Traumatic injuries to the head and spine 2: nursing considerations

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Cerebral blood flow

Intracranial hypertension leads to a decrease in cerebral blood flow (CBF) because blood flow is impeded as it attempts to flow through compressed blood vessels (Brinker et al., 1992). This can lead to a reduction in cerebral oxygen delivery (Cruz et al., 1993) and ultimately cerebral ischemia can develop. Normal CBF is 50 ml/min/100g of brain (or 750 ml/min in a 1500 g brain) (Hickey, 2002), and the brain has the ability to autoregulate the blood flow through it in order to maintain steady flow (Vander et al., 2000). This is usually in response to the cerebral perfusion pressure (CPP), or blood pressure gradient, across the brain. CPP is the difference between systemic mean arterial pressure (MAP) and the ICP, and is usually about 80–100 mmHg, with 60 mmHg being required for neuronal perfusion (Hickey, 2002).

Autoregulation is responsible for ensuring CBF is relatively stable, despite changes in systemic blood pressure (Vander et al., 2000). Thus, if the systemic blood pressure increases, the cerebral blood vessels will constrict; if the pressure should drop, the vessels will dilate. Autoregulation can only occur within a certain range of systemic mean arterial blood pressure (50–150 mmHg); outside this range, CBF becomes pressure-passive as it alters according to the MAP (Oh, 1997) (Figure 3), therefore if the MAP decreases, so does the CBF.

There is no totally reliable method of measuring CBF. Transcranial Doppler can be used to measure the blood flow through the middle cerebral artery and internal jugular bulb catheters can be used to measure the oxygen content of the blood leaving the brain (which will give an indication of oxygen extraction) (Oh, 1997). ICP monitoring and calculation of CPP is the usual method for estimating cerebral perfusion (Hickey, 2002), but CPP does not take into account the cerebral vascular resistance which will alter CBF. When the influences on CBF are taken into consideration, then the variables that can be manipulated in patient care can be identified. CBF can be calculated as shown below (Muizelaar and Schroder, 1994):

$$\text{CBF} = \frac{\text{CPP} \times d^4}{8 \times \text{Ivx}}$$

where $K = \text{constant}$, $l = \text{length of blood vessels}$, $d = \text{diameter of blood vessels}$, $v = \text{blood viscosity}$, CPP = cerebral perfusion pressure.

The CPP equation is CPP = MAP – ICP.

The variables that can be manipulated are the MAP and ICP and (therefore the CPP), the diameter of the cerebral blood vessels and the blood viscosity. Of these, the blood vessel diameter has the most effect on CBF and therefore oxygen delivery to the brain (Muizelaar and Schroder, 1994).

Cerebral blood flow and respiratory gases

Alterations in respiratory gases can markedly affect cerebral blood vessels and, therefore, CBF (Hickey, 2002).

Carbon dioxide: Vascular smooth muscle is affected by hydrogen ion concentration, in that an increase in hydrogen ion concentration (and lowering of pH) causes the smooth muscle to relax which results in vasodilatation (Hudak et al., 1998). This will increase CBF and also ICP according to the modified Monro-Kellie doctrine. Hydrogen ions are a byproduct of carbon dioxide metabolism so that any hypercarbia will increase the hydrogen ion concentration and can lead to cerebral vasodilatation (Yoshihara et al., 1995). In view of this, carbon dioxide is viewed as one of the main influencing factors on CBF and ICP (Figure 4).

Oxygen: If the oxygen content of blood falls, the CBF increases in an effort to maintain cerebral oxygenation (Figure 5). This too will increase ICP (Price et al., 2003a).

Cerebral blood flow and pH alterations

Changes in the arterial pH affect CBF in a similar manner to carbon dioxide. Therefore, if the pH drops (i.e. the blood becomes more acidic), relaxation of the smooth muscle in the cerebral blood vessels occurs. This increases CBF and, therefore, potentially ICP (Hudak et al., 1998).

Care strategies

As the maintenance of CBF is the focus of care, then strategies that control the factors that influence CBF form the basis of patient management in conjunction with accurately observing for any deterioration.

Neurological

The patient’s neurological status needs to be closely observed and any deterioration in level of consciousness promptly acted upon (Hickey, 2002). The Glasgow Coma Scale is a commonly used tool for assessment of level of consciousness that assesses the patient’s awareness of the environment and him/herself (Teasdale and Jennett, 1974). Three responses are assessed: eye opening, verbal response and motor response. The best score a patient can have is 15 out of 15; any score less than 8 is viewed as being unconscious and, therefore, the patient will need to be reviewed by an anaesthetist for possible intubation to ensure a patent airway is maintained (Hickey, 2002).

