

Initial Care Management for Patients with Acute Spinal Cord Injury

 

The Princess Royal Spinal Cord Injuries Centre

Sheffield

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The following guidelines are a summary for management of spinal cord injury patients within the Major Trauma Centre. While the principles stated below apply to any patient with a spinal cord injury, other clinical considerations such as the requirement to manage polytrauma (see STH Emergency Department Major Trauma Guidelines) or the presence of pre-existing co-morbidities may necessitate modification of priorities.

Please remember to contact the Spinal Cord Injuries Centre within 4 hours of diagnosis of traumatic SCI or within 24 hours of confirmed non-progressive non-traumatic SCI diagnosis and enter the patient on the National Database.

Undertake online referral at: nww.scireferrals.nhs.uk - from an NHS computer only.

Contact the Sheffield SCI Consultant on Call. Tel: 0114 2434343

The aims of this Pathway are to:

1. Prevent secondary spinal cord lesion

2. Manage the cardiovascular impact of spinal shock appropriately

3. Minimise incidence of DVT and PE formation

4. Prevent prolonged paralytic ileus and vomiting due to early commencement of enteral feeding

5. Prevent gastric ulceration

6. Promote postural chest drainage and improve ventilation and perfusion

7. Prevent bladder distension and agitate urine in the bladder to prevent sedimentation and catheter blockage

8. Prevent over-distension of the rectum with hard faeces and the development of constipation which can cause bowel perforation

9. Prevent pressure ulcer formation

10. Maintain appropriate body temperature.

11. Prevent drop foot and upper limb and finger contractures which might delay or prevent meaningful rehabilitation

Additional information: Autonomic Dysreflexia (page 14-16)

 Orthostatic Hypotension (page 17-19)

 References (page 20-21)

Abbreviations:

SCIC - Spinal Cord Injuries Centre

SCI – Spinal Cord Injury

NEWS /HEWS – National Early Warning / Hospital Early Warning Score <https://www.england.nhs.uk/ourwork/clinical-policy/sepsis/nationalearlywarningscore/>

1: Prevent secondary spinal cord lesion

***Rationale: Traumatic spinal cord lesions can further deteriorate due to inappropriate mechanical forces, hypoxia or circulatory insufficiency***

* 1. Immobilise the spine with an appropriate traction/collar/halo-brace system and manage in accordance with device instructions. Immobilisation by any means (e.g. sandbags) is better than struggling to apply a poorly fitting device.

1.2. Ensure spinal column alignment throughout all turns, positioning, therapeutic procedures and transfer manoeuvres.

Pictorial guidelines for acute handling and positioning of SCI patients at:

<https://www.mascip.co.uk/wp-content/uploads/2015/02/MASCIP-SIA-Guidelines-for-MH-Trainers.pdf>

1.3. Positional care should continue post-surgery as outlined above.

1.4. Bed rest is advised by Sheffield SCIC for the first 2 weeks post injury, then reviewed, however this should be discussed with local area consultant/SCIC consultant.

1.5. Even where surgical stabilisation has been carried out; to enable stabilisation of physiological parameters and to avoid further insult to the cord bed rest (less that 30 degrees) is advised.

1.6. Maintain clinical observations within parameters appropriate to patient presentation rather than standard HEWS.

1.7. For ventilated patients, maintain maximum 15 degree head-up whole bed tilt (reverse Trendelenberg).

1.8. Head-up angle may need to be increased in some brain injured/respiratory compromised patients after discussion with SCI Centre.

1.9. Document of extent of neurological impairment using the American Spinal Injury

Association (ASIA) Impairment Scale.

<https://asia-spinalinjury.org/wp-content/uploads/2016/02/International_Stds_Diagram_Worksheet.pdf>

2.0. Use of a dynamic wave mattress is contra indicated in the presence of an unstable spine.

2: Manage the cardiovascular impact of spinal shock

***Rationale: The loss of vasomotor tone throughout the paralysed areas of the body provides the classic diagnostic observations of spinal shock; hypotension, bradycardia and poikilothermia. The impact of this effect varies depending on the level of spinal cord lesion. Spinal shock can cause particular difficulty in determining the presence of actual hypovolaemia or cardiac failure***

2.1. Avoid fluid overload.

2.2. Introduce or maintain post-resuscitation intravenous fluids and inotropic medicines judiciously.

2.3. SCI patients with lesions above T6 will require alternative ‘triggers’ for NEWS.

2.4. Monitor core body temperature closely, actual body temperature can be as much as 1°C below normal due to influence of environmental temperature. Insulate the patient or distance paralysed areas of the body as appropriate from sources of heat or cooling. Utilise body warming or cooling devices cautiously.

