ACQUIRED BRAIN INJURY:

acute management

WHAT IS ACQUIRED BRAIN INJURY?

- ABI is a non-degenerative injury to the brain that has occurred **since birth**, and can be classified into two groups:
- **1. Non-traumatic** – strokes, other vascular accidents, tumours, infectious disease, hypoxia, metabolic disorders and toxic product inhalation/ingestion
- **2. Traumatic brain injury** is the commonest cause of acquired disability in childhood

INCIDENCE

- The prevalence rate for children (0–14 years) admitted to intensive care with ABI was 5.6 per 100 000 per year
- ABI was commonest in low socioeconomic class (overcrowding, decreased supervision, less secure play areas)
- In 65% of admissions ABI was an isolated injury
- There was a significant summer peak in admissions in children under 10 years
- Time of injury peaked late afternoon and early evening,
- Prevalence of ABI is higher in males than females in all age groups, with a 2.5:1 male-to-female ratio

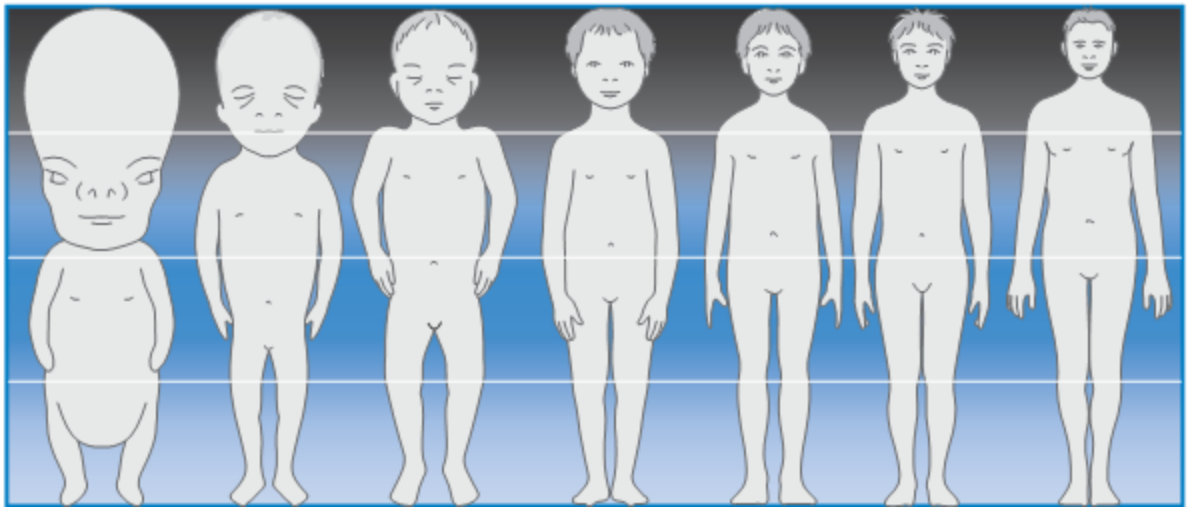
- Children with **premorbid** problems displayed a higher risk of ABI
- Children with existing **behavioural problems** are three times as likely to sustain ABI as compared to those without behavioural problems

CAUSES

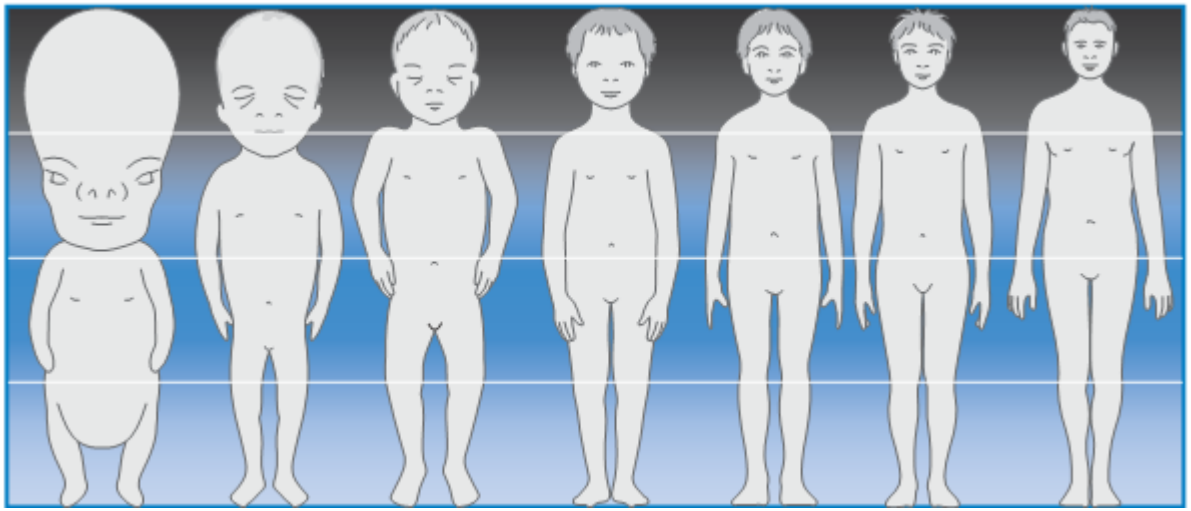
- The commonest mechanism of injury was a pedestrian accident (36%), most often occurring in children over 10
- Injuries involving **motor vehicles** have the highest mortality rates (23% of vehicle occupants, 12% of pedestrians) compared with cyclists (8%) and falls (3%)
- Infants fall from windows, furniture and down stairs, whereas older children fall from trees, roofs and playground equipment
- Cycling injuries account for 20% of all ABI in children
- Non-accidental injury is most common in infants

DEVELOPMENTAL CONSIDERATIONS

- Anatomical differences in the skull, cervical spine, brain and chemistry render the child's brain more susceptible to injury than the adult