At the time of neurological assessment, pupillary response to light should also be documented. This gives information about
the functioning of the third cranial nerve (oculomotor nerve), which arises from near the top of the brainstem. Dysfunction of the third cranial nerve, as indicated by dilated and/or a sluggish (or absent) response to light, implies that pressure is compressing the nerve (Hickey, 2002). Therefore, the brainstem is becoming compressed and if that pressure is not relieved, ischaemic damage can occur. This may lead to brainstem death (Lindsay et al., 1998). As the cranial nerves do not cross over, dilation of one pupil indicates that pressure is building up on the same side as the dilated pupil (Hickey, 2002).

Should the patient's level of consciousness deteriorate abruptly, the osmotic diuretic mannitol may be prescribed. This does not enter the brain and so draws water out of the brain helping to reduce ICP (Jastremski, 1998). However, when the blood–brain barrier is not intact, mannitol diffuses into the brain tissue and therefore takes water into the brain with it (Fisher, 1997). Mannitol can cause hypotension as well as electrolyte imbalances, e.g. hypokalaemia (Price et al., 2003a). If a patient has been given mannitol, it is also advisable to measure serum osmolality as this can give an indication of the patient's overall state of hydration and, therefore, whether further mannitol would be of use (Hickey, 2002).

Seizures may occur after a head injury; this can result from the brain injury itself or from hypoxia/hypotension (Price et al., 2003a). Seizures can worsen the secondary brain injury that develops after a severe head injury as cerebral hypoxia and ischaemia can be exacerbated (Adam and Osbourne, 1997). Seizures must be controlled with anticonvulsants, such as phenytoin, and therapeutic levels achieved (Price et al., 2003b).

Respiratory system

The control of respiratory gases is an essential part of acute head injury management because of the effect they have on CBF and, therefore, cerebral perfusion. Mechanical ventilation may be required to control the partial pressures of oxygen (pO₂) and carbon dioxide (pCO₂) (Hickey, 2002). If a patient is intubated and mechanically ventilated, admission to the intensive care setting is essential (Hillman and Bishop, 1996). Hypoxia should be avoided and a pO₂ within normal limits (11–13 kPa) should be the aim (Adam and Osbourne, 1997); pCO₂ needs to be carefully controlled and if the patient is mechanically ventilated, a pCO₂ that is towards the low end of normal is generally the ideal. Therefore, a pCO₂ of 4.5–5 kPa and a normal pH (7.35–7.45) is required (Adam and Osbourne, 1997; Wright, 1999; Wong, 2000).

Endotracheal suctioning (ETS) can cause ICP to rise (Johnson, 1999) as well as causing transient hypoxia. Additionally, coughing can lead to sharp increases in ICP (Johnson, 1999). However, pulmonary secretions can lead to hypoxaemia and hypercapnia (increased pCO₂), which will also have a potentially deleterious effect on cerebral perfusion (Johnson, 1999). Therefore, the decision to perform ETS should always be based on individual patient indications. Parsons and Shogan (1984) studied the effect of ETS and manual hyperinflation on patients who had a severe head injury. They found that ETS could be safely performed on this group of patients if their baseline ICP was <20 mmHg and their CPP was >50 mmHg. Even so, Pendergast (1994) recommends that nurses limit suctioning to one or two suction passes, with each pass being for a maximum of 10 seconds. Preoxygenation with 100% oxygen and optimal sedation/analgesia can help to avoid the complications associated with suctioning (Hall, 1997).

Cardiovascular system

One of the components that influences CBF is the CPP. Therefore, the control of the MAP is an essential part of caring for the patient with a cerebral injury. The MAP should be sufficient to maintain a CPP of >70 mmHg (Hickey, 2002). This may require a MAP of 85–90 mmHg (Eisenhart, 1994; Young and Meredith, 1995). To enable this, it is likely that the patient will require adequate fluid resuscitation that can be guided by central venous pressure readings. If necessary, vasoactive drugs, such as noradrenaline or dobutamine, may be required to support pharmacologically the MAP (Oh, 1997).

The thickness, or viscosity, of blood is also a factor that influences CBF. The administration of colloid solutions may reduce the viscosity of the blood by haemodilution (Oh, 1997). The extent of this can be ascertained by measuring the haemocrit, normally about 45–50% (Oh, 1997). This can be reduced to 30–33% during colloid therapy without having a negative effect on oxygen delivery, which may be reduced during haemodilution (Muirzela and Schroder, 1994).

Hydration and nutrition

Generally, patients with a severe head injury require about 80% of their normal fluid requirements in order to ensure adequate hydration but not to the point of precipitating cerebral oedema. Therefore, a fluid intake of about 100–125 ml/hr is advocated (Oh, 1997). Sodium chloride 0.9% is the intravenous fluid of choice to ensure extracellular electrolytes are replaced rather than causing cellular rehydration as is seen with the use of dextrose-containing solutions (Hudak et al., 1998). The dextrose-containing solutions contain free water, which then can exacerbate cerebral oedema (Oh, 1997). Enteral nutrition, if the patient is able to eat normally, should be commenced as soon as possible so that nutritional requirements are met. This is supported by the findings of a systematic review of nutrition after head injury in which early enteral nutrition was associated with better patient survival and outcome (Yanagawa et al., 2004). However, in any patient with head trauma an orogastric rather than a nasogastric tube should be used so that passage of the tube into the brain via a base of skull fracture is avoided (Greaves et al., 2001).

