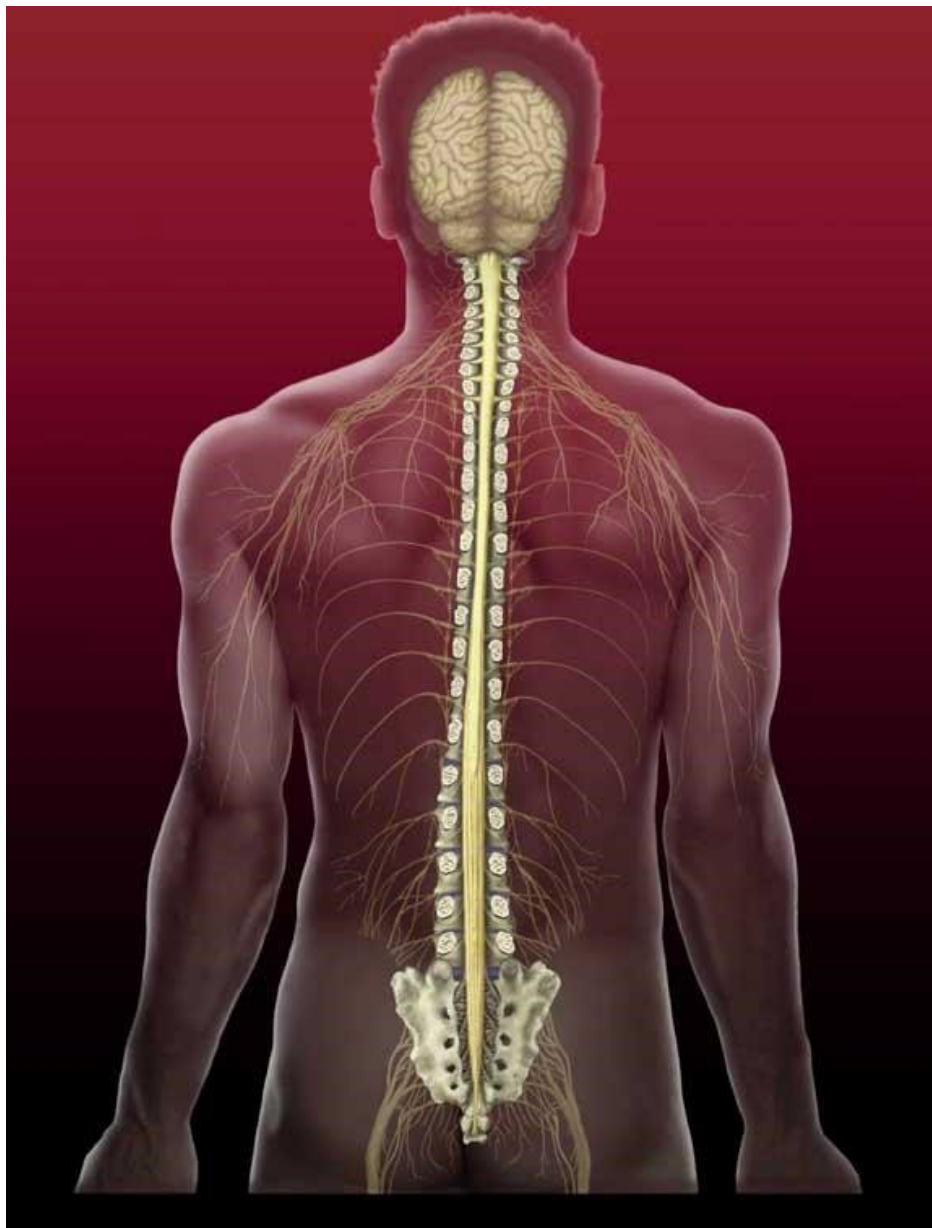


SPINAL CORD INJURY HANDBOOK



 **WVU** Critical Care &
Trauma Institute

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Cardiovascular care of Spinal Cord Injury Patients

Goals:

Maintain MAP > 85 during the first five days of SCI treatment

Treat symptomatic bradycardia

Treat autonomic dysreflexia

MAP Goals

Mean arterial pressure (MAP) is maintained above 85 for the first five days. After the prescribed five day period MAP goal is reduced to 65 (normotension).

