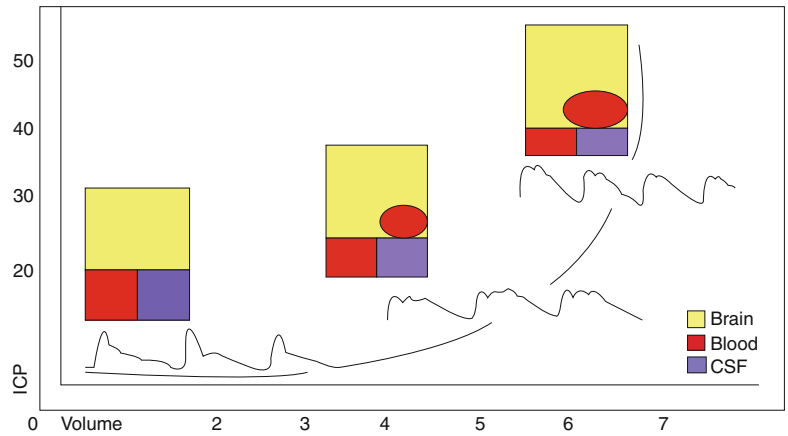

Fig. 7.12 Pressure-volume curve. The Monroe-Kellie Doctrine demonstrates the intracranial contents (brain = *yellow*, CSF = *blue*, blood = *red*). With expanding mass lesion (hemorrhage) in TBI, intracranial volume rises. Pressure rises once compensatory mechanisms fail (pressure-volume curve). Corresponding ICP waveforms are also pictured

which results from a change in volume). When any of the components increase in volume, there must be a compensatory decrease in the others to maintain equilibrium and to prevent an increase in intracranial pressure.

7.8.2 Compensatory Mechanisms

The brain is incapable of a decrease in volume. The CSF and the blood compartments can compensate to a point to maintain equilibrium. This is accomplished by forcing intracranial contents out through the foramen magnum (FM) along the craniospinal axis. Given absence of obstructive hydrocephalus, the CSF is most easily displaced from the ventricles and cerebral subarachnoid space into the thecal or spinal subarachnoid space. The intracranial CSF volume can be therapeutically decreased via an external ventricular drain, or, if the ventricles are compressed and the basilar cisterns are patent, a lumbar drain can be utilized to drain CSF (Levy et al. 1995). With severe increased ICP, the CSF ventricles and cisterns are easily compressed and are absent on neuroimaging. Intravenous blood can be displaced through the FM via the internal jugular veins. As pressure continues to increase, arterial blood is displaced, causing decreased cerebral perfusion pressure (CPP) and diffuse cerebral ischemia. Blood flow ceases when the ICP equals the mean arterial blood pressure. The result is massive infarction. Severe cerebral edema or expanding mass lesion can force the brain downward through the FM, causing cerebral herniation (Greenberg 2010).

7.8.3 Intracranial Compliance

Cerebral compliance is defined as the change in ICP, which results from a change in intracranial volume (Hickey 2009). Compliance is a measure of the brain's tolerance of increases in the ICP. Compliance is limited in that ICP will rise once the compensatory mechanisms are exhausted. The pressure-volume curve (Fig. 7.12) demonstrates that initial increases in intracranial volume are tolerated with little increase in pressure, indicating that intracranial compliance is high. Further increases in volume, especially after compensatory mechanisms are exhausted, result in low compliance, and ICP rises quickly. After compliance is lost, progressively smaller increases in intracranial volume are associated with significant increases in ICP. The normal ICP waveform depicts P1 (percussion wave) as the initial sharp peak, which indicates cardiac ejection. The tidal wave, or P2, is the second lower and more rounded peak, which reflects intracranial compliance. With rising ICP and poor compliance, there is a progressive rise in P2, while P1 and P3 rise much less, giving a rounded appearance to the overall pulse wave (Hickey 2009). The bedside nurse should monitor the ICP waveform for worsening intracranial compliance and as an indication of patient intolerance of nursing interventions.

7.8.4 Cerebral Blood Flow

Maintenance of cerebral blood flow (CBF) and oxygen delivery is critical to maintain normal cerebral metabolism and to prevent neuronal injury and ischemic cell death. In children, the brain receives 25 % of the total cardiac output and consumes 20 % of the total oxygen content. The brain rapidly becomes ischemic if CBF is decreased or compromised (Hickey 2009). Cerebral autoregulation is a protective mechanism, which balances vasoconstriction and vasodilation, to maintain homeostasis (constant cerebral blood flow) despite changes in systemic circulation. In other words, cerebral autoregulation is a protective process where large changes in systemic circulation result only in small changes in the cerebral circulation (Greenberg 2010). Failure of autoregulation after TBI renders the cerebral circulation completely dependent on the mean arterial blood pressure (MAP) and the cerebral perfusion pressure (CPP). Vasomotor dysfunction occurs and the resultant ischemia causes secondary cerebral injury (Kennedy and Moffatt 2004). Adelson et al. (1997) demonstrated that hypoperfusion was common in the first 24 h after pediatric TBI, when cerebral metabolic demand is highest, and is associated with poor outcomes. Hypotension must be treated aggressively to prevent secondary ischemic injury.

Hyperperfusion, also known as hyperemia, is defined as CBF in excess of metabolic demand (Bayir et al. 2003). Hyperemia following pediatric TBI increases the risk of intracerebral hemorrhage and further increases intracranial pressure, causing secondary ischemic injury. A prospective cohort study by Vavilala et al. (2004) measured cerebral autoregulation with transcranial Doppler ultrasonography in 36 children and found that impaired cerebral autoregulation was greater after moderate to severe TBI in children and was associated with poor outcome. Hyperemia was associated with impaired cerebral autoregulation and poor outcome.

Cerebral blood flow is also affected by changes in partial pressure of carbon dioxide ($PaCO_2$), PaO_2, chemical changes in electrolytes and pH balance, and by increased metabolism due to seizure activity and fever. The physiologic mechanism for this is a change in the tone or resistance of cerebral arteries due to local tissue biochemical responses. Hypercarbia is the most potent vasodilator, followed by hypoxia, when PaO_2 falls below 50 %. A low body pH, or acidosis, also causes vasodilation. Presence of fever and seizure activity increases cerebral metabolism and produces vasodilation to meet the increased metabolic demand. Vasodilation increases intracranial blood volume and, after compliance is lost, increases intracranial pressure (Hickey 2009).

Cerebral blood flow is also affected by decreased venous return. Venous return decreases due to increased ICP, as pressure is transmitted to the low-pressure venous system. Decreased cerebral venous return increases cerebral blood volume and further increases ICP. Improper positioning of the child's neck in a rotated position causes compression of the internal jugular vein (IJV) and impedes venous return. The child's head should be positioned midline with the head of the bed elevated to 30° to optimize venous return via the IJV. Increased intrathoracic pressures secondary to high positive end-expiratory pressure (PEEP) with mechanical ventilation can also impede cerebral venous return.

7.8.5 Cerebral Metabolism

The brain is dependent on constant delivery of glucose and oxygen for energy production. The neural cells lack the ability to store these critical substrates. Neurons utilize glucose to produce energy, in the form of adenosine triphosphate (ATP), to meet metabolic demand and maintain aerobic metabolism. Oxygen is required to activate the Krebs cycle. Glucose is required to energize the Krebs cycle. Without oxygen and glucose, the Krebs cycle does not function properly, leading to anaerobic metabolism. Anaerobic metabolism produces excess lactate and pyruvate, which contributes to tissue acidosis, decreased ATP, decreased energy, and cell death. Lack of ATP causes failure of the sodium-potassium pump and its protection of the cellular membrane (Hickey 2009). A hypermetabolic state exists after TBI

(Bayir et al. 2003). Seizure control, sedation, analgesia, fever prevention, and barbiturate administration are examples of therapies which aim to decrease the cerebral metabolic demand.