Blood glucose should be maintained within normal limits (Wong, 2000) as it is metabolized to carbon dioxide and water, and an excess of these could increase ICP as cerebral oedema will worsen (Woodrow, 2000). Additionally, normal electrolyte levels should be maintained, especially of sodium. A slightly elevated sodium (e.g. 147 mmol/l) is acceptable, but hypernatraemia is associated with dehydration and sluggish CBF; whereas hyponatraemia is associated with overhydration and cerebral oedema — both of these situations should be avoided (Hickey, 2002).

Excretion

A normal urine output greater than 0.5 ml/kg/hr should be maintained as this demonstrates normal renal function (Oh, 1997). Patients with a traumatic head injury may develop neuroendocrine disorders as a consequence of the head injury.
Therefore, diabetes insipidus (DI) may be observed, which can result from damage to the hypothalamus which controls the pituitary gland (Hickey, 2002). Normally, the posterior lobe of the pituitary secretes antidiuretic hormone (ADH) in response to osmotic stimuli. However, in DI, ADH is not released and therefore large volumes of very dilute urine are passed (Table 1). If left untreated, the patient can become dehydrated very rapidly and sodium levels can become elevated. This can lead to sluggish CBF and a risk of ischaemia. DI is treated by administering desmopressin, which is an analogue of ADH, thereby promoting the reabsorption of water in the collecting duct of the renal tubules (Oh, 1997).

Constipation should be avoided as the increase in intra-abdominal pressure can increase ICP because the raised intra-abdominal pressure increases intrathoracic pressure which in turn increases ICP (Hickey, 2002). Straining at stool will also increase ICP (Hickey, 2002). Stool softeners should be administered to prevent constipation and to avoid the use of enemas which may increase intra-abdominal pressure (and therefore ICP) (Hickey, 2002).

**Table 1. Diabetes insipidus**

<table>
<thead>
<tr>
<th>Description</th>
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<tr>
<td>Urine output &gt;3 ml/kg for 2 hours</td>
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<tr>
<td>Hypotonic urine (specific gravity 1000–1005)</td>
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<td>Urine osmolality inappropriately low compared with serum osmolality</td>
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**Source:** Oh (1997)

**Temperature regulation**

Hyperthermia in patients with a severe head injury has been shown to increase the cerebral metabolic rate, thereby increasing the demand of the brain for oxygen and glucose (Hickey, 2002). This increases the cerebral blood volume and hence ICP (Johnson, 1999). Studies into the benefits of induced hypothermia have been inconclusive so this cannot be recommended but active warming should be discouraged (Sertic, 2001).

Hyperpyrexia should be controlled with antipyretic agents, such as paracetamol, which 'reset' the hypothalamus and, therefore, induce normal physiological cooling (such as sweating) (Johnson, 1999) and passive cooling methods, such as tepid sponging, which can promote heat conduction in hyperthermia (Johnson, 1999). Price et al (2003b) compared various methods of cooling such as paracetamol, ice packs and cooling blankets. Their findings were inconclusive but they did conclude that paracetamol might be of use in managing hyperpyrexia in patients with an intact thermoregulatory response. One risk of cooling patients is that they may shiver — this should be avoided as it generates more heat and increases metabolic rate, thereby potentially increasing ICP (Johnson, 1999).

**Positioning and mobility**

An essential component of ICP management is to promote cerebral venous drainage. Therefore, head and neck alignment should be maintained (Williams and Coyne, 1993) and it may be of benefit to elevate patients' heads by 30° (Hickey, 2002). This could decrease the patient's MAP and, therefore, CPP. Increases in intra-abdominal pressure can also increase ICP, so it is recommended that patients' legs are not bent more than an angle of 90° at the hip when they are lying on their side (Hickey, 2002). Additionally, optimal sedation/analgesia during repositioning can prevent a rise in ICP (Hickey, 2002).

In view of passive movements, changes in ICP during passive limb exercises seem to be small (Price et al, 2003a). Studies have found no significant rise in ICP during limb movements (Koch et al, 1996; Brunnouette et al, 1997). However, it is unclear whether there is a cumulative effect if movements are performed at the same time as other activities and it is unclear whether passive limb exercises have a therapeutic benefit (Halbertsma et al, 1999).