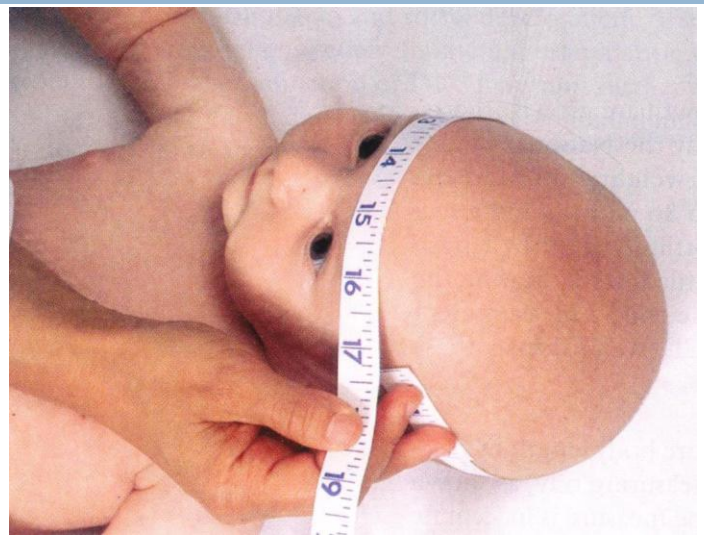


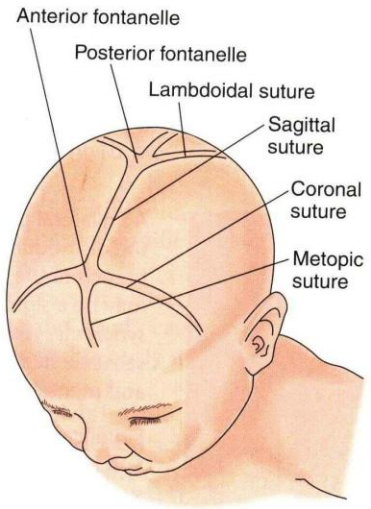
Head-to-torso proportions



It is easy to remember the following average figure.

- 35 cm at birth
- 47 cm (another 12 cm) at 12 months
- 49 cm (another 2 cm) at 2 years
- 50 cm at 3 years
- 52 cm at 6 years
- 53 cm at 10 years
- 56 cm as adult





Height of child

- The toddler’s head is at the level of the motor vehicle front, and isolated severe head injury is subsequently common.

Skull

- Children's skulls are only one-eighth as strong as adults and therefore more vulnerable to injury through deformation and fracture of the skull, leading to brain injury.
- Infants and young children tolerate increased intracranial pressure (ICP) better because open fontanelles and cranial sutures lead to a compliant intracranial space.
- a haemorrhage, is often masked by a compensatory increase in intracranial volume through fontanelles and sutures. Therefore increased ICP signs and symptoms present when the pathology is advanced.
- Mature suture closure occurs by 12 years but completion of fusion continues until the third decade

Brain growth and development rates

- **Skills that are emerging or developing may be affected differently by brain injury than skills that are already established**
- Some neurological deficits may not manifest for years after a head injury, e.g. frontal lobes develop relatively late in a child's growth, so that injury to frontal lobes may not become apparent until the child reaches adolescence as higher-level reasoning develops and social interaction and interpersonal skills are required

Brain water content

- The child’s brain has a higher water content (88%) than the adult brain (77%), meaning that the brain is softer and more prone to acceleration–deceleration injury.

Blood supply

- Cerebral blood flow (CBF) is the amount of blood in transit through the brain at a given point in time.
- A child has a larger percentage of cardiac output directed to the brain, as the head accounts for a **larger percentage of body surface area and blood volume .**
- This can make maintenance of cerebrovascular stability difficult.

Age	Cerebral blood flow (ml/100 g per min) (approximate)
0–6 months	40
3–4 years	108
9 years onwards	71

Pituitary gland

- The pituitary gland can be damaged in moderate to severe ABI. If production of **growth hormone is affected**, hormone therapy may be needed to prevent the long-term ill effects of low pituitary output, which may affect the heart, the psychiatric state of the child and produce sexual dysfunction.

CLASSIFICATION OF HEAD INJURY

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Injuries can be divided into primary and secondary injuries.

- **Primary injury** is due to **direct mechanical damage** inflicted at the time of injury. Except for preventive measures, little can be done to alter primary brain damage which is irreversible . If primary damage is not extensive, outcome becomes dependent upon the management of the secondary damage
- **Secondary injury** is represented **by systemic and intracranial events** that occur in response to primary injury and further contribute to neuronal damage and cell death.

Primary	Secondary
• Intracerebral hemorrhage • Subdural hemorrhage • Subarachnoid hemorrhage • Epidural hemorrhage • Cerebral contusion • Cerebral laceration • Axonal stretch injury	• Edema • Impaired metabolism • Altered cerebral blood flow • Free radical formation • Excitotoxicity

- Recovery from any type of brain injury depends on the extent of the initial injury and the secondary damage
- Injuries are also classified by **mechanism** (closed versus open), **morphology** (fractures, focal intracranial injury and diffuse axonal injury) and **severity** (mild, moderate or severe).

MECHANISMS OF INJURY

- **Open** – also referred to as penetrating injuries; these occur when both the skin and the dura are penetrated by a foreign object (e.g. bullet) or a bone fragment of a fractured skull
- **Closed** – are the most common type; the skin remains intact and there is no penetration of the dura. Closed ABI falls into two categories: focal and diffuse.
- The severity of these injuries varies according to the velocity of impact and the vector (linear versus rotational) of forces applied. History of the accident is utilized to determine the velocity of the injury.

- **Low-velocity injuries** include a child falling a short distance, an accidental blow to the head, e.g. with a bat, or an aggressive hit in a football game. Injuries are usually mild and only require observation.
- **High-velocity injuries** include a fall from an upperstorey window and a pedestrian being struck by a moving car. Even in the absence of neurological dysfunction children are usually observed in hospital.
- **Crushing injuries** may also occur where the head might be caught between two hard objects (e.g. the wheel of a car and the road). This is the least common type of injury, and often damages the base of the skull and nerves of the brainstem rather than the brain itself

MORPHOLOGY OF INJURY

- Skull fractures may be in the cranial vault or skull base and may be linear or stellate, depressed or non-depressed.
- The main significance of finding a fracture is that it is **an indication of the force of the injury**.
- **Depressed skull fractures** are invariably associated with **high-velocity** injury and may result in brain injury. A CT scan is mandatory.
- Basal skull fractures may involve the floor of the skull's brain cavity and are occasionally associated with cerebrospinal fluid (CSF) (leakage of spinal fluid into the nose and ears). Bruising around the eyes, ears and back of neck may also be present

CONTUSIONS

- A bruise of the **brain** tissue. Like bruises in other tissues, cerebral **contusion** can be associated with multiple microhemorrhages, small blood vessel leaks into **brain** tissue.