Maintain MAP $\geq$ 85 x initial 5 days

Must maintain euolemia. Monitor urine output closely.

Norepinephrine is vasopressor of choice.

Consider invasive hemodynamic monitoring or echocardiography to evaluate need for inotropes.

Maintain MAP $\geq$ 65 after initial 5 days

All IV vasopressors and inotropes must be weaned off prior to transfer out of ICU

If unable to maintain MAP > 65 off of vasopressors consider the following:

Re-evaluate intra-vascular volume status and need for volume resuscitation to maintain euolemia

Consider NaCl tablets (total dietary intake of 10g sodium per day)

Consider fludrocortisone 0.1 mg PO daily

Consider Midodrine 5 mg PO Q8H

Consider Pseudoephedrine 30 mg PO Q8H

If these agents are unsuccessful discuss with your pharmacist the need for further supplementary therapy (see supplement)

Prevent orthostatic hypotension

ACE wraps to bilateral lower extremities and abdominal binder in place at ALL TIMES when patient is out of bed. May remove when back to bed.

HR Goals

Treat bradycardia

Apply pacing pads

Atropine 0.5 mg IV Q1H PRN for HR < 40

Norepinephrine as above. Consider dopamine if norepinephrine ineffective

If medical therapy is ineffective initiate external pacing

Consider cardiology consultation for temporary intravenous pacemaker insertion

Maintain HR > 60

Autonomic Dysreflexia

This is a sudden, significant increase in systolic and diastolic blood pressures (in general 20-40 mm Hg above baseline) frequently associated with bradycardia and a severe headache. Other signs and symptoms include profuse sweating above the level of the spinal cord lesion, piloerection, cardiac arrhythmias, and anxiety.

- Immediately sit the patient up if they are supine
- Evaluate for instigating causes (most commonly bladder distension; see supplement)
 - Insert urinary catheter if not already in place
 - Consider 2% lidocaine jelly insertion into urethra prior to insertion, if possible.
 - Check for catheter obstruction if urinary catheter is already in place
 - Irrigate with 10-15 ml of fluid
 - If catheter is obstructed, replace the catheter
- Evaluate for fecal impaction and disimpact manually as tolerated by the patient.
- Evaluate for other causes including unidentified fractures, compartment syndromes of the abdomen or extremities, tight abdominal binders, constrictive clothing, etc. Anything that results in pain or sustained stimuli below the level of the injury can result in autonomic dysreflexia.

Appendix

There is not a preponderance of evidence to assist the clinician in determining a mean arterial pressure goal for spinal cord injured patients¹. Most authorities have come to the consensus that a MAP goal of greater than 85 likely provides the best benefit. To achieve this goal, the intensivist has a variety of modalities available.

Volume resuscitation is the first line of therapy to maintain hemodynamic goals. This will only work insofar as volume increases preload and thereby cardiac output. Once a patient is assessed to be to the right of the steep part of the Frank-Starling curve, alternative means to increase blood pressures will need to be applied. **Vasopressors**, such as norepinephrine, are generally employed early to achieve this goal once euvolemia is achieved. It should be emphasized that pressor therapy in a patient who is hypovolemic is unlikely to improve tissue perfusion and can, in fact, be detrimental and cause ischemia. Pure alpha agonists such as phenylephrine may also be used for patients who are in spinal shock. Caution is advised as the reflex bradycardia can compound the already uninhibited vagal activity of cervical spinal cord injury patient. **Inotropes** can be necessary for patient as well. Medications such as dobutamine increases the contractility of the heart and can be used to augment blood pressures in patients who have poor cardiac output as seen on bedside echocardiography or via invasive hemodynamic monitoring. Epinephrine may be preferred in these situations as it provides inotropy as well as peripheral vasoconstriction.