**Communication and the environment**

Communication and environmental stimuli have both been shown to alter ICP and may be a positive influence (Price et al, 2003a). Common stressors, such as excessive noise, painful procedures, unnecessary lighting and an unfamiliar environment, can all increase ICP (Chudley, 1994; Hall, 1997; Jastremski, 1998), but the impact of these can be minimized by nurses recognizing the negative effect and controlling these environmental factors, including pain. Relatives/loved ones can also influence ICP, with some evidence suggesting that they reduce ICP (Hendrickson, 1987; Allan, 1989; Chudley, 1994). However, in a small study (n = 12) Trecha et al (1991) found that there was no statistical significance between familiar and unfamiliar voices on the ICP of comatose patients. Waldeck (1983) found that stroking patients’ cheeks reduced their ICP (25 out of 30 patients) but their ICP did not change when their hand was touched. This may indicate that patients, even when comatose, respond to a sense of comfort and reassurance.

**Nursing interventions**

The timing and frequency of nursing interventions have been shown to influence ICP (Hickey, 2002). There is no consensus as to whether nursing activities should be clustered together or spread over a period of time; however, there is agreement that response to nursing interventions should be assessed on an individual patient basis, with the aim of avoiding prolonged increases in ICP (Hickey, 2002).

**Spinal injuries**

The aim of care in the management of patients who have sustained a spinal injury is to control the development of secondary damage, to optimize stability of the spinal cord and to commence rehabilitation as soon as possible (Grundy and Swan, 2002). Much of the care required by the patient with a spinal injury is supportive in nature; therefore, complications need to be identified and managed accordingly (Hickey, 2002). If possible, the patient should be referred to a specialist spinal centre as early as possible for specialist nursing and multidisciplinary team input (Grundy and Swan, 2002). If the patient has to wait for a bed, the referring hospital should commence establishing the specialist centre's protocols and referrals, especially in relation to bowel and bladder management.

**Immobilization**

The cervical spine is the most mobile part of the spinal column and immobilization of this area is essential for any patient who has been involved in, for example, a road traffic accident (Greaves et al, 2001). Immobilization of the cervical spine is crucial in any
patient where a neck injury is suspected, to help prevent any further damage to the cervical spine through movement. Cervical spine immobilization can be achieved through the use of a rigid cervical collar and sand bags can be placed on either side of the head (Chiles and Cooper, 1996). The thoracic spine has considerable intrinsic stability because of the rib cage and limited mobility of this area. However, the thoracolumbar junction (T11–L2) is a point of mobility and is the second most common site of spinal injury (Chiles and Cooper, 1996). Injuries to this area of the spinal column should be considered as being unstable, with patients at risk of further damage to the spinal cord.

Logrolling, which is turning the patient in complete alignment, helps to prevent further damage to the spinal cord, irrespective of what anatomical level is at risk by helping to prevent flexion or extension of the spine (Grundy and Swain, 2002). It requires four people to perform the turn and usually a fifth to, for example, inspect the back/changes sheets (Table 2; Figure 6). The staff member who has responsibility to maintain the spine stabilization of the head and neck acts as the team leader and coordinates the roll. With logrolling, it is essential that clear instructions are given and everyone is aware of their role so that the logroll is performed smoothly and safely in a coordinated manoeuvre (Hickey, 2002).

Patients with an unstable spinal injury should be nursed on a specialized spinal bed (Hickey, 2002). If this is not available, a normal bed can be used but the mattress must be firm and not a pressure-relieving mattress that uses fluctuating pressure in air sacs. These mattresses can cause instability in the spinal cord because of differing pressures and support being placed through the spine (Zejdlík, 1992).

Stabilization of the spine can either be achieved through conservative management or surgical intervention (Grundy and Swain, 2002). Conservative management usually consists of bedrest for a period of time allowing the vertebral column to heal.

Cervical spine

Patients with cervical spine injuries may initially need spinal traction to reduce any fractures or dislocations, to relieve pressure on the spinal cord and to splint the spine. This may require skull clamps and weights, which are gradually reduced. A neck roll should be placed under the neck to maintain normal lordosis, or curvature, of the spine (Grundy and Swain, 2002). Pressure ulcers on the patient's occipital region can develop so this area needs to be cushioned when the patient is positioned. Skull traction will be needed for at least 6 weeks, if surgery is not performed (Grundy and Swain, 2002). The decision about when to operate will depend on the available expertise and facilities, but this may be undertaken in the district general hospital rather than a spinal injuries unit.

Neck stability is assessed with frequent X-rays and at 6 weeks will show whether there is any bony union if fractures were sustained. Once stability has been achieved, the patient can gradually start to sit up, with the use of a hard cervical collar, such as a Miami or Philadelphia collar. Patients with high cervical injuries may have postural hypotension when first mobilized and this is owing to alterations in their autonomic nervous system function (Grundy and Swain, 2002). The use of antiembolic stockings and an abdominal binder can help to prevent pooling of blood.

Halo traction, which is a form of skull traction, can be used as an alternative to skull clamps, once any fractures or dislocations have been reduced. It provides stability and allows for earlier mobilization. It will need to be in place for approximately 12 weeks (Grundy and Swain, 2002).