- There are often two **contusion sites**. One occurs at the impact site – **coup injury** (frontal and temporal lobes are commonly involved). The other arises where the brain bounces off the skull when it has been moved away from the site of the original blow – **contrecoup injury**

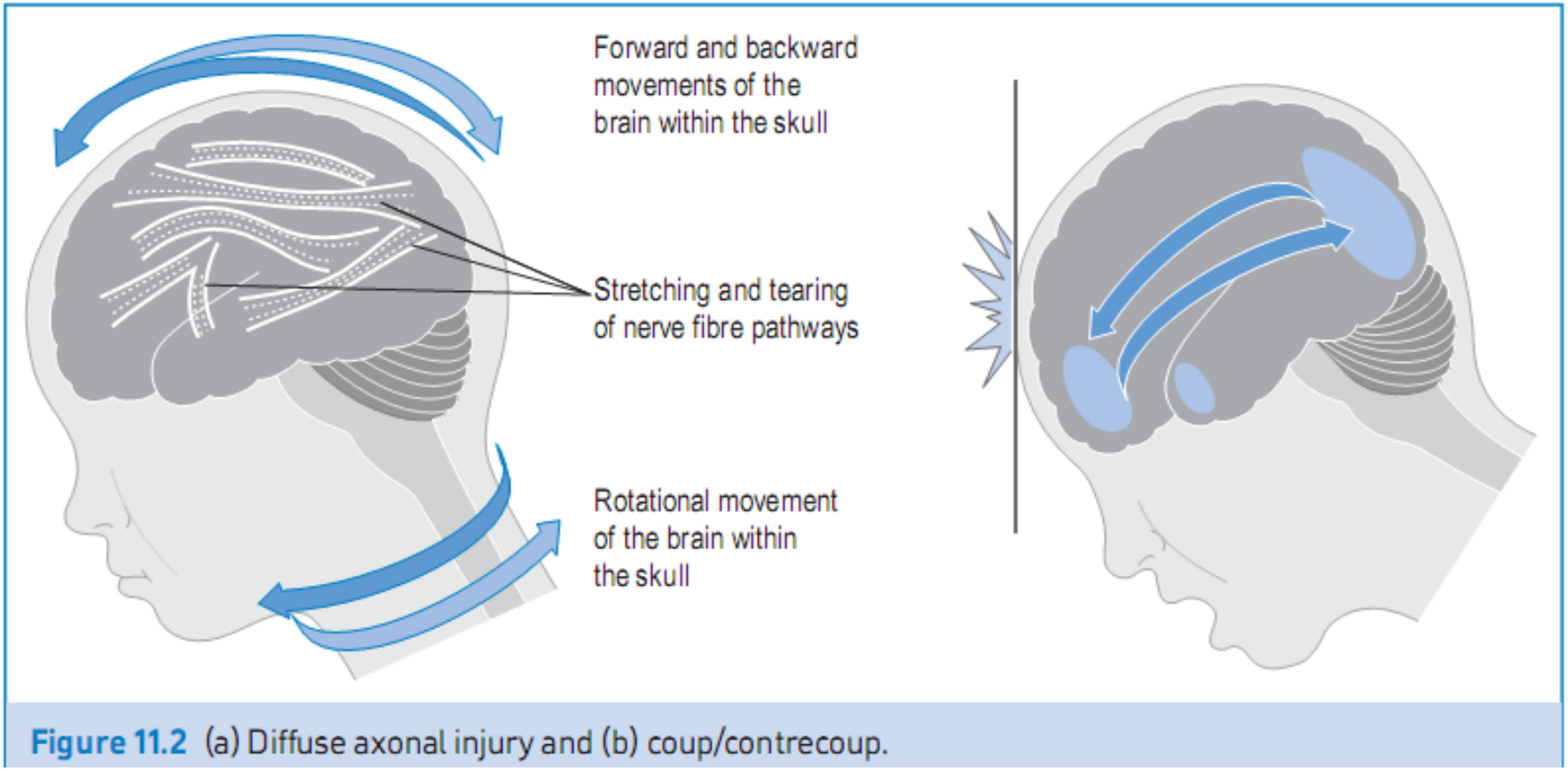
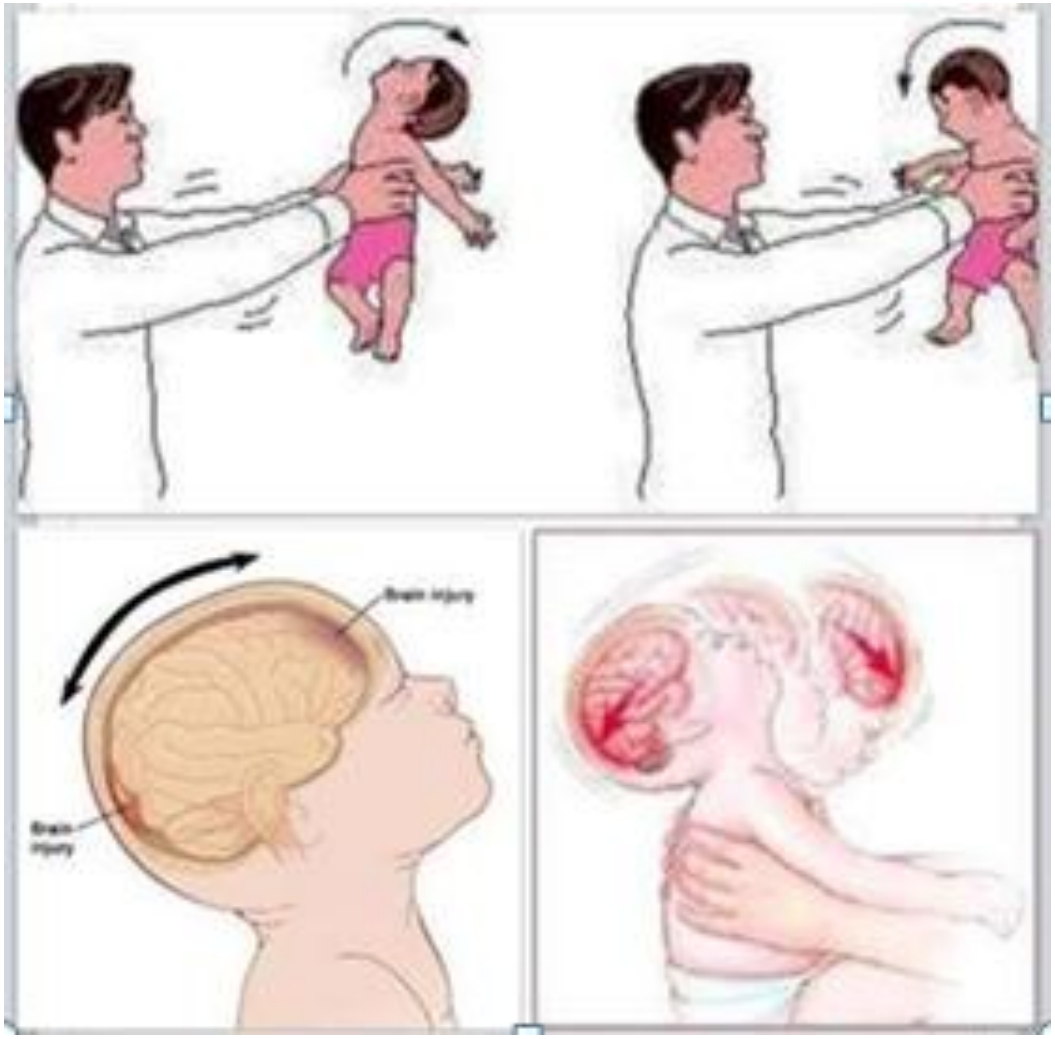