7.9 Pathophysiology of Intracranial Hypertension

Primary traumatic brain injury is caused by an impact that directly disrupts brain tissue. Secondary injury is caused by late effects of the primary injury or new injury due to processes which compromise cerebral blood flow and tissue oxygenation. The pathophysiology of secondary injury is complex with many interrelated processes that interfere with cerebral function. These secondary processes include failure of compensatory mechanisms, altered cerebral blood flow (dysautoregulation and resultant hypoperfusion or hyperemia), anaerobic metabolism, excitotoxicity (excitatory amino acids such as glutamate), inflammation (increased permeability of the blood–brain barrier and resultant swelling), cerebral edema, oxidative stress (excess free radicals), and ischemic cascade resulting ultimately in cerebral infarction (Kennedy and Moffatt 2004; Hickey 2009). The final fatal pathway is malignant intracranial hypertension. The majority of our knowledge about increased ICP and the treatments employed are directed at therapies, which attenuate cerebral inflammation, prevent or reduce intracranial hypertension, control cerebral hemodynamics, and facilitate substrate delivery (Krieger et al. 2008).

7.9.1 Cerebral Edema

Cerebral swelling is defined as an increase in cerebral blood flow from regional or generalized hyperemia or cerebral blood flow in excess of metabolic demand. Peak swelling usually occurs 2–4 days after the initial injury. Cerebral edema is an increase in brain tissue volume, either local or generalized, due to increased intracellular and extracellular water content (Hickey 2009). There are three types of cerebral edema:

- Vasogenic – extracellular edema of the white matter. Diffuse injury produces an alteration in the permeability of the capillaries of the blood–brain barrier, which is more vulnerable to disruption in the pediatric brain. Plasma and protein leak into the extracellular space locally around brain tumors, infarction, and abscess. Vasogenic edema can also be generalized following TBI (Hickey 2009).
- Cytotoxic – increased intracellular swelling as a result of the ATP-dependent sodium-potassium pump failure, allowing fluid and sodium to accumulate in the cells. Cytotoxic edema results in diffuse brain swelling in both the gray and white matter. Cytotoxicity occurs after hypoxic-ischemic injury and in conditions of hypo-osmolality, such as hyponatremia and SIADH. Edema secondary to hypoxic-ischemic injury peaks at 48–72 h or longer after injury. Vasogenic and cytotoxic injuries occur within hours of injury (Greenberg 2010) and often coexist after TBI (Hickey 2009). Use of osmotic diuretics (mannitol) is useful in acute treatment of vasogenic and cytotoxic edema.
- Interstitial – occurs in severe hydrocephalus where CSF (under pressure) crosses the ependymal tissue, out of the ventricle and into the periventricular white matter.

7.9.2 Intracranial Hypertension

Intracranial pressure is the pressure exerted on the intracranial contents. Normal fluctuations of the ICP occur with any mechanism that increases cerebral venous pressure, such as coughing, crying, and the Valsalva maneuver. The normal value for ICP varies with age in children:

- Infants = 1.5–6 mmHg
- Children = 3–7 mmHg
- Adolescents = less than 10–15 mmHg (Greenberg 2010)

Prevention or treatment of intracranial hypertension in severe TBI is critical to maximize a child's outcome and potentially ensure survival. The threshold for treatment of intracranial hypertension is any sustained ICP of 20 mmHg or

higher. No data exists to support treating children at lower thresholds (Carney et al. 2003; Krieger et al. 2008). It is theorized, however, that the optimal ICP treatment threshold may be age dependent, given that normal blood pressures and ICP values are age dependent. Further investigation is needed (Kochanek et al. 2012).

7.9.3 Cerebral Perfusion Pressure

Cerebral perfusion pressure (CPP) is an estimated measure of the adequacy of cerebral perfusion. CPP is the difference between the mean arterial blood pressure (MAP) to perfuse the brain and the opposing intracranial pressure (ICP) [thus, CPP = MAP – ICP]. The optimal CPP threshold and therapeutic approach to achieve it both remain to be defined. A minimum CPP of 40 mmHg may be considered in children with TBI. Age-specific thresholds in the range of 40–50 mmHg may be considered, with infants at the lower end and adolescents at the upper end of this range (Kochanek et al. 2012). CPP should be determined in a standard fashion with ICP zeroed at the tragus (foramen of Monro or midventricular level) and MAP zeroed to the right atrium with the head of the bed elevated 30°. Cerebral perfusion pressure can be improved through measures that lower the ICP and by measures that raise the MAP (volume or vasopressor administration).

7.9.4 Cerebral Herniation Syndromes

The brain is not acutely compressible, but it will shift within the cranium. Excessive pressure gradients between compartments lead to herniation, where part of the brain is herniated into an adjacent compartment (i.e., supratentorial, infratentorial) or into the spinal column. It is important to consider the anatomic landmarks, which separate the intracranial compartments (Hickey 2009). The tentorium cerebelli is a tent-like partition between the cerebrum and the cerebellum. The space above the tentorium is referred to as the supratentorial space, while the space below the tentorium is referred to as the infratentorial space. The falx cerebri divides the left and right cerebral hemispheres within the longitudinal fissure. The falx cerebelli separates the cerebellum into a left and right side. The foramen magnum (FM) is the opening at the base of the skull through which the brainstem and spinal cord are connected. Three cerebral herniation syndromes, described by Plum and Posner, are demonstrated in Figure 7.13. The cerebral herniation syndromes include cingulate herniation (cingulate gyrus under the falx), uncal herniation (the medial, inferior portion of the temporal lobe, uncus, herniates down through the incisura or tentorial notch into the infratentorial compartment), and central herniation of the cerebellar tonsils and brainstem down through the foramen magnum into the spinal column. Cingulate herniation usually is not clinically significant. Uncal herniation, evidenced initially by ipsilateral sluggish pupillary response, progresses to ipsilateral pupillary dilation, contralateral hemiparesis, trochlear and abducens cranial nerve paralysis, and a decreased level of responsiveness with eventual rostral-caudal deterioration. The mass effect causes lateral displacement of the midbrain, forcing the opposite cerebral peduncle against the tentorium, producing Kernohan's notch (resulting in hemiparesis ipsilateral to the expanding mass lesion) (Hickey 2009). Central herniation causes deterioration in a rostral-caudal pattern, eventually causing cessation of cerebral blood flow and brain death (see Sect. 7.4).

7.10 Collaborative Management of Intracranial Hypertension

Treatment of the child with a TBI focuses on preventing secondary insults and optimizing functional outcomes. Maintaining CBF and optimizing oxygen and substrate delivery and utilization, while preventing or effectively managing intracranial hypertension, accomplish these goals. Additionally, the child needs to have adequate airway support, effective oxygenation and ventilation, and good perfusion. When these fundamental needs are addressed, the child's chances