Thoracic injuries

The thoracic spine is relatively stable because of the articulation with the rib cage. The majority of patients who have a thoracic spinal cord injury are managed conservatively by 6–8 weeks of bedrest (Grundy and Swain, 2002).

Thoracolumbar and lumbar injuries

Patients with a thoracolumbar injury are generally managed conservatively with 6–8 weeks’ bedrest, after which mobilization can commence with the use of a spinal brace (Grundy and Swain, 2002). During the period of bedrest normal spinal curvature needs to be maintained through placing a pillow under the lumbar spine (Figure 7).

Pharmacological treatment

Within 8 hours of the injury, high-dose methylprednisolone should be administered, which has been found to improve maximal motor function over the long term (Bracken et al, 1992). If this is not started within 8 hours, administering steroids over an extended period of 48 hours may be of benefit (Bracken et al, 1997). The regimen is: 30 mg/kg bolus over 15 minutes immediately; 5.4 mg/kg/hr over 23 hours (commenced 45 minutes after initial bolus) (Greaves et al, 2001).

Respiratory system

Patients with a cervical spine injury involving C1–4 will require intubation and mechanical ventilation, as the nerve impulses to the respiratory muscles will be affected by the injury (Hudzik et al, 1998). These patients should have also early tracheostomy.

Table 2. Positions in logrolling

| 1st person | Maintains head–neck alignment |
| 2nd person | Coordinates turn |
| 3rd person | Holds patient’s shoulder with one hand and places the other on the patient’s waist/pelvis |
| 4th person | Holds pelvis with one hand and places the other under the patient’s opposite thigh |
| 5th person | Places both arms under the patient’s opposite lower leg and supports it during the turn |

Source: Greaves et al (2001)

Figure 6. Positions in logrolling (Grundy and Swain, 2002).

Figure 7. Support for patients with a thoracolumbar spinal injury (Grundy and Swain, 2002).
placement, as it is likely that they will require mechanical ventilatory support permanently (Hudak et al., 1998). Patients who have a spinal injury at C5–7 may develop respiratory insufficiency as spinal oedema increases (Hudak et al., 1998). The requirement for mechanical ventilation in these patients can be assessed by measuring vital capacity, which is an indicator of respiratory muscle strength. Generally, this will deteriorate before arterial blood gas alterations, and a vital capacity of \(<15–20\,\text{ml/kg}\) would normally indicate the need for mechanical ventilatory support (Greaves et al., 2001).

Complications associated with poor respiratory function, such as chest infections and lobar collapse, need to be avoided as they will worsen an already poor gas exchange (Hickey, 2002). Judicious chest care needs to be performed and chest infections treated. This will require close coordination of the multidisciplinary team and patient education regarding assisted coughing (Grundy and Swain, 2002). Assisted cough is when a nurse or carer supports the diaphragm during coughing in order to elicit a stronger cough. Additionally, clinical signs of a chest infection need to be assessed including respiratory rate, oxygen saturations and chest auscultation.

**Cardiovascular system**

After a spinal injury, neurogenic shock can develop. Injuries at or above T6 are associated with loss of sympathetic autonomic outflow (Greaves et al., 2001). Therefore, vasomotor tone is reduced and sympathetic innervation of the heart can be reduced if the spinal injury is high enough. The reduction in vasomotor tone results in profound hypotension because of peripheral vasodilation and the loss of sympathetic innervation to the heart leads to significant bradycardia (Hudak et al., 1998). The onset of neurogenic shock can be minutes or several hours after the injury occurred.

Management of neurogenic shock is aimed at re-establishing vasomotor tone until the shock resolves. This normally requires the use of vasopressors such as noradrenaline (Oh, 1997). Bradycardias can be treated with, for example, isoprenaline (Hudak et al., 1998). Careful fluid management is required as pulmonary oedema could be precipitated if large volumes of fluids have been used once vasomotor tone returns (Oh, 1997).

**Autonomic dysreflexia**

This is a serious potential complication that all patients with an injury that is at T6 or above may experience. It is created by an abrupt and excessive discharge from the sympathetic nervous system, leading to hypertension, cardiac rhythm changes, severe headache and profuse sweating (Gardner and Klugger, 2004), as well as flushing or blotchiness of the skin above the level of the injury (Grundy and Swain, 2002). It can be caused by any stimuli arising from below the level of the injury, including constipation, a blocked urethral catheter, ingrowing toenails and pressure ulcers. Management of autonomic dysreflexia includes identification and removal of the cause, sitting the patient upright and administering vasodilators such as sublingual nifedipine and glyceryl trinitrate (Gardner and Klugger, 2004). As the patient will not be able to feel the stimulus (such as a distended bladder), patient and family education will need to be provided so that they recognize the symptoms of this potentially dangerous situation.