Figure 11.2 (a) Diffuse axonal injury and (b) coup/contrecoup.

SHEARING FORCES

- shearing forces, plays a role primarily in injuries that involve **rapid and forceful movements of the head, e.g.**
- motor vehicle accidents,
- shaken-baby syndrome.
- Rotational forces, associated with rapid acceleration–deceleration of the head,
- The resultant shearing forces cause different levels in the brain to move relative to one another. This movement **produces stretching and tearing** of the axons (diffuse axonal injury)
- insulating myelin sheath, injuries which are the major cause of loss of **consciousness** in a head trauma. Small blood vessels are also damaged, causing bleeding deep in the brain
- Collectively these injuries can result **in swelling of the brain**. If swelling continues the brain will gradually be pushed down through the foramen magnum (coning). Brainstem nuclei controlling breathing and cardiac function will eventually be compressed, resulting in death


Brain injury
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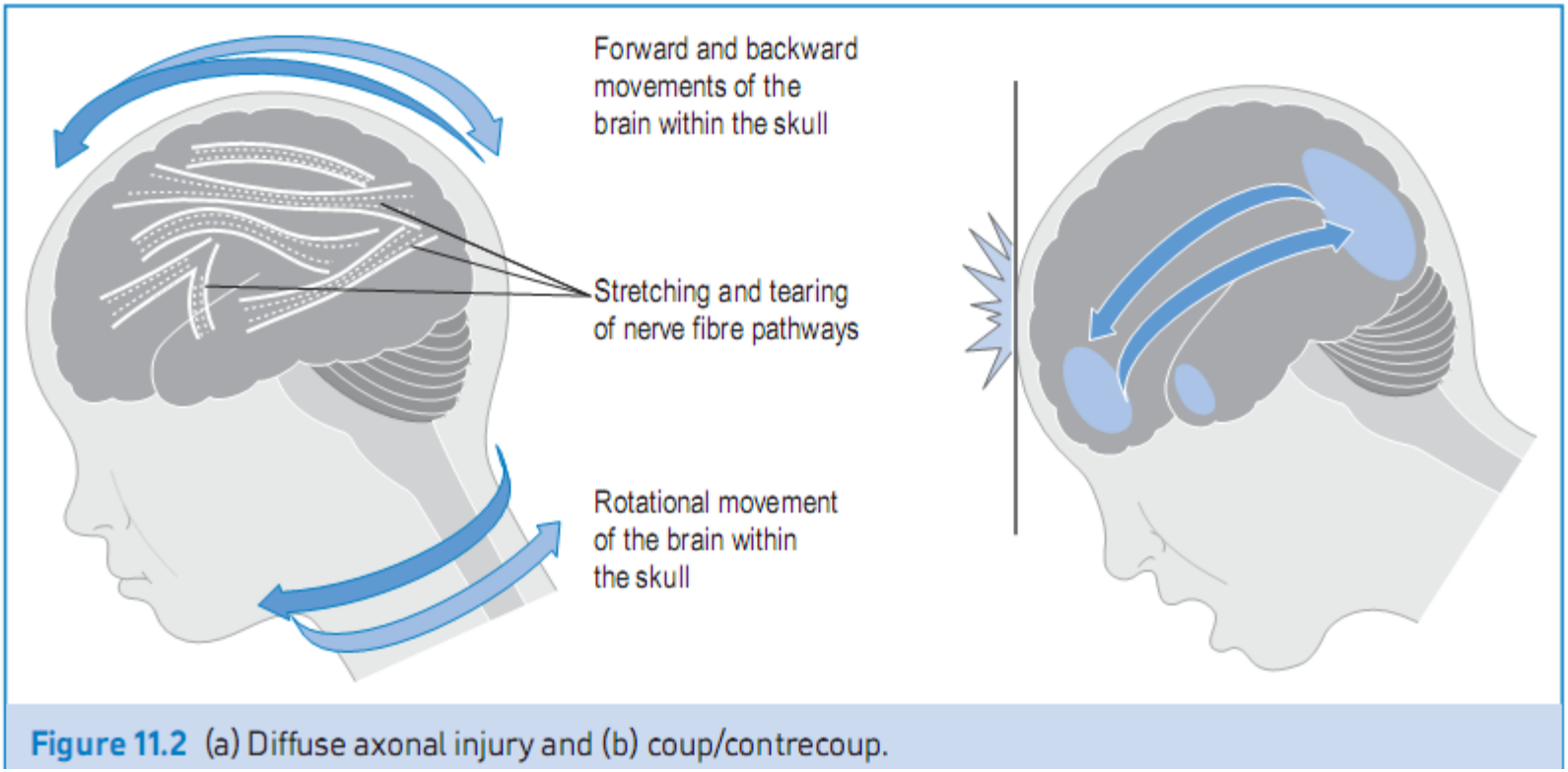


Figure 11.2 (a) Diffuse axonal injury and (b) coup/contrecoup.