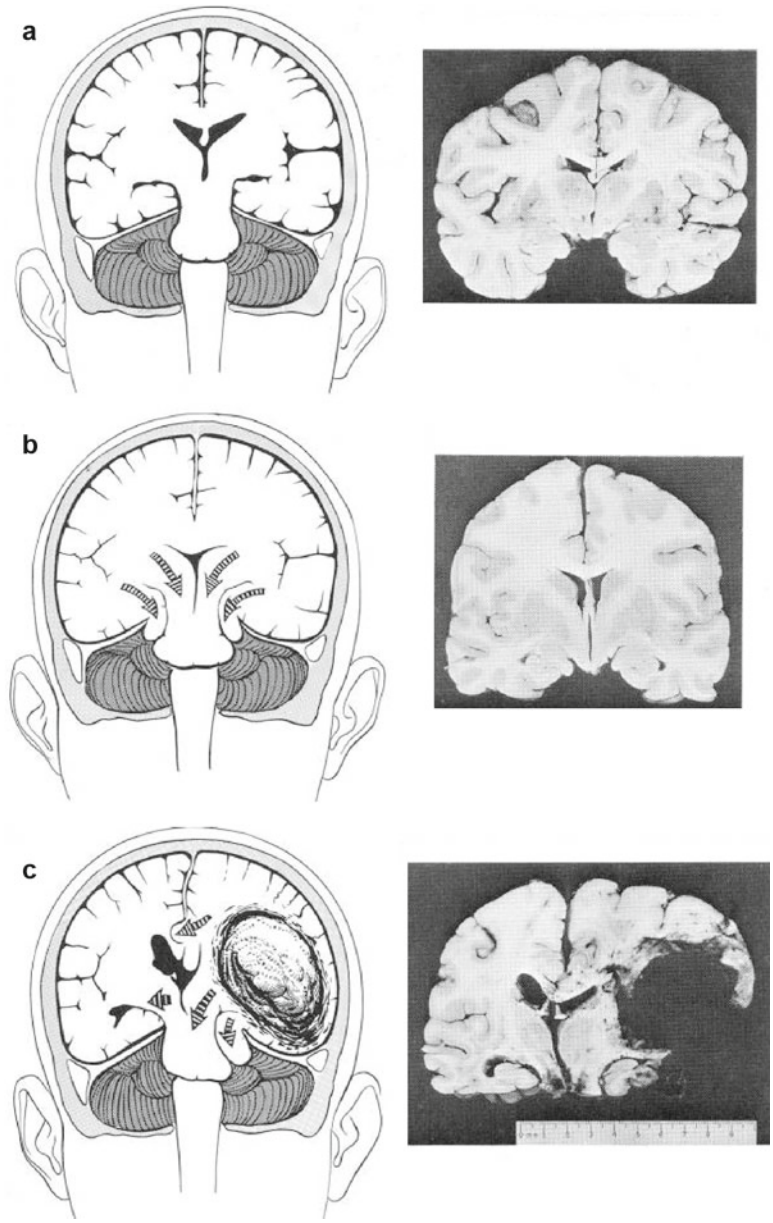

Fig. 7.13 (**a**–**c**) Cerebral herniation syndromes. (**a**) Coronal view: normal relationship of the supratentorial and infratentorial compartments. (**b**) Central herniation occurs when excessive increased ICP in the supratentorial space causes herniation of the cerebellar tonsils and brainstem through the foramen magnum into the spinal column, causing cessation of cerebral blood flow and brain death. (**c**) Uncal herniation occurs when the uncus or medial, inferior portion of the temporal lobe herniates downward through the incisura of the tentorium (Reprinted with permission from Plum and Posner (1982))

of meaningful recovery and effective brain injury management are improved.

The main focus of management is to prevent or minimize secondary injuries, such as cerebral ischemia, cerebral edema, and neurochemical alterations. Since hypoxia and hypotension are known to worsen secondary injury by causing cerebral vasodilatation, care must be taken to quickly recognize and treat these mitigating factors (Huh and Raghupathi 2009). In general, the goal of treatment is to maintain an ICP < 20 mmHg, along with age-appropriate CPP. ICP management interventions include therapies to decrease cerebral volume, control CSF volume, control cerebral blood volume, and decrease cerebral metabolic rate. Because there exists a lack of data from well-designed and controlled pediatric studies to direct the treatment of children with brain trauma, recent guidelines have been released that assimilate the research results that are available and provide

expert consensus on therapies (Carney et al. 2003; Kochanek et al. 2012). There is some evidence in adult studies to suggest that adherence to evidence-based treatment guidelines in severe TBI can lead to improved outcomes (Arabi et al. 2010; Fakhry et al. 2004; Palmer et al. 2001). Figure 7.14 shows an example of a severe TBI treatment guideline utilized in a pediatric intensive care unit. The guideline is based on a tiered gradation system, where tier I is the initial intervention, and then intervention progresses to tier II and tier III as the maximum level of intervention for each category. The level of intervention is advanced to the next tier if set ICP and CPP parameters are not met. In addition, children with severe TBI are more likely to survive when they are transported to a pediatric trauma center if one is available or an adult trauma center with added capabilities to treat children (Potoka et al. 2000; Badjatia et al. 2007).

7.10.1 Initial Resuscitation

Pre-hospital care of the child with TBI begins at the scene with rapid assessment and support of airway, breathing, and circulation. Supplemental oxygen should be administered and hypoxia (oxygen saturation < 90 % or P_aO_2 < 60 mmHg) avoided. In general, if the GCS is ≤8, endotracheal intubation should be considered, although there is no research evidence that supports endotracheal tube intubation over bag-valve-mask ventilation for pre-hospital management (Carney et al. 2003). Upon arrival at a medical center, rapid-sequence intubation should be performed if the CT scan demonstrates diffuse cerebral edema, there is risk of neurologic decompensation, respiratory instability, or loss of protective airway reflexes (Carney et al. 2003). The intubation procedure should include medications to facilitate the process, which do not further increase ICP, such as thiopental, etomidate, lidocaine, and a short-acting, non-depolarizing neuromuscular blocking agent (American Heart Association 2006). Normoventilation (P_aCO_2 35–40 mmHg) should be ensured during initial resuscitation (Carney et al. 2003; Badjatia et al. 2007).

Hypotension has been shown to increase the morbidity and mortality of traumatically brain-injured children (Stocchetti et al. 2010; Badjatia et al. 2007). Because hypotension is a serious and potentially preventable secondary insult, signs of inadequate perfusion should be recognized and treated aggressively. Cerebral perfusion is partially dependent on an adequate MAP; therefore, age-appropriate blood pressure must be maintained to ensure adequate end-organ perfusion and CPP and to prevent ischemia and resultant infarction. The following formula is used to determine median blood pressure (50th percentile) for children greater than 1 year of age: 90+ (2× age in years) (American Heart Association 2006). Table 7.9 shows ranges for median to 90th percentile age-related blood pressures in children. Because of the risk of poor outcome for children with systolic blood pressure below the 75th percentile for age, a higher blood pressure should be targeted initially (Badjatia et al. 2007). Children can be severely hypovolemic without demonstrating decreased blood pressure, and so rapid fluid volume resuscitation should occur both at the scene and upon arrival at the hospital. If appropriate amounts of fluid volume resuscitation do not improve signs of inadequate perfusion, vasopressor support should be initiated (Marcoux 2005).

7.10.2 Intensive Care Management

The child with TBI should be initially evaluated in the emergency department and then taken to the medical imaging department for further evaluation. Radiographic testing may include cervical spine evaluation, CT scan, and possible MRI. From there, the child may proceed to the operating room for removal of expanding lesions, control of hemorrhage, evacuation of significant hematomas, insertion of an ICP catheter or catheter for extraventricular drainage, or decompressive craniectomy (Figaji et al. 2008; Jagannathan et al. 2007). Although randomized controlled trials of the safety and efficacy of decompressive craniectomy in pediatric patients with severe brain injury have not been undertaken, there are instances when this procedure may lower