**Neurological status**

In complete spinal injury, assessment of level of sensation needs to be performed, as this will provide information as to the level of the spinal cord at which the injury has occurred. In incomplete injury, motor and sensory function need to be assessed in order to ascertain the level of injury and the type of injury that has been sustained (Hickey, 2002) (Table 3). Ongoing assessment is important as function may return as oedema resolves or, if oedema ascends up the spinal cord, levels could change. This is especially important in the C5–7 level injuries when ascending oedema could create respiratory difficulties (Oh, 1997).

Spinal shock can occur after a spinal cord injury and it is viewed as the complete loss of all neurological function, including reflexes, rectal tone and autonomic control, below the level of the injury. However, it is unrelated to neurogenic shock or other forms of shock. The complete loss of function usually lasts for about 24–72 hours; however, despite the profound paralysis that occurs in this stage, compete recovery is possible. Until spinal shock resolves, complete and incomplete injury cannot be determined (Greaves et al., 2001).

**Nutrition**

After acute spinal cord injury, support may be required to maintain nutritional status. A nutritional risk assessment should be performed with the dietitian (Grundy and Swain, 2002). If necessary, nasogastric feeds should be commenced once bowel sounds are heard in all four quadrants of the abdomen. There is a risk of paralytic ileus after a spinal cord injury, which may be slow to resolve, so total parenteral nutrition may be required (Hickey, 2002). Initially, the nasogastric tube may need to be on free drainage to decompress the stomach, thereby avoiding respiratory compromise because of gastric distension (Chiles and Cooper, 1996). There is a potential risk of regurgitation and aspiration of enteral nutrition while on flat bedrest which can be minimized with gently tilting the bed after agreement by medical staff. Once
able, an oral diet should be encouraged. Feeding aids may be required depending on the level of the injury and motor function.

Bladder function

Bladder dysfunction may occur after a spinal injury. During the spinal shock phase, an indwelling catheter will be required to ensure bladder emptying. Once the spinal shock phase has resolved and the level of injury has been determined, bladder management depends on the level of injury.

In injury above L1, the patient may have a hyperreflexic (or spastic) bladder (Figure 8) and this occurs because the level of the injury is above the reflex voiding centre in the sacral portion of the spinal cord (Zejdlik, 1992). Both motor and sensory function are damaged, resulting in a loss of sensation to void and a loss of voluntary control over the voiding centre. Therefore, patients with hyperreflexic bladders are unaware of the normal sensation of fullness and cannot control voiding. When the bladder becomes full, the stretch receptors in the muscle wall of the bladder become stimulated and a simple reflex arc results causing an uncontrolled contraction of the bladder (Zejdlik, 1992).

Where the injury is below L2 (Figure 9), the bladder is areflexic because the injury to the sacral portion of the spinal cord damages the voiding reflex centre itself (Zejdlik, 1992). The interruption in the sensory stimulation and motor reflex arcs at this spinal cord level breaks the communication pathways with the intact sensory and motor tracts above the level of the injury (Zejdlik, 1992). This results in diminished reflex activity and bladder tone. The patient will be unaware of the normal sensation of bladder fullness and is unable to initiate emptying of the bladder and therefore it becomes overdistended (Zejdlik, 1992).

Bladder management

Alteration in bladder control is probably one of the most disruptive problems a person with a spinal cord injury will face. The maintenance of bladder emptying is a major nursing responsibility requiring a combination of knowledge, skill and patience in order to ensure individualized, tailor-made programmes (Zejdlik, 1992). Assessment of voiding patterns must initially be undertaken. Any voluntary control of voiding must be observed for and documented, as well as any awareness that the patient has of sensation of fullness or spontaneous voiding. This may not be a simple lower abdominal sensation of pressure but, for example, the quadriplegic patient may perspire more when his/her bladder is full (Zejdlik, 1992).

Bladder management also requires an assessment of functional ability, such as hand motor function, as well as patient cooperation. Successful implementation will require patient motivation, responsibility, and receptiveness (Zejdlik, 1992).

An indwelling catheter is almost always necessary during at least the first 12 hours after injury and it may be required for a longer period to monitor the seriously ill patient through the acute phase (Grundy and Swain, 2002). However, the method of choice, and the desired aim in patients with neurogenic bladder dysfunction, is intermittent catheterization (Zejdlik, 1992). The underlying principle is to empty the bladder at regular intervals with the use of a non-retaining urethral catheter, thereby avoiding overdistension and the complications associated with this, such as ascending infections, which can cause kidney damage (Hudak et al., 1998). If the patient has the motor abilities, intermittent self-catheterization can be taught.

In conjunction with intermittent catheterization, manual techniques to elicit bladder emptying can be used. Intermittent catheterization facilitates this by allowing filling and emptying of the bladder, thereby stimulating any existing spinal cord reflexes and also maintaining bladder tone (Zejdlik, 1992). Manual techniques are trigger voiding, straining to void and manual expression (or Credé manoeuvre):

**Trigger voiding**: after a trigger response, the detrusor muscle of the bladder contracts through a spasm or reflex, expelling urine and emptying the bladder. This can be useful as the reflex contractions that can occur when the bladder is full do not usually empty the bladder completely; therefore, by creating an additional trigger, the voiding contraction is augmented (Zejdlik, 1992). However, this technique can only be used if the reflex arcs to the sacral voiding centre are intact. Trigger stimuli include firmly tapping, stroking or pinching the abdomen and inner thigh (Zejdlik, 1992).