SEVERITY OF HEAD INJURY

- The Glasgow Coma Scale (GCS) is the most widely accepted tool to determine severity of head injury.
- It is a simple scale developed in 1974 to assess conscious levels and has a relatively **high degree of interobserver reliability**
- It serves as an immediate prognostic guide and provides a useful baseline with which future examinations can be compared.
- The scale has been adapted for infants and young children – the **Paediatric Coma Scale**

- The GCS is scored between 3 and 15,
- 3 being the worst and 15 the best
- It is composed of three parameters: best eye response, best verbal response and best motor response

Table 11.3 Grading of acquired brain injury (ABI) using the Glasgow Coma Scale (GCS) score

Grade of ABI	GCS score
Mild	13–15
Moderate	9–12
Severe	3–8

Best eye response (E)

- 1. No eye-opening
- 2. Eyes opening to pain
- 3. Eye-opening to verbal command
- 4. Eyes open spontaneously.

Best verbal response (V)

- 1. No vocal response
- 2. Incomprehensible sounds
- 3. Inappropriate words
- 4. Confused
- 5. Oriented

Motor response (M)

- 1. No motor response
- 2. Extension to pain (decerebrate posturing)
- 3. Flexion to pain (decorticate posturing)
- 4. Withdrawal from pain
- 5. Localizing to pain
- 6. Obeys commands

- To be useful the ‘GCS of 12’ needs to be broken down into its components, e.g. E4V3M5 GCS 12
- The GCS has limitations in the assessment of some children, including those who are intubated, dysphasic, have periorbital haematomas and facial swelling and immobilized broken limbs.

INTRACRANIAL DYNAMICS AND AUTOREGULATION

- At present **ICP** and cerebral perfusion pressure **(CPP)** monitoring remain the most commonly used clinical parameters for assessing intracranial dynamics.
- By continuous observation and regulation of the ICP and mean arterial pressure (MAP), CPP can be maintained.

What is intracranial pressure?

- It is the pressure exerted by the volume of the three intracranial components inside the skull:
- **1. Brain tissue: 80%**
- **2. Blood: 10%**
- **3. CSF: 10%**
- Under normal conditions ICP ranges between 0 and10 mmHg, although it will rise transiently with coughing or straining
- with no significant pressure gradient between the two cerebral hemispheres or between supratentorial and infratentorial compartments

- Following trauma, this situation may change.
- When the volume of any of the intracranial components increases, the volume of one or both of the others must decrease or the ICP will rise.
- Normally the brain has the ability to autoregulate its blood flow by dilation and constriction of blood vessels. This ensures a constant blood flow to all areas of the brain.
- Autoregulation is apparently preserved in the majority of head-injured children

Compensatory mechanisms for increased intracranial pressure

- The brain may try to compensate for the increase in one of the intracranial components by shunting CSF to the spinal subarachnoid space, increasing CSF absorption or decreasing CSF production or shunting venous blood out of the skull.
- However, cerebral trauma may disrupt autoregulatory mechanisms and cause a sustained increase of ICP to 15 mmHg or higher
- Monitoring of ICP is important and this is closely linked to the maintenance of an adequate CPP and the importance of normovolaemia.

Clinical effects of increased intracranial pressure

raised ICP will produce signs and symptoms but does not cause neuronal damage provided CBF is maintained

Infants	Children
• Tense, bulging fontanelle • Separated cranial sutures • 'Cracked-pot' sound on skull percussion • Irritability • High-pitched cry • Increased occipitofrontal circumference • Distended scalp veins • Changes in feeding • Cries when held or rocked • 'Setting-sun' sign (impaired upward gaze)	• Headache • Nausea • Vomiting • Diplopia, blurred vision • Seizures • Changes in behaviour and personality **Late signs (all ages)** • Decreased consciousness • Decreased motor and sensory responses • Alteration in pupil size and reactivity (pupillary response changes*) • Decerebrate and cortical posturing • Change in respiration pattern • Papilloedema†

What is cerebral perfusion pressure?

- CPP is the pressure at which the brain tissue is perfused with blood and is a measure of the adequacy of the cercirculation.
- It is maintained by supporting MAP
- Normal CPP in paediatric patients is variable and dependent upon the age-related MAP.
- Monitoring of ICP as a means of calculating CPP is widely used, aiming for a CPP of 50 mmHg in infants under 1 year and 60 mmHg in children above that age

MINIMUM CPP	
Age band (years)	**CPP (mmHg)**
2–6	53
7–10	63
11–16	66

SECONDARY BRAIN DAMAGE

- Cellular events that occur after the primary insult.
- Initial vascular and parenchymal disruption leads to ongoing neuronal degeneration, resulting in neuronal ischaemia and cell death
- **Haematoma**
- **Brain swelling**
- **Brain shift**
- **Ischaemia**
- **Infection.**