2.5. In the presence of potential pyrexia with no indication of sepsis, check forehead temperature against foot to check for poikilothermic (hot head-cold feet) to avoid misdiagnosis and inappropriate treatment.

2.6. Monitoring and treating patients presenting with multiple injuries in the presence of SCI requires a cautionary approach to interpreting observations to ensure potentially life threatening complications are not over looked in the presence of spinal shock.

3: Minimise DVT and PE formation

***Rationale: Enforced bed rest and systemic paralysis increases risk of thromboembolism.***

3.1. Commence prophylactic anticoagulation within 24hrs of diagnosis.

3.2. Apply properly sized thigh-length (if available) graduated compression stockings/ pneumatic compression device.

3.3. Provide twice daily range of passive limb movements by physiotherapist supported by regular turning and repositioning of limbs by nursing staff.

3.4. Position lower limbs on pillows when patient is supine to encourage venous drainage.

3.5. Remove compression stockings/pneumatic compression device at least once daily to wash underlying skin.

3.6. Check and re size stocking regularly as SCI often results in changes of lower limb shape and size.

3.7. Monitor for unexpected pyrexia or limb swelling.

4: Prevent prolonged paralytic ileus and vomiting

***Rationale: Acute SCI patient presents with initial paralytic ileus. Gut peristalsis must be nurtured.***

5.1. Maintain ‘nil by mouth’ for first 48 hours, even if bowel sounds are present on admission.

5.2. Avoid passing NG tube unless indicated.

5.3. Monitor abdominal girth if stasis/ileus is suspected.

5.4. Assess for nutritional support within 72 hours.

5.5. Introduce enteral fluids gradually.

5.6. Provide initial TPN if nutritionally compromised.

5.7. Patients with accompanying abdominal injury/surgery needs special consideration.

5: Prevent gastric ulceration

***Rationale: Increased risk of ulceration due to vagal over activity and initial ‘nil enterally’ requirement as advised due to potential paralytic ileus***

4.1. Risk assess each patient for bleeding potential based upon previous medical history

4.2. Commence prophylactic pharmacological gastric protection within 24 hours of diagnosis.

* 1. . Assess patient for commencing enteral nutrition within 72 hours.

4.4. Observe for abdominal distension or discomfort relieved by anti-acid preparations or referred vagal pain in perceived area above lesion (burning pain sensation referred to apex of left shoulder).

6: Promote sputum clearance/ chest drainage

***Rationale: Pulmonary complications can have a significant impact for SCI Individuals and can be life threatening***

6.1. Encourage patient to clear secretions, if any. Cough efficiency will be reduced and may require assistance from physiotherapy.

6.2. Direct treatment towards reducing sputum viscosity (e.g. saline nebulisers, mucolytics) and bronchodilation (e.g. beta agonists).

6.3. SCI patients with isolated cervical injuries should not normally be nursed with head of bed above 15 degree head up angle.

6.4. SCI patients with thoraco- lumbar injuries should preferably be nursed with whole bed tilted to 15 degree reverse Trendelenburg to maintain full spinal alignment.

6.5. Regular turning assists in mobilising chest secretions reducing potential for sepsis, 2 hourly turning regimes to a 30 degree position is the ideal standard. The need to turn a particular patient beyond 30 degrees should be qualified with specialist SCI teams. Inappropriate siting contributes to multiple avoidable complications in SCI physiology.

6.6. Monitor heart rate throughout suctioning and keep atropine to hand as per local policy. Oro-pharyngeal and tracheal suctioning in the SCI patient with a lesion above T6 can induce significant vaso-vagal stimulation sufficient to induce cardiac syncope. An obstructed or occluded tracheal/tracheostomy tube can induce syncope.

6.7. Monitor for respiratory fatigue and sleep apnoea at all times.

6.8. Monitor for abdominal distension which can splint diaphragm monitor for hypoxia and hypercapnia which may also affect neurological recovery.

6.9. Liaise with SCI centre respiratory support team regarding weaning or management of long term ventilator support.

7: Bladder management

***Rationale: The paralysed bladder is at significant risk of nosocomial infection and sedimentation creates an increased risk of catheter blockage.***

7.1. Catheterise on admission with long-term all-silicone urethral catheter (FG16 ) to reduce the risk of blockages due to the increased amount of sediment laid down in the paralysed bladder .Ensure the catheter is secured to body to reduce urethral erosion.