**Straining to void**: using a Valsalva manoeuvre can help with bladder emptying. This technique requires strong abdominal muscles and the level of injury needs to be below T6–12 as it is at these levels that the abdominal muscles are innervated. This technique can be useful in patients with an areflexic bladder (Zejdlik, 1992).

**Manual expression (Credé manoeuvre)**: this involves the application of external pressure over and around the bladder, aiming to increase the pressure within the bladder and, therefore, overcoming the resistance of the bladder neck and urinary sphincters. This technique is only used in a flaccid bladder and when voiding cystourethrography (which visualizes the voiding cycle through fluoroscopy) confirms the absence of ureterovesical reflex (Zejdlik, 1992). It should be used with caution as it can increase the risk of hernias and haemorrhoids and if undertaken in patients with a hyperreflexic bladder can cause reflex, thereby increasing the risk of infection (Zejdlik, 1992).

Despite various techniques and management programmes, an unbalanced bladder with high residual volume may persist, leading to a constant, dribbling incontinence or an inability to completely empty the bladder (Zejdlik, 1992). These symptoms are caused by bladder and sphincter dysfunctions occurring at the same time and, for the patient with a spastic bladder, detrusor–sphincter dyssynergia can create major difficulties in bladder management. The detrusor muscle and the external urethral sphincter contract at the same time, leading to obstructed urine flow (Zejdlik, 1992). Anticholinergic drugs, such as oxybutynin, may be of help as these inhibit bladder contractility (Grundy and Swain, 2002). In some
patients, surgical intervention, such as sphincterotomy, may be required to relieve the symptoms, especially in view of bladder neck obstruction (Grundy and Swain, 2002).

**Bowel function**

As with bladder function, independent bowel function after a spinal cord injury can be difficult and bowel management must commence from the day of admission. During the spinal shock phase, stool softeners should be commenced and either daily or alternate day suppositories should be given (Zejdlik, 1992). An accurate bowel chart must be established to document clearly the effectiveness of the bowel regimen. Once spinal shock has resolved, then the level of the injury will influence bowel management.

Where the injury is above T12, the reflex defaecation centre, which is in the sacral portion of the spinal cord, is intact (Zejdlik, 1992). Reflex activity is uninhibited but ascending sensory signals are interrupted. Thus the patient is unable to feel the normal sensation or urge to defaecate. However, descending motor signals are also blocked and so normal control of the external anal sphincter activity is also blocked. Therefore, the patient has a spastic bowel with contraction of the anal sphincter (Zejdlik, 1992). If the injury is below L1, the reflex defaecation centre is directly damaged, leading to a flaccid bowel (Zejdlik, 1992). Even though intrinsic contractile responses remain, there are ineffective peristaltic actions because of the diminished spinal reflex. Also, the patient loses the normal sensation to defaecate and cannot voluntarily control the external anal sphincter (Zejdlik, 1992).

**Bowel management**

In the early days after spinal cord injury, it is a nursing responsibility to maintain bowel elimination and an effective bowel-management programme should be established (Coggrave, 2004). However, loss of control over bowel function can be distressing and upsetting for the patient. Success of the programme will depend on patient cooperation and the aim of the programme is to ensure a planned, predictable bowel movement (Zejdlik, 1992). This then should avoid both incontinence and constipation. A number of strategies can be used to achieve this aim:

**Diet:** high-fibre foods, such as fruit and vegetables, should be encouraged, with additional fibre taken if necessary (Coggrave, 2004). High-fibre diets can contribute to abdominal distension because of fermentation of food which may be uncomfortable.

**Fluids:** a fluid intake of 2000-3000 ml/day is necessary to keep stools soft (Zejdlik, 1992) and helps to avoid cramping constipation when high-fibre and/or stool softeners or bulking agents are used.

**Exercise and activity:** this should be within the patient's functional ability but even when on bedrest, passive movements can help to prevent sluggish bowel activity, which can lead to constipation (Zejdlik, 1992). Once the patient is in a wheelchair, encouraging him/her to participate in dressing, feeding or pushing the wheelchair will also help to promote bowel function as well as rehabilitation.

**Increasing abdominal pressure:** patients who have strong abdominal muscles can perform the Valsalva manoeuvre to evacuate their bowel. Leaning forward also increases intra-abdominal pressure and may help (Zejdlik, 1992).

**Abdominal massage:** this is thought to stimulate the colon to push faeces into the rectum. It can be used as an adjunct to other techniques to assist in bowel evacuation such as suppositories, anorectal digital stimulation or manual evacuation (Coggrave, 2004).