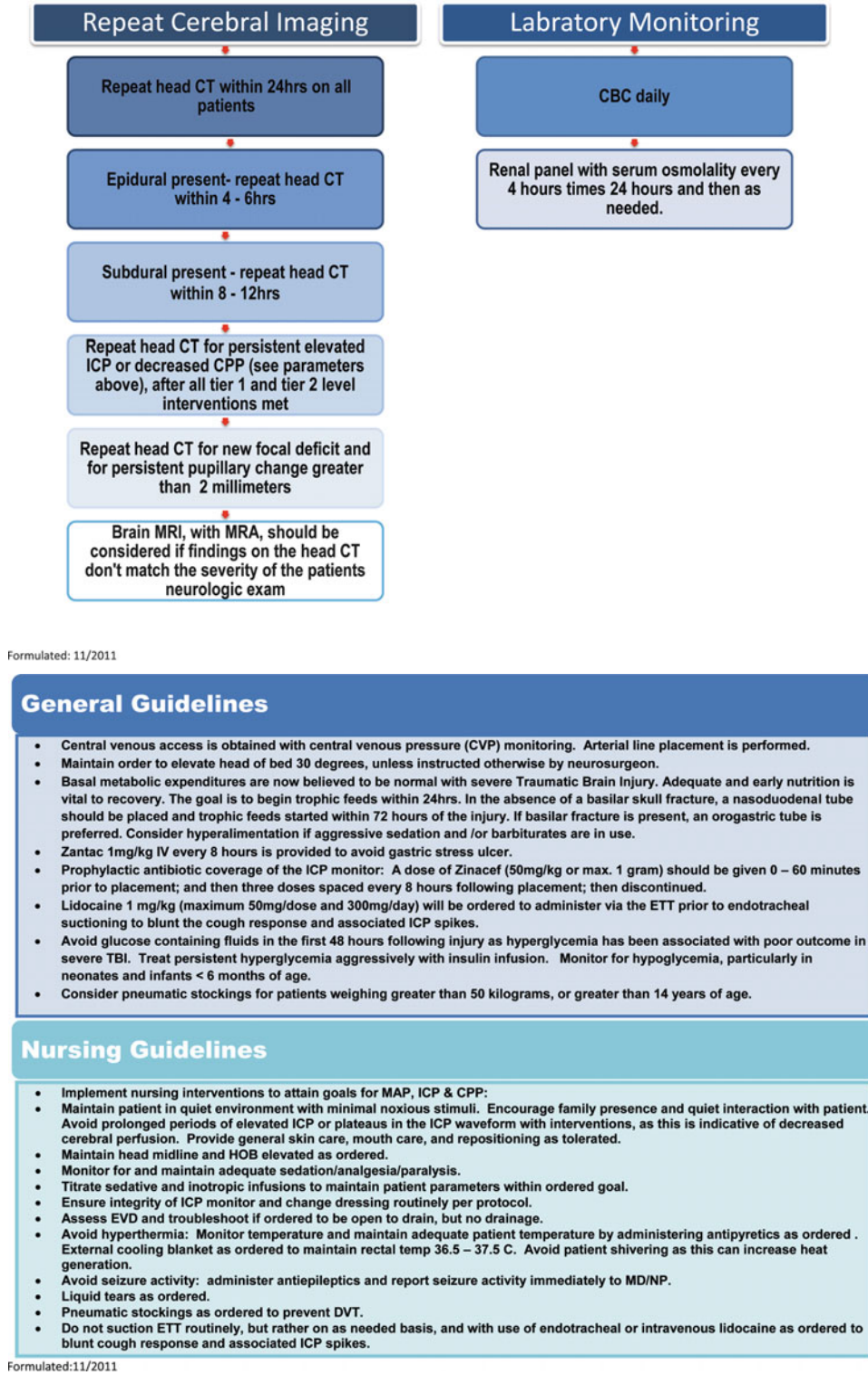

Fig. 7.14 Evidence-based treatment guideline for severe pediatric TBI with Glasgow Coma Scale (GCS) score <.8. The guideline is based on a tiered gradation system, where tier I is the initial intervention; and then intervention progresses to tier II and tier III as the maximum level of intervention for each category. The level of intervention is advanced to the next tier if set ICP and CPP parameters are not met. Used with permission from The Children's Medical Center of Dayton

Table 7.9 Normal blood pressure in children (50–90th percentile)

Age	Systolic pressure[a] (mmHg)	Diastolic pressure[a] (mmHg)
Birth (3 kg)	50–70	25–45
Neonate (96 h)	60–90	20–60
Infant (6 months)	87–105	53–66
Toddler (2 years)	88–102	42–59
School age (7 years)	97–111	57–72
Adolescent (15 years)	103–127	61–79

National High Blood Pressure Education Program Working Group on High Blood Pressure in Children and Adolescents (2004)
[a]For 50th percentile of height

ICP and improve outcomes. Table 7.10 lists criteria to guide the practitioner in determining if the child is a candidate for decompressive craniectomy. After determining if any surgical intervention is necessary, further head injury management then generally takes place in the intensive care unit.

7.10.3 Intracranial Pressure Monitoring

The ability to identify rapidly increasing pressure is crucial in the treatment of severe TBI and in the prevention of cerebral ischemia and infarction. ICP monitoring is recommended for the child with a traumatic brain injury that has a GCS ≤ 8 (Carney et al. 2003; Kochanek et al. 2012). It should be noted that the presence of an open fontanel does not negate the utility of ICP monitoring or preclude the development of intracranial hypertension. In addition, a monitor may be placed in the child that has clinical signs of increasing ICP, after major neurosurgical procedures, or when serial neurologic assessments may be masked by sedation, neuromuscular blockade, or anesthesia.

Either a fiberoptic-tipped wire, a microchip sensor, or a fluid-filled catheter system can be placed in the intracranium, which allows for the continuous measurement of ICP. While fiberoptic-tipped devices can be placed in the intraventricular, parenchymal, and less often in the epidural, subdural, and subarachnoid spaces, fluid-filled ICP catheters are generally placed in the intraventricular space, particularly if CSF drainage is desired. Table 7.11 lists potential complications associated with the use of intracranial catheters.

Table 7.10 Criteria for decompressive craniectomya

1. Severe traumatic brain injury
2. Refractory intracranial hypertension
3. Diffuse cerebral edema on CT imaging within 48 h of injury
4. No episodes of sustained ICP >40 mmHg prior to surgery
5. GCS ≤ 4 at any point prior to surgery
6. Secondary clinical deterioration
7. Evolving cerebral herniation syndrome

[a]Some or all may be present (Carney et al. 2003)

Table 7.11 Potential complications associated with ICP monitoring catheters

Hemorrhage
Infection
Over-drainage of cerebral spinal fluid
Catheter misplacement
Catheter migration
Catheter obstruction

The fiberoptic-tipped catheter and microchip sensor are zeroed before insertion and require a monitoring unit supplied by the manufacturer for ICP readings. Alternatively, a fluid-filled ventriculostomy catheter system can be set up to allow for the continuous drainage of CSF. This system contains an external strain-gauge transducer that is coupled to the patient's intracranial space through a fluid-filled line. The transducer must be secured at a fixed reference point, usually the foramen of Monro or midventricular level, which can be approximated by positioning the transducer level with the tragus or external auditory meatus. The system must be

Table 7.12 Nursing priorities for the child undergoing ICP monitoring

1. Keep the ICP monitoring system operational and intact, ensuring that all connections are secure
2. Prevent strain on the tubing and cables, particularly during patient repositioning and transport
3. Zero and level the system according to manufacturer's recommendations
4. Ensure the transducer of a fluid-filled system is leveled at the appropriate anatomical location, especially after patient repositioning and transport
5. If draining CSF, maintain the drainage chamber at the level ordered
6. Empty the drainage chamber regularly, recording the amount of CSF output
7. Do not allow the air filter of the drainage chamber to get wet
8. Document ICP and CPP readings
9. Note child's ICP response to interventions and ICP trends over time
10. Maintain a dry sterile dressing around the insertion site per hospital policy
11. Monitor for and notify the neurosurgeon of CSF leakage around the insertion site, other drainage, and signs of infection

zeroed to atmospheric pressure and leveled to ensure accuracy of the ICP data. Nursing care of the patient with an ICP monitor is outlined in Table 7.12.

Intracranial hypertension is defined as an ICP ≥ 20 mmHg, and treatment designed to reduce ICP and improve cerebral perfusion should be initiated at this point (Brain Trauma Foundation 2007). Prolonged periods of increased ICP and dramatic elevations in ICP are associated with poor outcomes in the pediatric patient (Carney et al. 2003). Depending on the child's age, a CPP threshold of 40–50 mmHg should be maintained at all times during head injury management (Kochanek et al. 2012). In addition to ICP data, evidence of intracranial hypertension should be corroborated by frequent patient assessment, other physiologic parameters, and cranial imaging studies.

7.10.4 Jugular Venous Oxygenation Saturation Monitoring

Continuous measurement of venous saturation can be obtained using a fiberoptic catheter placed retrograde into the jugular vein. This monitoring technique can provide information on cerebral oxygen delivery and consumption and the effectiveness of therapies. Because most of the cerebral circulation drains into one jugular vein, oxygen saturation is measured after cerebral perfusion has occurred. Normal jugular venous oxygen saturation ($S_{jv}O_2$) values are 55–70 %. Values below 55 % indicate inadequate oxygen delivery or utilization within the injured brain, and treatment interventions should be initiated when the jugular venous saturation is <50 % (Brain Trauma Foundation 2007). Conditions that decrease $S_{jv}O_2$ are hypoxia, hypotension, increased ICP, and hypocarbia. $S_{jv}O_2$ monitoring provides a global picture of CBF and metabolic need, but does not provide any information about oxygen consumption at the site of injury.