7.2. SCI patients with lesions above the level of T6 are at risk of Autonomic Dysreflexia, a potentially fatal hypertensive response to visceral pain or discomfort. This complication of SCI can occur in both new and established cases (see appendix).

7.3. Change catheter every 6 weeks to reduce risk of blockage.

* 1. . If catheter blocks change catheter rather than ‘flushing’ to enable continued patency.
	2. . If the patient is stable clipping and releasing can be commenced two weeks post injury.
	3. Do not attempt trial without catheter (TWOC) without prior discussion with the SCI centre/ SCI Link worker.

8: Bowel Management

***Rationale: Individuals with a Spinal Cord Injury invariably have neurogenic bowel dysfunction which needs to be managed appropriately to avoid complications.***

***Key Point: Following spinal cord injury, there is an initial period of approximately 48-72 hours in which the rectum and anus are flaccid. This is due to ‘spinal shock’, which causes a loss of reflex activity below the level of the injury (however, this can continue for up to 14 days)***

8:1. Digital rectal examination (DRE) within 24 hours of diagnosis to establish sphincter status and presence of faeces.

8:2. Daily digital removal of faeces (DRF) in presence of areflexic (‘flaccid’) sphincter.

8:3. Continue with daily DRE check in the presence of spinal shock. Assess on a daily basis.

8:4. In the presence of a sphincter reflex, introduce a daily regime of rectal stimulant and digital rectal stimulation (DRS) as advised by SCI Centre. Do not use large volume (50ml) enemas.

8:5. Document all results utilising Bristol Stool Scale; report all extreme results and treat appropriately.

8:6. Introduce oral aperients and stool softeners as appropriate.

9: Prevent pressure ulcer formation

***Rationale – SCI individuals are at increased risk of pressure ulcer formation due to anaesthetic skin and immobility***

9.1. Check skin integrity at every opportunity.

9.2. Implement routine 2 to 4 hourly turning regime maintaining spinal alignment where indicated.

9.3. Support body in position and all limbs with pillows between turns (see link in aim 1, 1.2).

9.4. Use of a dynamic wave mattress is contra indicated in the presence of an unstable spine

9.5. Do not position patient on marked/red/broken areas of skin.

9.6. Use pillows to ‘block feet’ to prevent foot drop. Use of resting foot drop splints can cause skin damage.

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10: Thermoregulatory impact of spinal shock

***Rationale: Poikilothermia, the inability of the body to internally regulate body temperature, is characteristic of SCI. Impact varies depending on the level of and extent of complete paralysis. The paralysed body will adopt the ambient environmental temperature.***

10.1. Monitor body temperature closely, actual body temperature can be as much as 1°C below normal due to influence of environmental temperature.

10.2. Respond to visible symptoms of hypothermia (pyrexia above lesion, cold below) or systemic septic pyrexia (pyrexia all over) in the non-paralysed areas of the body, irrespective of core temperature findings.

10.3. Utilise body warming or cooling devices cautiously, insulating or distancing paralysed area of the body as appropriate from the source of heat/cooling.

10.4. Take extra precautions when transporting patients between departments and during CT/MRI scanning which utilise cooling fans.

10.5. Ensure the need for continuing appropriate protection and monitoring is communicated if patient transferred to operating theatre. Monitor effects of anaesthetic agents on body temperature.

11: Management of upper and lower limbs

***Rationale: Limb contractions occurring during initial management can have a detrimental effect on rehabilitation potential.***

11.1 It is recommended that passive range of movement is carried out in conjunction with positioning and prolonged stretch and should be conducted at least once a day unless there is a clinical indication for them to be performed more frequently such as increased tone or pain.

11.2 ‘Block’ feet to representative 90 degree resting angles using pillows, do not force into position maintain throughout periods of bed rest.

11.3 Position hands on small pillow/padding to assist venous return. Monitor closely for signs of gravitational oedema.

11.4 Where permitted, exercising fingers and toes during hygiene cares or throughout the day is advised to maintain range of movement. The regular application of moisturising cream to reduce hardening of skin is encouraged.

11.5 Avoid positioning cannula in paralysed feet to reduce chance of thrombophlebitis and DVT. Where possible, avoid positioning cannula in tetraplegia hands to reduce chance of thrombophlebitis. Cannula should be removed as soon as they are no longer clinically indicated.