**Oral laxatives:** these are not required for all spinal-injured patients and should not be viewed as an essential part of their bowel programme, even if required during the acute phase (Coggrave, 2004). Laxatives may have undesired side-effects, such as cramping, flatulence, loose stools and dehydration (Coggrave, 2004). If used, a response may not be seen until 24 hours later because of the reduced bowel activity (Zejdlik, 1992).

**Stimulation techniques:** these are used in the patient with a spastic bowel. In this situation, digital stimulation is required. However, initially, a glycerine suppository can be administered; this may have the effect of lubrication and stimulation of reflex bowel activity. If no stool is evacuated after this, anorectal digital stimulation can be used, which encourages the anal sphincter to relax and thus the stool to be passed (Zejdlik, 1992).

**Manual evacuation:** a patient with a flaccid bowel will require manual evacuation to remove the stool, especially if other techniques, such as bearing down and leaning forward, have been unsuccessful (Coggrave, 2004). A suppository, such as bisacodyl, that will stimulate the colon to empty stool into the rectum may be useful (Zejdlik, 1992).

Privacy is paramount throughout and, whenever possible, normal toileting facilities should be used as this will aid bearing down (Coggrave, 2004).

**Pressure areas**

Pressure ulcers are among the most common complications in patients with a spinal injury (Hickey, 2002). This patient group will be unable to relieve their pressure areas independently and because of sensory abnormalities, may not be able to feel pain in these areas. Therefore, meticulous pressure area care is a priority of therapy and patient education around maintaining intact skin should commence as soon as possible. In this way, patients take a responsibility within their care. To help facilitate pressure area care, a turning clock can be used, which divides the day into 2-hour periods and indicates what position the patient should be in at any given time (Zejdlik, 1992). An electric spinal bed may be useful, particularly in heavy patients or those with multiple injuries (Grundy and Swain, 2002). This allows the patient to be turned at small increments such as 10-20° at a given time period. Careful alignment must be maintained at all times and logrolling will still be required for examination of pressure areas and for bowel care.

**Care of the joints**

The joints must be passively moved through a full range of movement to prevent stiffness and contractures. This is essential, so that joints which may recover function remain functional, thereby enabling rehabilitation (Grundy and Swain, 2002). Splints for the hands are particularly important to maintain the hand in a functional position, while ankle splints can help to avoid foot drop and shortening of the Achilles tendon (Grundy and Swain, 2002). Once any recovery of limb function has occurred, participation in daily activities should be encouraged.

**Psychosocial care**

Patients with spinal injury will experience a number of emotional responses as they pass through the initial injury phase and commence rehabilitation (Hickey, 2002). Sensory deprivation is a
major concern for these patients. They will not have normal sensory input from their body, which means that they have visual, auditory and tactile sensory deprivation (Hickey, 2002). Confinement to bed, which is usually kept flat or slightly sloping upwards, means that patients will not be able to see around them and will normally only have the ceiling within their visual range. Sound may be distorted by traction and the machinery around them. Therefore, the patient can feel very isolated and socially deprived (Hickey, 2002).

Nursing interactions, such as providing bedside mirrors and reading tables, may help to avoid the sensory deprivation these patients may experience. Additionally, the provision of a radio or television can be beneficial.

Patients’ self-concept will have been altered and their social position threatened. They will be aware that they may never be able to function independently in the future, and if they are the main wage earner in the family they will be worried and concerned about finances (Zejdlík, 1992). If patients are young, they are likely to be anxious and worried about their development as a functional person within society as well as having plans for their future development as an adult curtailed.

Therefore, many patients with a spinal injury will go through the steps of the grieving process in their path to accepting their injury: denial; anger; bargaining; depression; and acceptance (Kubler-Ross, 1969). Nurses will be required to support them through this and provide information as required. The psychological rehabilitation of the patient is as important as the physical rehabilitation as it is by accepting the change that has resulted from the injury that patients can take hold of their life and take responsibility for it themselves (Zejdlík, 1992).

Conclusion

The nurse caring for the patient with a traumatic neurological injury will face a challenge to ensure the best possible outcome for the patient. The aim of care is to minimize the impact of factors that can cause secondary injury and therefore, prevent ischaemia from occurring. However, the impact of the primary injury cannot be removed and it is this, especially in spinal injuries, that will greatly influence the patient’s future prospects. The care of this patient group requires insight and skill in the management of physiological systems, but the psychosocial aspects are as important, especially in the acceptance and future circumstances of both the patient and his/her family and loved ones.

KEY POINTS

- Trauma to the central nervous system can have serious implications for the person who is injured and his/her family/loved ones.
- Head injury management requires careful manipulation and control of the variables that affect cerebral blood flow.
- Care of the patient with an acute spinal cord injury requires early rehabilitation once spinal shock has resolved.