7.10.5 Monitoring Partial Pressure of Oxygen

$P_{bt}O_2$ is a measure of cerebral (brain tissue) oxygenation. A microprobe is inserted into uninjured parenchymal brain tissue or the area of primary injury (penumbra) of an intracerebral lesion. While placing the microprobe into uninjured brain tissue will more closely assess global cerebral oxygenation, placement in an intracerebral lesion will provide data regarding cerebral oxygenation in an area most at risk (Haitsma and Mass 2002; Maloney-Wilensky et al. 2009). Either method should give an indication of cerebral oxygenation and ischemia (Maloney-Wilensky et al. 2009). Normal values for non-injured brain tissue range from 20 to 35 mmHg. Instances that can decrease $P_{bt}O_2$ are hypocarbia, hypoxemia, hyperthermia, decreased CBF and ischemia, decreased CPP, and elevated ICP (Littlejohns et al. 2003). Treatment interventions to improve cerebral oxygenation should begin when $P_{bt}O_2$ values are <15 mmHg (Brain

Trauma Foundation 2007; Lang et al. 2007; Littlejohns et al. 2003). Values below this have been associated with poor outcomes and death.

7.10.6 CSF Drainage

External ventricular drainage of CSF is a common management therapy that is often used in conjunction with ICP monitoring. The CSF is drained to reduce intracranial fluid volume and thus decrease ICP. Drainage can be continuous or intermittent, depending on the child's situation and the neurosurgeon's preference. For instance, drainage may be ordered any time the ICP is above a preselected value for a specified time. Moving the CSF collection device up or down in relation to the reference level point will control the amount of drainage. The higher the drain is above the reference level, the higher the ICP must be for CSF to flow into the collection device. Conversely, lowering the drain will cause CSF to flow at a lower ICP. Care must be taken when draining CSF to treat elevated ICP, as excessive drainage of CSF may cause the lateral ventricles to collapse. In addition, excessive CSF output may lead to hyponatremia, which is corrected with intravenous fluid administration designed to replace the CSF fluid volume.

The use of lumbar CSF drainage has occasionally been reported in pediatric patients with ICP refractory to other management therapies (Carney et al. 2003; Levy et al. 1995; Murad 2008; Kochanek et al. 2012). The lumbar drain is used simultaneously with a functioning ventricular catheter when the basilar cisterns are open. Patients for whom lumbar drainage is contraindicated include those with intracranial masses or shifts because transtentorial herniation could result (Kochanek et al. 2012).

7.10.7 Analgesia, Sedation, and Neuromuscular Blockade

Analgesia and sedation are important adjuncts to other treatments for the brain-injured child. Because pain and anxiety contribute to increased ICP and the cerebral metabolic rate, it is imperative to provide relief from pain, anxiety, and agitation. Additionally, the use of these agents can also facilitate the child's tolerance of other therapies, such as mechanical ventilation and suctioning, intradepartmental transport, and monitoring devices. The nurse is in a vital position to assess, advocate for, and effectively manage the child's level of sedation and analgesia. Depending on practitioner preference, common agents used are opiates, benzodiazepines, and barbiturates. Although routinely used in the adult with traumatic brain injury, the use of continuous infusions of propofol is not recommended for sedation of patients in the pediatric intensive care unit because it has been associated with fatal metabolic acidosis, rhabdomyolysis, and hypoxia.

While the use of short-acting neuromuscular blockade agents may facilitate intubation and the tolerance of therapies, the patient's neurologic exam is blunted. Therefore, these agents are generally used only when the child's agitation and increased ICP persist despite adequate doses of sedation and analgesia. The paralytic agent is generally allowed to wear off at intervals to allow for a complete neurologic examination. Neuromuscular blocking agents should never be used without the concomitant administration of a sedative or analgesia.

7.10.8 Hyperosmolar Therapy

Osmotic diuresis for the treatment of the head-injured child is accomplished through the use of mannitol and/or hypertonic saline (HS). While mannitol has been the mainstay of therapy for many years, there is increasing evidence that using HS in children with intractable intracranial hypertension results in a decrease in ICP and subsequent increase in CPP (Khanna et al. 2000; Peterson et al. 2000; Upadhyay et al. 2010; Yildizdas et al. 2006). The nurse should keep in mind that the overall goal is euvolemia (fluid balance); therefore, hyperosmolar therapy may be contraindicated in the presence of hypotension (Brain Trauma Foundation 2007).

After mannitol administration, an osmotic gradient between plasma and parenchymal tissue

develops, drawing fluid from brain tissue into the vascular space. Osmotic diuresis then occurs, which results in a net reduction of brain water content. Further, because of fluid movement, mannitol reduces hematocrit and blood viscosity, improving CBF and reducing blood vessel diameter. ICP and cerebral blood volume decrease almost immediately. Therefore, because of its rapid onset of action, a 20 % mannitol solution is usually administered as a bolus dose of 0.25–1 g/kg body weight (Brain Trauma Foundation 2007; Carney et al. 2003). The nurse should be aware that mannitol may crystallize, so an in-line filter should be used to prevent the administration of any crystals. Serum osmolarity must be monitored when using hyperosmolar therapies. The maximum recommended serum osmolarity when using mannitol is 320 mOsm/l. Because mannitol is excreted unchanged in the urine, renal failure can result with higher serum osmolarity. With chronic administration, mannitol may cause rebound cerebral edema because it is believed to disrupt the blood–brain barrier and accumulate in the interstitial space of the brain parenchyma, causing a reverse osmosis (Carney et al. 2003). Additional side effects of mannitol therapy are dehydration with resultant hypotension following overly effective osmotic diuresis and electrolyte imbalance.

Hypertonic saline works by increasing serum sodium and serum osmolarity, creating an osmotic gradient to pull water from the intracellular and interstitial compartments of the brain, reducing cerebral edema and ICP. Sodium chloride creates a driving force to bring water from the brain into the intravascular compartment in regions with intact blood–brain barrier, thus reducing water content, mass effect, and ICP. In addition, intravascular volume expansion occurs after administration of HS solutions. While there is no change in systemic vascular resistance, MAP increases because of increases in cardiac output and intravascular volume. This increase in MAP can further improve CPP. For acute management of intracranial hypertension, the recommended dose of 3 % HS is 6.5–10 ml/kg (Kochanek et al. 2012). Subsequently, 3 % saline can be administered via continuous infusion at a dose of 0.1–1 ml/kg/h as needed to maintain ICP < 20 mmHg (Carney et al. 2003; Peterson et al. 2000; Kochanek et al. 2012). An osmolarity up to 360 mOsm/l has been well tolerated in children receiving HS (Carney et al. 2003; Kochanek et al. 2012). Although there are no reports in pediatric studies of HS, a potential side effect in patients with preexisting chronic hyponatremia is osmotic demyelination syndrome (ODS), which involves demyelination, primarily of the pons, that can be noted clinically by the onset of lethargy and quadriparesis. Other possible side effects of the use of HS include coagulopathy, rebound rises in intracranial hypertension, electrolyte imbalance, hyperchloremic acidosis, and the risk of phlebitis if the solution is administered via a peripheral vein.

7.10.9 Hyperventilation

During hyperventilation, P_aCO_2 decreases, resulting in cerebral vasoconstriction and a reduction in cerebral blood volume. Although ICP rapidly decreases in this situation, cerebral perfusion is compromised and ischemia can result (Skippen et al. 1997; Stiefel et al. 2006). Because of these concerns, prophylactic hyperventilation and hypocarbia should be avoided. Mild hyperventilation (P_aCO_2 30–35 mmHg) may be instituted when other therapies such as adequate sedation and analgesia, neuromuscular blockade, hyperosmolar therapy, and CSF drainage have not been effective in correcting intracranial hypertension (Carney et al. 2003). Depending on practitioner preference, aggressive hyperventilation may be used in the event of acute brain herniation or significant ICP elevation. However, prophylactic severe hyperventilation (P_aCO_2 < 30 mmHg) should be avoided for 48 h after injury (Kochanek et al. 2012). In this instance, $P_{bt}O_2$ and/or $S_{jv}O_2$ may be monitored to allow for immediate detection of cerebral ischemia (Stiefel et al. 2006).

7.10.10 Temperature Regulation

Hyperthermia is known to increase cerebral metabolic rate and ICP and should be avoided in the traumatically brain-injured child. Core body

temperature may be measured via the bladder or rectal route or through a pulmonary artery catheter. Brain temperature can also be assessed during both $P_{bt}O_2$ and $S_{jv}O_2$ monitoring. Because core body temperature measurement may be lower than actual brain temperature (Mcilvoy 2007; Soukup et al. 2002), the nurse may need to institute treatment for fever at a rectal temperature of 37.5 °C, instead of the usual 38.5 °C. Fever can be treated with antipyretics and external cooling devices, although shivering must be avoided because it will further increase cerebral metabolic rate and ICP. Furthermore, it is important to identify and treat the cause of the elevated temperature. Some common causes of fever after a TBI include atelectasis, infection, cerebral irritation from hemorrhage, and injury to the hypothalamus.