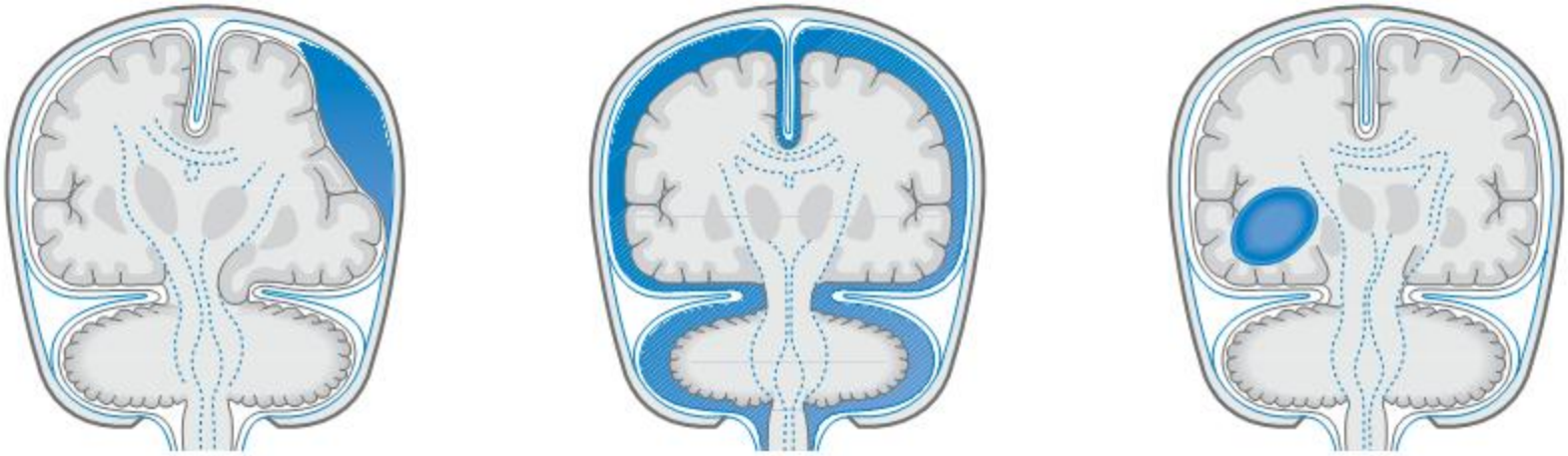
Haematoma

- **Extradural** –

a skull fracture may cause tearing of the middle meningeal vessels, causing a **bleed into the extradural space**. There may be a lucid interval after the injury, then increasing **headache (the dura is painsensitive**) and a subsequent clinical deterioration as the mass lesion increases in size.

- **Intradural** –

which may evolve into a life-threatening mass as bleeding from the torn veins continues, consisting of a mixture of both subdural and intracerebral haematomas. Bridging veins may be ruptured following impact, producing a subdural haematoma. It is usually associated with high-velocity injury with immediate severe neurological dysfunction



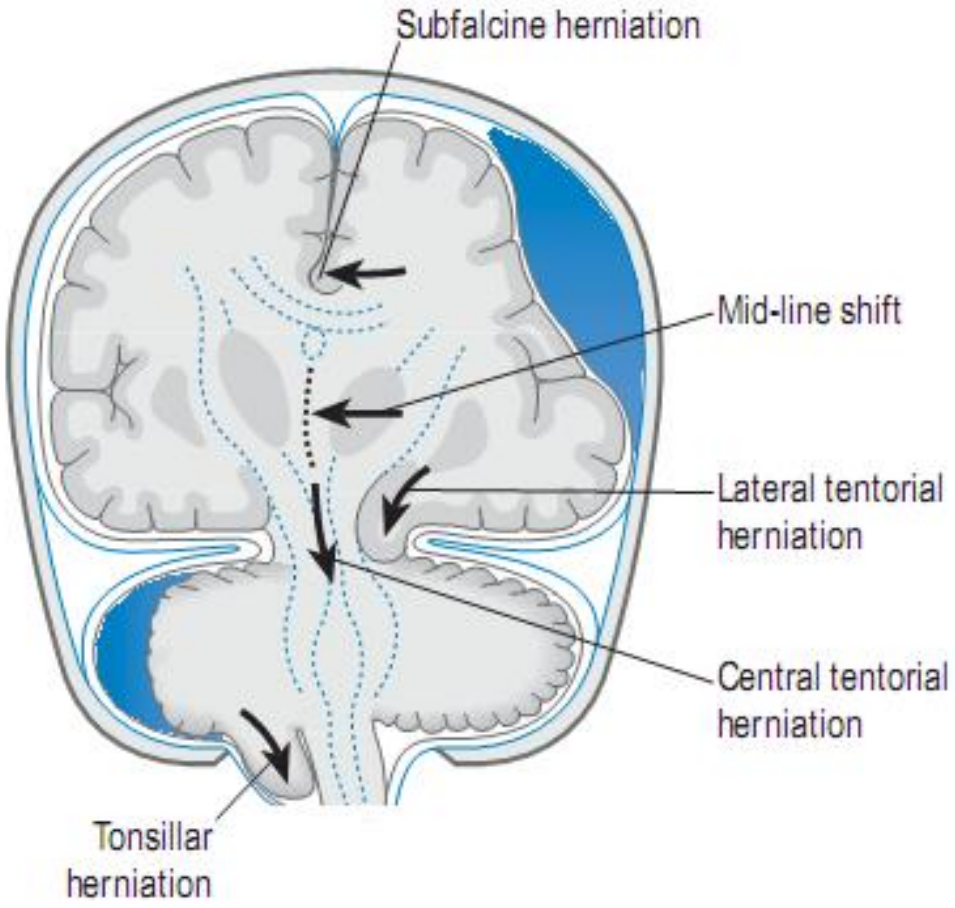
(a) Subdural haematoma

(b) Subarachnoid haemorrhage

(c) Intracerebral haemorrhage

Brain shift

- A progressive rise in ICP due to a supratentorial haematoma at first produces midline shift followed by a series of herniations, causing progressive midbrain and lower brainstem compression .


Subfalcine herniation
Mid-line shift
Lateral tentorial herniation
Central tentorial herniation
Tonsillar herniation

PRIMARY MANAGEMENT AND INTERVENTIONS FOR ACQUIRED BRAIN INJURY

- The first **24–72** hours is the vital period in management of ABI.
- Adequate **oxygen delivery and hemodynamic** stability in the child at the earliest moment remain the most important aspects of the management plan.
- Most patients who survive the first few hours after severe ABI require **several days of intensive care**, and ICP is usually measured during this time .

- The combination of **hypoxia (oxygen saturation less than 90%) and hypotension (systolic blood pressure less than 90 mmHg)** is universally associated with unfavorable outcome.