11.6 Where splints are used they must be well padded/moulded, applied by an appropriately qualified therapist, utilised sparingly and relevant pressure areas monitored carefully for signs of tissue damage.

For further information please see **Princess Royal Spinal Cord Injury Centre Therapy Guidance for the Management of Spinal Cord Injury Patients at Referring Hospitals.**

AUTONOMIC DYSREFLEXIA

Autonomic dysreflexia is the term used to describe the autonomic response to painful (noxious) stimuli perceived below the level of lesion. This is a potential complication for all patients with spinal cord lesions above the level of T6. The most common stimulus is a blocked catheter. This problem manifests itself as acute hypertension. Systolic blood pressure can easily exceed 200mmHg. Unresolved it can cause significant complications including stoke, seizures, severe myocardial ischemia and death.

This reflex response is usually suppressed during the period of spinal shock however should still be considered.

The main presenting features of Autonomic Dysreflexia are:

* Severe (pounding) headache
* Profound vaso-dilation (flushing) above the level of cord lesion and vaso-constriction (pallor) below this ‘line of demarcation’ – visible even in different skin types
* Profuse sweating above the level of cord lesion

In the presence of visible primary symptoms, it is recommended that the initial investigation and treatment of cause should not be delayed through a poorly prioritised need to take and document a patient’s blood pressure and pulse.

Mechanism

Body functions involve central and reflex control systems within the brain, spinal cord and nerve structures. The autonomic nervous system is a regulatory branch of the central nervous system that helps people adapt to changes in their environment acting though its two branches the parasympathetic nervous system and the sympathetic nervous system. The branches work complementary to each other usually with one activating and the other inhibiting the actions of internal organs. Connections to different parts of the body are organised segmentally from the spinal cord. The sympathetic nervous system connections come from T1 to L2 segments those to the major blood vessels in the trunk, abdomen and legs are from T5 to T12/L2 spinal cord levels.

Autonomic dysreflexia results from widespread reflex activity of the sympathetic nervous system below the level of injury, triggered by an ascending sensory (usually noxious) stimulus. Following stimulation, over activity of the sympathetic ganglia remains uncontrolled due to isolation of the spinal cord below the injury from normal regulation by vasomotor centers in the brainstem. Release of substances, such as noradrenaline, cause severe vasoconstriction with skin pallor, pilo-erection and a sudden rise in blood pressure (BP), which is usually accompanied by a pounding headache.

When the body’s baroreceptors in the aortic arch and carotid bodies sense the increase in blood pressure they increase the parasympathetic signaling as a compensatory mechanism resulting in bradycardia (via the vagus nerve) and flushing (focal peripheral vasodilation); probably also responsible for headache and profuse sweating above the level of injury (via sympathetic inhibitory outflow from vasomotor centers). However, both these mechanisms are insufficient to satisfactorily control paroxysmal hypertension due to massive sympathetically mediated vasoconstriction of the splanchnic bed.

The commonest presenting symptoms of Autonomic Dysreflexia are:

* Flushed appearance of skin above the level of lesion
* Profuse sweating above the level of lesion
* Pallor below the level of lesion
* ‘Pounding’ headache
* Non-drainage of urine (urinary obstruction being commonest cause)
* Severe hypertension
* Nasal congestion
* Pilo erection

The commonest causes of Autonomic Dysreflexia are:

* Distended bladder (usually due to catheter blockage or some other form of bladder outlet obstruction)
* Distended bowel (usually due to constipation or impaction)
* Ingrown toenail
* Trauma below level of lesion
* Pressure sore / burns/ sunburn
* Urinary tract infection / bladder spasms
* Renal calculi / bladder calculi
* Deep vein thrombosis / pulmonary embolism

Actions in the event of Autonomic Dysreflexia should be prioritised as follows:

1. Identify or eliminate the most common (most potentially lethal) cause of Autonomic Dysreflexia which is non-drainage of urine. If this is not the cause, then proceed to investigate alternative causes according to the list provided above. Reassure your patient throughout as anxiety increases problem.
2. Identify and remove the noxious stimulus e.g. re-catheterise immediately in the event of a blocked catheter (do not attempt a bladder washout as there is no guarantee that the fluid will be returned). If no alternative catheter is available it may be appropriate to remove the blocked catheter completely to allow for possibility of urethral drainage until re-catheterisation is possible.
3. If appropriate, sit the patient up, or tilt the bed head-up, to induce some element of postural hypotension. Do this gradually, as it may actually worsen symptoms and hinder investigation where distended bladder or bowel is the cause.
4. If symptoms remain unresolved after removal of noxious stimulus or if noxious stimulus cannot be identified then administer prescribed proprietary chemical vaso-dilator such as sublingual glyceryl trinitrate (GTN) or sublingual captopril (25mg).