Mild to moderate hypothermia (32–34 °C) as a treatment for intracranial hypertension has been considered in both adults and children, although the results do not yet provide clear direction for treatment (Adelson 2009; Adelson et al. 2005; Bourdages et al. 2010; Clifton et al. 2009; Hutchison et al. 2008, 2010; Li et al. 2009; Shafi and Mariscalco 2006; Sydenham et al. 2009). The goal of hypothermia therapy is to slow the body's metabolic processes. In addition, induced hypothermia may improve ICP, increase oxygen supply to areas of ischemic brain, and help prevent seizures (Bernard and Buist 2003). The sequelae of induced hypothermia include increased risk of acquired infection, lactic acidosis, sludging blood flow, cardiac arrhythmias, and seizures. Because controlled hypothermia has not yet shown an improvement in outcome, it is not currently recommended for the treatment of severe TBI in children outside of clinical trials (Adelson 2009; Sydenham et al. 2009). Should hypothermia therapy be initiated, care should be taken to avoid short periods of cooling (<24 h), and the patient rewarmed at a rate of <0.5 °C/h (Kochanek et al. 2012).

7.10.11 Barbiturate Therapy

Barbiturates (e.g., pentobarbital) have been used for many years as a therapy for intracranial hypertension that is not responsive to other treatments. Despite this history of use, limited clinical trials have been performed in children, and other studies in adults have not consistently demonstrated improved outcomes (Brain Trauma Foundation 2007). However, in the pediatric patient with a potentially recoverable brain injury that has elevated ICP which is not responsive to other management strategies, barbiturate coma therapy may be used (Carney et al. 2003).

Barbiturates decrease cerebral blood flow and the cerebral metabolic requirements of the brain with a resultant decrease in ICP. As a side effect, barbiturate therapy causes myocardial depression and vasodilation, which results in hypotension. The child who is receiving barbiturate therapy should have cardiovascular parameters, including blood pressure and central venous pressure monitored continuously. The nurse should be prepared to administer fluids and inotropic agents as needed during barbiturate therapy. Continuous electroencephalogram (EEG) monitoring is needed to evaluate burst suppression. The barbiturate is administered via continuous intravenous infusion following a loading dose. The infusion is titrated based on the patient's ICP response and EEG tracing. Since rebound intracranial hypertension can occur after discontinuation of barbiturate therapy, the medication should be weaned slowly over a few days (Brain Trauma Foundation 2007).

7.10.12 Hydration and Nutrition

During the first 48–72 h, the child with a TBI should receive intravenous fluid therapy to maintain a euvolemic state. Fluid boluses, including blood products as indicated, may be administered to maintain adequate perfusion, age-appropriate blood pressure, central venous pressure, and urine output. A bladder catheter is essential for monitoring accurate urinary output. The intravenous fluid of choice is usually normal saline, lactated Ringer's, or hypertonic saline. Hypotonic fluids should be avoided and hyponatremia prevented, since both contribute to cerebral edema.

Research results are beginning to suggest a relationship between hyperglycemia and poor outcome from pediatric TBI (Cochran et al. 2003;

Sharma et al. 2009; Smith et al. 2012). In adult studies, the harmful effects of hyperglycemia on the traumatically brain-injured patient have been repeatedly verified (Jeremitsky et al. 2005; Laird et al. 2004; Salim et al. 2009). Because of this, glucose-containing parenteral fluids are normally not used during the first 48 h after injury. Serum glucose must be monitored and appropriate glucose correction therapy initiated prior to the patient becoming hypoglycemic (serum glucose < 75 mg/dl) since this may also result in harm (Faust et al. 2011). The current recommended blood glucose target for most critically ill patients is 140–180 mg/dl (Moghissi, et al. 2009).

Meeting nutritional requirements is of utmost importance. The patient with a TBI requires approximately 130-160% of their expected metabolic expenditures (Bratton et al. 2007). Enteral feedings should begin by 72 h after injury (Vizzini and Aranda-Michel 2011), with full caloric feeds established within 7 days (Bratton et al. 2007). Nutritional formula appropriate for age and caloric requirements is administered via a gastric or transpyloric tube, which can be inserted by the nurse at the bedside (Mehta 2009). In cases where enteral feeding cannot be initiated, total parenteral nutrition should be started. In addition, because the patient is likely to be immobile and receiving opiates, a bowel regimen should be established early and should include a stool softener.

7.10.13 Additional Nursing Care

The nurse at the bedside caring for the traumatically brain-injured child has an important role in preventing secondary injury and optimizing outcomes. The nurse must also balance the care needs of the patient with the requirement not to further increase ICP. Vigilance to proper patient positioning is necessary. The head of the bed should be elevated 30°. Moreover, the patient's head should be maintained in a midline position to prevent obstruction of venous outflow, which can further worsen intracranial hypertension. The nurse should check that the cervical collar or tracheostomy ties are not so tight that they constrict venous outflow. Complications from immobility should be assessed and prevented. The child will need to be repositioned as tolerated, keeping the head in midline with the body. The risk of skin breakdown should be assessed, skin integrity routinely monitored, and prevention measures started for at-risk patients (Curley et al. 2003; Noonan et al. 2006; Schindler et al. 2011). Preventive measure for deep vein thrombus, such as passive range of motion or sequential compression devices, should be initiated as indicated based on the child's age and body size.

Depending on the child's response to stimuli, the environment should be quiet and free from extraneous noise. The nurse will need to determine if the child will tolerate the clustering of care activities or if care needs must be met in increments with rest periods between activities. The child's response, vital signs, ICP, and CPP parameters should be monitored during care activities. For instance, if the ICP rises significantly during care, the nurse may need to cease care and allow the ICP to return to baseline for a period of time before meeting further care needs (Tume et al. 2011). Also, the nurse should assess the child's need for additional analgesia or sedation during care activities that must take place.

Because this child is likely to be intubated and ventilated, the nurse should institute measures to prevent ventilator-associated pneumonia, such as oral care and head of bed elevation. Endotracheal tube suctioning should be initiated based on the child's clinical assessment and should not be performed on a scheduled basis if the patient does not demonstrate the need for suctioning. Coughing during suctioning can increase ICP. Endotracheal lidocaine may need to be administered before endotracheal tube suctioning to blunt the gag and cough response. Preoxygenation is necessary during the suctioning procedure so that hypoxia can be avoided.

During this time of intensive care, the child's family will need much support and education. They are likely to experience uncertainty and fear about their child's recovery and future. The nurse will need to describe the equipment

surrounding the child and should provide the family with anticipatory guidance on what to expect during the child's initial care and recovery. The family may need to be given direction on the role they can assume in the intensive care unit. A family-centered intensive care unit environment that establishes a partnership between the family and the healthcare team is vital. The family may be encouraged to interact with their child and touch the child as appropriate. The nurse should observe the patient's response to this interaction, including ICP response. The presence of family has not been shown to be detrimental to patient outcomes and should be evaluated on a case-by-case basis.

7.11 Endocrine Complications

7.11.1 Diabetes Insipidus

Diabetes Insipidus (DI) can occur after a traumatic head injury or intracranial surgery because of damage to the cells in the hypothalamus that produce antidiuretic hormone (ADH). DI can also occur after injury to the posterior pituitary gland, whereby the injured gland does not release ADH. When there is not enough ADH present, the collecting ducts and the distal tubules of the kidneys do not reabsorb free water, which results in intravascular volume depletion. Signs and symptoms of DI include large amounts of very dilute urine, severe dehydration, thirst, hypernatremia, and elevated serum osmolality. Excessive thirst in an alert child may also be indicative of DI and often is an effective mechanism to prevent severe dehydration. Table 7.13 lists laboratory values associated with DI.