- Hypotension has been shown to increase mortality rates significantly in children with ABI but isolated ABI rarely leads to hypotension.
- Blunt abdominal trauma and long-bone fractures frequently occur in association with ABI and may be a major source of blood loss.
- A child'sblood volume should be restored with **crystalloid solutions and/or blood products**. These children should undergo aggressive fluid therapy resuscitation with isotonic fluids until appropriate fluid balance is achieved .

- Hypoxia and hypercapnia are both potent vasodilators, resulting in increased CBF and increased ICP. Therefore mechanical ventilation should ideally keep the following levels:

$Sa\mathrm{O}_2$ 92–100%
$Pa\mathrm{O}_2$ 10–14 kPa
$Pa\mathrm{CO}_2$ 4–4.5 kPa

intracranial pressure (ICP)

Intervention	Details
Head midline	Prevents kinking of the jugular veins (Johnson 1999) Cervical collars and endotracheal tube ties not too tight as may impair cerebral venous drainage (Arbour 1998)
Nurse head elevated	15–30° of head elevation is optimal >30° elevation reduces CPP (Dixon & Vyas 1999)
Inotropes	May be indicated to maintain the MAP and CPP
Induced hypothermia	Fever (>38°C) can arise due to hypothalamic dysfunction or infection and decreases seizure threshold (Chambers 1999) Using cooling devices (32–34°C) decreases inflammatory responses, excitoticity, metabolic demands and oxidative stress (Marion et al 1993, Chambers 1999, Johnson 1999) Increases risk of bleeding and infection, arrhythmias and exacerbation of chest infection (Schubert 1995)

Sedation	Barbiturates (heavy sedation) cause a 'barbiturate coma' Decrease cerebral metabolic rate and ICP (Arbour 1998)
Anticonvulsants	Seizures impose major metabolic burden on the brain and increase ICP Used if seizure activity is identified clinically or on EEG (Dixon & Vyas 1999)
Hyperosmolar therapy	Reduces elevated ICP by creating an osmotic gradient that draws cerebral oedema fluid from brain tissue into the circulation Mannitol widely used Hypertonic saline solutions may also be used (Knapp 2005)
Hyperventilation	Decreases Pco_2 to between 4 and 4.5 kPa, producing a reflex cerebral vasoconstriction, therefore decreasing CBF and ICP (Arbour 1998) Effects noted less than 30 seconds from onset, and the peak effect is noted at approximately 8 minutes (Oh 1997)
Positive end-expiratory pressure (PEEP)	Used to maintain airway patency, but PEEP above 10 cmH_20 mmHg may cause pressure decreased intracranial compliance (Dixon & Vyas 1999)
Paralysing agents	Can be given continuously or in intermittent boluses as required Disadvantages of use include masking of seizure activity (Dixon & Vyas 1999), prevention of coughing and decrease in effective secretion clearance (Felice 2005) and development of muscle weakness which may prolong ventilation (Dixon & Vyas 1999)

Analgesia	Reduction of painful stimuli (e.g. tracheal suction, intravenous cannulation)
Craniotomy	Removal of a section of the skull (bone flap) to access the traumatized brain underneath and then it is replaced (Lindsay et al 1986)
Burrhole	A small opening is made and minimally invasive procedures used, e.g. to drain a blood clot (Lindsay et al 1986)

MAP, mean arterial pressure; EEG, electroencephalogram; CBF, cerebral blood flow; PEEP, positive end-expiratory pressure.

DIFFERENT STAGES OF RECOVERY

- 1. Unresponsive/coma stage
- 2. Early responses stage
- 3. Agitated/confused stage
- 4. Higher-level responses stage.

Unresponsive/coma stage

- **Coma is a state of unawareness** of self or environment, and the inability to sense or respond to bodily or environmental needs, **caused by injury to the arousal centre in the brainstem.**
- Coma is caused by **brainstem damage and severe injury to both sides of the cortex**. The brainstem is highly interconnected with other parts of the brain; therefore, when it is injured other parts of the brain are affected as well.
- The **child's eyes remain closed**, he or she is unable to communicate, fails to move in a purposeful manner or respond in a consistent or appropriate manner and a normal sleeping pattern is not re-established.

- The **auditory sense is often present in a state of coma,** therefore discussion about the child's status should be discouraged

stereotyped postures

- Decerebrate posture –
- bilateral upper- and lower-limb extensor posture, usually the consequence of bilateral midbrain or pontine lesions. Opisthotonos, a severe muscle spasm of the neck and back, may accompany decerebrate posture in severe cases

- Decorticate posture –
- bilateral flexion of the upper limbs and extension of the lower limbs, usually the consequence of an upper-brainstem lesion. Although a serious sign, it is usually more favourable than decerebrate posture and may progress to a decerebrate posture, or the two may alternate

- Unilateral decerebrate or decorticate postures can be seen and are an indication of a unilateral lesion. This asymmetry has some localizing value .

- Damage above the brainstem results in flaccidity of muscle tone, is asymmetrical and may be valuable in the localization of structural damage (Bateman 2001). Flaccidity can occur before spasticity appears but may persist indefinitely

- Limb weakness with decreased level of consciousness can be determined by comparing the response in each limb to painful stimuli

- Hemiparesis usually occurs in the limbs contralateral to the side of the injury but may also occur in the ipsilateral limbs.

vegetative state

- A child may start to appear to be ‘more wakeful’, with cycles of eye-opening and closing, but reveals no sign of awareness or wakefulness. This stage is sometimes referred to as the ‘vegetative state’, when the child is breathing spontaneously and has a stable circulation. The shorter the period of coma/vegetative state, the better the prognosis for recovery (Jennett 2002).

coma arousal therapy

- The use of **coma arousal therapy**, also known as sensory stimulation,
- It is intended to promote awakening and enhance rehabilitative potential by using an **intensive programme of visual, auditory, olfactory, gustatory, cutaneous and kinaesthetic sensory input**.
- Sensory stimulation can start as soon as the child's medical condition is stable but there are conflicting views on its efficacy. There is limited reliable evidence to support its use (Lombardi et al 2002) and constant stimulation may even be detrimental (Wood 1991).

Early responses stage

- Children will now be keeping their eyes open for longer and need less vigorous stimulation to wake them up, e.g. initially arouse only to painful stimuli, then touch, then sound.
- Children start to respond to the environment and responses will be more appropriate but may be inconsistent or slow.
- Localized responses, e.g. turn towards a sound, pull away from something uncomfortable, follow with eyes.