NB: Nifedipine capsules, which were previously recommended for use in treating Autonomic Dysreflexia have been withdrawn from routine use in UK due to being linked with post-incident hypotensive crises.

1. Record blood pressure and give further reassurance. Monitor patient’s condition.

**Orthostatic (Postural) Hypotension in Spinal Cord Injured Individuals**

**What is it?**

The definition of orthostatic hypotension (OH) is typically accepted as a decrease in systolic blood pressure of 20mmHg or more, or a reduction in diastolic blood pressure of 10mmHg or more, upon changing body position from a supine position to an upright posture, regardless of the presence of symptoms

Spinal cord Injured (SCI) individuals often suffer from orthostatic hypotension when they begin to mobilise. OH is more common in tetraplegia than paraplegia. This condition is most evident in the acute period post-injury, but it can persist in some individuals for some years. Individuals most at risk of long-term OH are those with lesions at or above T2. Symptoms are less likely to occur in SCI below T6 and with incomplete injuries.

**Why does it happen?**

The mechanisms underlying orthostatic hypotension following SCI are unclear, but are likely to be multifactorial.

Contributing factors include:-

1) **Lack of tonic sympathetic control**

Spinal cord injury (SCI) results in dysregulation of the autonomic nervous system and associated compensatory mechanisms.

The disruption of the descending spinal cardiovascular pathways, leads to sympathetic hypoactivity resulting in low resting blood pressure and loss of adequate blood pressure control.

2) **Impaired baroreceptor regulation:**

Baroreceptors are stretch receptors located in the aortic arch, carotid sinus and coronary arteries that produce a reflex response to changes in arterial pressure and re-modulate sympathetic and parasympathetic outflow in order to maintain blood pressure homeostasis.

This reflex response (baroreflex) is reduced markedly in those with high-level SCI (above the T6 level) and affects the SCI individual’s ability to maintain adequate blood pressure regulation.

3) **Lack of skeletal muscle pumping activity in the dependant limbs of paralysed individuals,**

In able bodied individuals, when moving between lying-to-sitting or sitting-to-standing the ‘anti-gravity’ muscles in the abdomen, thighs and calves contract to prevent all the blood in the body rushing into lower limbs and therefore prevent hypotension. After SCI, this mechanism is absent and so the SCI person has to rely on the slower contraction of peripheral blood vessels under hormonal control to maintain blood pressure.

4) **Cardiovascular Deconditioning**

Following SCI, some cardiovascular deconditioning is to be expected due reduced mobility and is manifest by profound orthostatic intolerance, thought to be mediated via a diminished blood volume, decreased muscle / tissue pressure in the extremities.

**Signs of orthostatic hypotension**

Affected individuals typically complain of dizziness and/or sensory disturbance shortly after being positioned in their wheelchair. If uncorrected, this can progress to syncope (fainting) as a result of which the individual may be difficult to rouse or may be at risk of falling. In extreme cases, respiratory arrest is possible.

**Prevention**

Moving too rapidly between positions during lying-to-sitting to will predispose a sudden onset of postural hypotension, therefore, SCI people need to move slowly and gradually between positions to acclimatise.

Risk increases following a heavy meal (blood diverted to stomach for digestion) and also immediately following bladder or bowel emptying.

1) Gradual changes in position from lying-to-sitting

2) Gradual bed-to-chair transfer

3) Exercises in bed before sitting out

4) Use of elastic stockings or binders

5) Chemical vaso-constrictor medicines (ephedrine, midodrine etc)

6) Rest periods back on the bed– particularly after meals or continence management

**Actions**

1) Act quickly, but calmly. With appropriate action, in the majority of cases, symptoms will resolve and it is unlikely this will become a medical emergency.

2) If safe to do so, tip the individual back in their chair. The aim is to raise the affected individual’s legs above the level of the heart. If it is not possible to tip the chair safely, return the individual to bed.

3) Observe the individual visually to ensure they are responding. There is no need to take their blood pressure unless the individual is slow to respond or appears unwell afterwards. If the individual fails to respond quickly to your initial actions, escalate to ‘collapsed patient’ -A-B-C scenario

4) Wait about 15 minutes after the individual recovers before trying to sit them up again.

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