The treatment for DI includes frequent assessment of laboratory values, as well as accurate calculation of fluid administration and urine output. Fluid resuscitation with isotonic solutions may be needed initially to treat severe volume depletion. After stabilization, fluid volume deficits and ongoing urine volume losses are replaced hourly. An intravenous infusion of vasopressin may become necessary if fluid replacement therapy alone is unsuccessful. For chronic management of DI, desmopressin is administered via the nasal or oral route.

Table 7.13 Selected laboratory values associated with DIa, SIADHb, and CSWc

	DI	SIADH	CSW
Urine			
Specific gravity	<1.005	>1.020	>1.010
Osmolality	<250	>500	>300
Urine output (ml/kg/h)	>3	<1	≥1
Sodium (mEq/l)	<40	>60	>120
Serum			
Osmolality	>305	<275	<275
Sodium	>150	<130	<130

[a]Diabetes insipidus
[b]Syndrome of inappropriate secretion of antidiuretic hormone
[c]Cerebral salt wasting

7.11.2 Syndrome of Inappropriate Secretion of Antidiuretic Hormone

SIADH involves overproduction or release of ADH from the pituitary. This excess ADH increases the permeability of the collecting ducts and the distal tubules of the kidneys, causing water to be retained. The child can become fluid overloaded and fluid can further shift from the intravascular to interstitial spaces, worsening cerebral edema. Clinically, the child will demonstrate decreased urine output, nausea and vomiting, dilutional hyponatremia with the risk of seizures, and mental status changes. Table 7.13 lists laboratory values associated with SIADH.

The treatment for SIADH, like that for DI, also includes frequent assessment of laboratory values and accurate calculation of fluid administration and urine output. Fluid restriction therapy is usually initiated. The child is also monitored frequently for changes in mental status and seizures. Severe hyponatremia, especially when associated with seizures, is treated with 3 % hypertonic saline via the intravenous route (Clark et al. 2008). Rapid correction of sodium levels should be avoided because this can cause CNS osmotic demyelination syndrome.

7.11.3 Cerebral Salt Wasting

Cerebral salt wasting (CSW) is a condition characterized by true loss of body sodium through natriuresis. Though the exact mechanism by which CSW occurs is unclear, it is theoretically caused by natriuretic peptides (Clark et al. 2008). This sodium loss results in decreased plasma volume, increased concentration of nitrogen (in the form of urea) in the blood, and a negative salt balance. Treatment of this condition involves first distinguishing it from SIADH. Fluid and sodium replacement therapies are then initiated, along with ongoing monitoring of serum sodium levels. Table 7.13 lists laboratory values associated with CSW.

7.12 Postoperative Nursing Care and Complications

Surgical interventions for pediatric head trauma were discussed throughout this chapter and include placement of an ICP monitor with external ventricular drain (EVD) or lumbar drain; craniotomy for elevation of depressed skull fractures; evacuation of hematomas, bony fragments, and pulped brain; and decompressive craniectomy. Postoperative nursing care following neurosurgery includes observation of the vital signs, baseline as well as frequent ongoing neurologic assessments, management of neuro-monitoring devices, monitoring for postoperative complications, and good general postoperative care to include pain management, prevention of infection, nutritional status, and psychosocial support of the child and family (Hickey 2009).

7.12.1 Preoperative Baseline

The preoperative and immediate postoperative assessment provides a baseline for comparison when assessing for postoperative deterioration or complications. For consistency, it is ideal for the same nurse to care for the child before and after neurosurgery. When this is not possible, the nurse assuming care should seek knowledge of the preoperative assessment from the previous caregiver, surgeon, anesthesiologist, post-anesthesia care unit nurse, the medical record, and the parents in order to recognize and anticipate changes in the child's postoperative neurologic assessment.

The conscious child should be prepared for the operative experience in a developmentally appropriate manner. Reassure the child that they will remain asleep and unable to feel pain during surgery and awaken afterward to see their parents. Educate them that pain is expected and that medications will be available to alleviate their pain. Advise them to anticipate a large number of people and equipment to help take the best care of them when they awaken. Parents need to be prepared for the appearance of the child postoperatively with a head turban dressing, ICP/EVD monitor, multiple intravenous lines, monitors, and possible endotracheal tube and ventilator.

7.12.2 Assuming Postoperative Nursing Care

Hand-off report following surgery should include type of neurosurgical procedure and region of brain affected, intraoperative complications, anesthetic and analgesic agents used, preexisting medical problems and neurologic deficits, presence of new neurologic deficits since surgery, surgeon's insight on what new deficits may develop, fluid/electrolyte status, lab values, and the postoperative physician's orders (Hickey 2009). Appropriate monitoring equipment should be attached and may include any or all of the following: cardiorespiratory monitor, mechanical ventilation, pulse oximetry, end tidal CO_2, arterial line, central venous line, ICP monitor, EVD, $SjvO_2$, and $PbtO_2$ monitoring. Ideally, the reporting nurse and the oncoming nurse should assess the patient together to facilitate a clear understanding of the patient's baseline exam, to decrease the chance of miscommunication, and to prevent missed signs of subtle deterioration (Hickey 2009).

7.12.3 Vital Functions

Protection of the pediatric airway following neurosurgery is paramount, whether the child is extubated immediately following neurosurgery or remains intubated and on mechanical ventilation. The nurse should monitor for signs of respiratory distress and assist with bag-valve-mask breathing and reintubation if required. Possible causes of airway or ventilatory deterioration include tube displacement, tube obstruction, pneumothorax, and equipment failure (American Heart Association 2006). Inadequate ventilation results in hypoxia and hypercarbia, which cause increased CBF, ischemia, and increased ICP.

Vital signs should be recorded frequently, according to your institution's policy. Normothermia should be maintained as infants can become cold-stressed very quickly. Fever should be avoided as it increases cerebral metabolic demand. Postoperative tachycardia is expected secondary to the stress response, but it can also indicate anemia, hypovolemia, cardiorespiratory distress, fever, or pain. Bradycardia is very concerning in the child and may indicate increased ICP or hypoxemia, which requires immediate evaluation and treatment. The nurse should maintain euvolemia by calculating the intake and output totals from surgery and alerting the physician of indications of hypovolemia. Hypovolemia occurs in the neurosurgery postoperative period due to fluid loss and use of osmotic diuretics. Indicators of hypovolemia include low CVP and signs of low cardiac output, including tachycardia; rapid respirations; cool, pale, or mottled skin; low urine output; decreased mental status; and finally hypotension (Hickey 2009). Administration of fluid boluses and vasopressors may be necessary to prevent and treat hypotension, which is a known cause of secondary brain injury and ischemia resulting in further increased ICP and poor outcome (Zebrack et al. 2009). Cushing's response is a late ominous sign of impending cerebral herniation and includes hypertension, bradycardia, and an irregular respiratory pattern (Greenberg 2010; Dias 2004; Hickey 2009). When appropriate, monitor ICP values, evaluate the ICP waveform, and calculate the CPP at frequent intervals (Vernon-Levett 2006).

7.12.4 Neurologic Function

A full discussion of neurologic assessment and management of increased ICP precedes this section 7.10. The general neurologic assessment includes assessment of level of consciousness and responsiveness (GCS), pupillary and CN assessment, and motor exam. Serial neurologic assessments are completed as ordered and as the child's condition warrants. A general guideline is to assess every 15 min if the child's condition is deteriorating and then every hour and beyond once the child's condition stabilizes (Vernon-Levett 2006).

7.12.5 General Postoperative Nursing Care

Good general postoperative care includes pain management, prevention of infection, nutritional status, and psychosocial support of the child and family. Multiple scales exist for assessment of pediatric pain (see Chap. 1). One such scale should be used consistently to assess pain in the postoperative neurosurgery patient. Pain physiologically increases ICP and should be adequately controlled with administration of analgesics in the post-op period. It is important to note that administration of narcotics and anxiolytics will decrease the patients LOC and responsiveness, blunting the neurologic assessment.

The surgical site should be evaluated for presence of bleeding, dehiscence, wound infection, and CSF leak, all of which should be reported to the neurosurgeon. Bandages should be changed according to institution policy. Enteral nutrition should be started as early as possible to promote wound healing.