- At this stage **communication should be encouraged** but children often experience fatigue and a short attention span. Tips for improving communication include:
- ● Speaking slowly and clearly
- ● Encouraging eye contact if possible
- ● Clarifying names of body parts to help movement requests
- ● Using age-appropriate language
- ● Blinking – once for yes, twice for no. Blinking is an involuntary action so must be done very definitely
- ● Simple commands, e.g. open and shut eyes
- ● Thumbs up and grip and release of hand
- ● Awareness of slow processing and be patient for a response
- ● Hand gestures or physical guidance with hands or with verbal cues.

- At this stage the goal is to increase the consistency of responses.
- **Recording** achievements of recovery, however small, in a diary is a way for all carers to follow the child's progress.
- **Rest periods** are essential throughout the day with decreased stimulation of the surroundings if possible.
- **Methylphenidate** appears to be an effective treatment to improve arousal in the minimally responsive child (Hornyak et al 1997).

Agitated/confused stage

- Children will be responding more consistently at this stage.
- However, they will probably be confused and disoriented in time and place, with memory and behaviour difficulty.

- A consistent approach to inappropriate behaviour is vital from all carers and family, e.g. not laughing at the patient's behaviour or language

- As the child becomes more aroused, it is important for all carers and visitors to speak to the child in an ageappropriate manner. Inappropriate language and actions can lead to inappropriate responses from the patient and may lead to future behavioural problems

- The goal at this stage is to help the child become more oriented and continue to treat the physical needs. The child may be moving about randomly in bed and trying to climb out of bed; padded cot sides can be helpful to prevent injury and/or tissue damage. Elbow and knee pads can also be useful. Children are very vulnerable at this stage, as once out of bed, they can be disoriented, lack safety awareness and have decreased balance reactions. At this stage they require constant supervision. A child may become very frustrated if not allowed to move about and mats on the floor can be useful where the patient can roll about or crawl. Sometimes the presence of a family member can be enough to calm the patient.

- At this stage a child benefits greatly from low noise levels and short periods of activity with hopefully increased attention span times. Orientate the child to their surroundings using visual and verbal information. Clocks, calendars and diaries can be useful to write a child's daily schedule of timetable, mealtimes, therapy input, visitors and special appointments.

Higher-level responses stage

- Routine tasks become easier but help is needed with problem-solving and making judgements and decisions. Children will have become aware of any residual physical problems and conscious of their body image. The goal at this stage is to decrease the amount of supervision needed and increase their independence

THE PHYSIOTHERAPIST'S ROLE FOR CHILDREN WITH ACQUIRED BRAIN INJURY

- ● Prevention of secondary respiratory problems, primarily in intensive care but also after transfer to the rehabilitation ward
- ● Prevention of secondary soft-tissue shortening and joint contractures

- During the acute management of ABI, physiotherapy intervention is known to raise ICP, therefore:
- ● Short, more frequent and efficient treatments are essential
- ● Allow time for acute increased ICP to recover to acceptable values (15–20 mmHg) both between general procedures, e.g. position change, and in individual treatments (Johnson 1999)
- ● Monitor the child's reactions to different procedures and modify or avoid them accordingly.
- An ICP of more than 20 mmHg for longer than 3 minutes requires immediate medical intervention. Elevations that return to baseline within 30 seconds are usually well tolerated (Prasad & Tasker 1990).

Prevention of secondary respiratory problems

- Children with ABI who present with a GCS 8 are likely to have impaired respiratory function at the time of the injury or later on.
- There may be direct damage to the chest wall or fractured ribs, or lung damage in the form of contusions.
- The child may have vomited and aspirated at the time of the injury.
- Any damage to any part of the respiratory system may potentially lead to hypoxia and hypercapnia which ultimately leads to cerebral oxygenation problems

Prevention of secondary soft-tissue shortening and joint contractures

- ● Passive stretches/movement
- ● Positioning and postural management
- ● Serial casting.

Positioning and postural management

Table 11.7 Physiotherapy interventions and effect on intracranial pressure (ICP)

Physiotherapy intervention	Details
Positioning	Although patients with raised ICP are nursed with head elevation of 30°, postural drainage can be performed if needed when strict guidelines are followed (Imle et al 1997)
Changing position	Changes in head and body positioning can increase ICP (Chudley 1994) Log-rolling maintains head in relation to the body Side-lying may increase ICP with only small changes in CPP (Rising 1993) Hip flexion of >90° limits venous drainage and increases ICP (Arbour 1998)
Manual techniques	Percussion performed slowly does not increase ICP and may even lower it Shakes may increase ICP over time, whereas vibrations done in isolation have no effect on ICP (Imle et al 1997)
Manual hyperinflation	Known to increase ICP (Imle et al 1997) Should be interspersed with short-duration hyperventilation to decrease Pco_2 and ICP, prior to or following suction (Kerr et al 1997)
Suctioning	Causes a progressive rise in ICP with each insertion of the catheter. Elevations in ICP are transient and return to baseline levels within minutes (Kerr et al 1998) Stimulation of the cough due to the direct tracheal stimulation causes a rise in ITP, decreased cerebral venous return, increased CBV and ICP (Kerr et al 1998) Hypoxia can be minimized by use of closed-suction circuits and hyperoxygenation (Johnson 1999)

CPP, cerebral perfusion pressure; CBV, cerebral blood volume; ITP, intrathoracic pressure.

Table 11.8 Key deformities in acquired brain injury

Joint	Most common deformity
Shoulder	Adducted/internally rotated
Elbow	Flexed (decorticate) Extended (decerebrate)
Forearm	Pronated
Wrist	Flexed/ulnar-deviated
Fingers	Flexed
Thumb	Adducted/flexed into palm
Hip	Adducted/medially rotated Extended if mass extensor tone very high, or held flexed
Knee	Extended if decorticate/ decerebrate posturing present Sometimes flexed
Ankles/subtalar	Plantarflexed/inverted (equinovarus)
Great toe	Flexed/extended

ABI REHABILITATIONS

THANKS

QUESTIONS?