7.12.6 Postoperative Complications

Postoperative deterioration, including worsening neurologic status compared to preoperative assessment, requires emergency evaluation and treatment as indicated (Greenberg 2010). Worsening neurologic assessment in the postoperative period

warrants repeat imaging with CT scan to rule out hematoma formation, worsening cerebral edema, and acute hydrocephalus.

7.12.6.1 Intracranial Hypertension

Increased ICP can develop or become worse during neurosurgery or in the postoperative period. Intracranial pressure, ICP waveforms, and calculated CPP values must be monitored and recorded, with deterioration reported to the physician immediately. Cerebrospinal fluid (CSF) can be drained via an EVD, either intermittently or continuously, to therapeutically lower ICP. See previous section 7.10 for full discussion of the pathophysiology, assessment, and management of increased ICP.

7.12.6.2 Seizure

The incidence of seizures following TBI in children is 10 % and can occur during or following neurosurgery. Seizure activity increases cerebral metabolic demand and, therefore, must be prevented or treated immediately. Prophylactic administration of antiepileptic medications is controversial (see Sect. 7.7.8.8) Management of postoperative seizures includes administration of intravenous antiepileptic medications and insertion of an artificial airway and ventilation if the child is unable to protect the airway. A stat CT scan should be performed to rule out postoperative development of a hematoma (Greenberg 2010). Seizure should be considered as a potential cause of increasing or refractory ICP.

7.12.6.3 Complications After Supratentorial Craniotomy

Complications related to supratentorial neurosurgery include hemorrhage, cerebral edema, and cerebral ischemia secondary to increased ICP. Swelling is an expected finding after neurosurgery and peaks within 72 h (Hickey 2009). Postoperative hemorrhage should be considered when there is a rapid increase in ICP and bloody output from an EVD or drain. An unexpected decrease in hemoglobin can indicate intracranial hemorrhage. Presence of coagulopathies in the postoperative period should also be considered as a cause for hemorrhage and managed rapidly. Clinical manifestations of supratentorial complications include symptoms of increased ICP (headache, emesis, decreasing LOC), signs of rostral-caudal deterioration, cranial nerve dysfunction (II – visual deficit; III – ptosis; III, IV, and VI – EOM deficit), focal motor deficits, and seizures. Stat repeat imaging with CT scan is performed. Neurologic deterioration due to an expanding mass lesion requires rapid intubation and control of ventilation, administration of an osmotic diuretic (mannitol), and emergent surgical intervention.

7.12.6.4 Complications After Posterior Fossa Craniotomy

Postoperative complications after posterior fossa (PF), or infratentorial, craniotomy deserve further discussion. Complications include acute bleeding or clot formation, cerebral edema or swelling, and CSF fistula. Often the first indication of posterior fossa complications is respiratory arrest, prompting many neurosurgeons to delay extubation in the postoperative period for 24–48 h (Greenberg 2010). Hypertension after PF craniotomy can cause hemorrhage from tenuous vessels and precipitates postoperative use of antihypertensives. The child's blood pressure should be monitored closely, with any sudden increases reported to the neurosurgeon immediately.

The PF is a physically small compartment, which contains important structures such as the cerebellum and the brainstem. Cerebral swelling or postoperative hematoma with mass effect on the brainstem can be rapidly fatal. Increased pressure in the PF can cause obstruction of the outlet of the fourth ventricle, resulting in acute hydrocephalus (see Chap. 2). Increased pressure in the PF is associated with rapid change in respiratory pattern and hypertension. Headache, lower cranial nerve dysfunction (III to X), and cerebellar findings, such as ataxia, fine motor, or coordination deficits, are common (Greenberg 2010; Hickey 2009). Pupillary reflexes, level of consciousness, and ICP are not affected until late. An immediate reoperation is indicated to prevent cerebral herniation and is often not

delayed to obtain neuroimaging or transport the patient to the operating room (Greenberg 2010).

CSF fistula occurs in 5–17 % of posterior fossa craniotomies and is evidenced by a persistent leak of clear fluid from the wound. CSF can also accumulate beneath the surgical skin flap (pseudomeningocele) without external leak or fistula. Pseudomeningocele can be observed and may resolve spontaneously. CSF fistula or active leak of CSF requires surgical wound revision. Lumbar drainage or placement of a ventricular shunt may be required if the leak is persistent or to promote healing of the surgical wound (Greenberg 2010). CSF leak is a potential source for infection or meningitis, and prophylactic antibiotics are administered.

7.13 Outcomes

Outcomes for children with traumatic brain injury can be difficult to predict. Children tend to have better outcomes, especially as compared to adults. Children with more severe injuries have higher mortality rates (Farrell et al. 2004; Jagannathan et al. 2008). Other factors associated with increased mortality include arriving at the hospital comatose, with a CPP < 40 mmHg, and remaining in that state for at least 6 h (Catala-Temprano et al. 2007; White et al. 2001). It is also known that the presence of hypotension and the number of hypotensive episodes have an effect on outcome (Chiaretti et al. 2002; Coates et al. 2005; Hutchison et al. 2010; Schreiber et al. 2002). Additionally, in one study, the inability to maintain CPP ≥ 50 mmHg during the first 24 h after injury and the presence of bradycardia in the emergency department were factors associated with mortality (Hackbarth et al. 2002). Finally, the child who experiences brain ischemia and hypoperfusion is more likely to have a poor outcome (Kochanek et al. 2000).

Children with TBI tend to have more impairment than those with other injuries (Johnson et al. 2009; Winthrop 2010). Because of this, rehabilitation must begin early in the recovery period, ideally in the intensive care unit. An interdisciplinary team that includes the child's family is necessary to coordinate treatment. Physical, occupational, and speech therapists should participate in the child's care as soon as possible. Families will need anticipatory guidance about potential impairments and disabilities their child may experience, as well as about behaviors the child may display. They will also need to develop strategies to advocate for their child's maximal recovery and ongoing needs (Aitken et al. 2009). Because the child's brain is still developing, it is difficult to predict final functional outcome. Children may experience neurocognitive delays or may never reach milestones related to visual-motor abilities, language, cognition, intelligence and school achievement, and behavior (Keenan and Bratton 2006; Keenan et al. 2007). A structured rehabilitation program will provide the child with the best opportunity to meet his or her potential following a TBI.

7.14 Prevention Efforts

Major prevention efforts have included reduced speed limits, seat belt laws to include a lap and shoulder harness, proper use of child safety seats, and use of safety helmets for bicycling and other activities on wheels. Many local communities and states have passed mandatory bike helmet laws. The difficulty for law enforcement officials becomes how to enforce the law and what, if any, penalty or reward should be placed on the minors and their parents. Nurses should take an active interest in lobbying legislators to continue head trauma prevention efforts and encourage parents to use seat belts and other safety devices. Further, nurses should be advocates for child abuse prevention programs, such as the Period of PURPLE Crying (www.pur plecrying.info or www.dontshake.org).

Pearls

1. Due to their developmental age, young children are more difficult to assess than older children and adults. Young children may have significant intracranial injury with little external evidence, secondary to the more flexible pediatric skull.

2. Accidental head injury is uncommon under the age of 2 years. In the face of inadequate mechanism of injury, or a change in the explanation of injury, assess for inflicted injury.
3. Establish baseline neurologic assessment and communicate clearly and objectively at change of shift. It is critical for the nurse to recognize early signs of neurologic (rostral (head)-caudal (tail)) deterioration, such as worsening LOC, cranial nerve dysfunction, and posturing, so that there is potential to reverse the process before herniation and death occur. Report any and all changes/deteriorations to the physician immediately.
4. Minimal stimulation must not prevent a good assessment and intervention when necessary; base interventions on patient's response, ICP value, and waveform.
5. When evaluating child for causes of increased ICP, assure that the head of bed is elevated 30° with head midline, that adequate sedation and analgesia are provided as ordered, and that the ventilator and EVD/ICP monitor are functioning appropriately, prior to calling physician.
6. A worsening LOC suggests neurologic deterioration. Caution should be exercised not to mistake neurologic deterioration for pain or anxiety, as treatment of the same with narcotics or antianxiety agents will further blunt the neurologic exam and delay treatment. By the same token, abnormal motor posturing should not be mistaken as seizure activity.
7. The classic presentation of an expanding mass lesion is decreased LOC, ipsilateral pupillary dilation, and contralateral hemiparesis.

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