It should be emphasized that the treatment plan for a patient may change over the course of their stay. Patients who have previously achieved euvolemia may proceed to dehydrate during their stay. For example, often our SCI patients are young with very healthy kidneys. The augmented blood pressure they see because of the pressor will cause an abnormally high rate of diuresis and this can contribute to volume depletion. Hence, it behooves the clinician to reassess patient's hemodynamic needs, their I/O distribution and other markers of volume status and hemodynamics. Close communication with the bedside nurse is paramount as they will often have valuable information about how their patient's blood pressure is affected during periods when vasopressor drips are off or weaned. If the underlying patient state is normotension, then pressor therapy to augment the MAP is likely correct. If the underlying state is hypotension, then the clinician should consider other sources of hypotension that should be corrected.

Orthostatic hypotension should be prevented in these patient population via the use of ACE wraps to the bilateral lower extremities as well as abdominal binders.

Once the course of MAP augmentation is completed the IV drips are to be weaned. If this proceeds without complication the patient may then be transferred to step down. Not infrequently, however, the patient's underlying sympathetic activity is severely affected and their baseline perfusion pressure now result in hypotension. These situations may result in the need for PO vasopressors as are mentioned in the guideline. In unusual circumstances additional modalities may need to be used such as methylphenidate, dextroamphetamines, caffeine, erythropoietin, clonidine, yohimbine and even beta blockers². The use of these drugs should be individualized.

During the course of care of a spinal cord injury patient, the clinician should also be cognizant of any and all causes of autonomic dysreflexia. This is a phenomenon that occurs in patients who have an injury above the level of T6. Noxious stimuli below the level of injury can result in diffuse, exaggerated sympathetic activity resulting in diffuse vasoconstriction and hypertension. A compensatory parasympathetic response results in bradycardia and vasodilation above the level of injury but this is insufficient to affect the severe hypertension. Lesions below T6 do not result in this syndrome because intact splanchnic innervation allows for compensatory dilation of the splanchnic vascular bed. The complication is unusual in the first month of injury, but will often appear within the first year. The most common causes include bowel and bladder distension, pressure sores and bone fractures. However, occult visceral disturbances, sexual activity, tight fitting clothing or abdominal binders, labor and delivery, and compartment syndrome can all contribute i.e. **any source of noxious stimuli below the level of injury**.

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Respiratory care of Spinal Cord Injury Patients

Ventilated SCI Patients

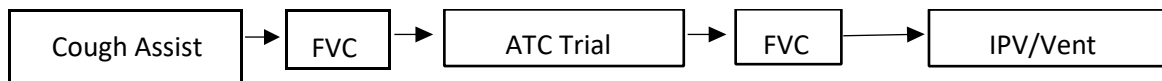
Ventilator management will be per SICU attending and individualized based on the patient's injury profile. However, consider the following guidelines especially for isolated high SCI patients

- Have a low threshold to secure the airway in these patients particularly in light of diminishing breath sounds in the lung fields or gradually decreasing FVC measurements
- Consider using higher tidal volumes of around 10-15 ml/kg IBW
- Maintain plateau pressures < 30 mm Hg
- VAP protocol
 - Maintain HOB > 30 at all times
 - Oral care Q4h
 - H2-blockers for GI prophylaxis (see GI section)
 - Minimize sedation and perform sedation holidays
 - Peridex oral rinse 15 ml swish and suction Q6h
- FVC, NIF, TV ASAP then Q8h
 - May wean to Q24h if >1.5 L x 48 hrs
- Peridex oral rinse 15 ml swish and suction Q6h
- Cough Assist Mechanical Insufflation-Exsufflation Q4h
 - Applies rapid variations of positive and negative pressure to the airway to help loosen secretions. The resulting high expiratory flow simulates a cough.
 - May be weaned to Q4h PRN if no secretion production
- Bronchodilator therapy
 - Must be started before bronchospasm is apparent. Bronchospasms are frequent in high SCI patients with no baseline pulmonary dysfunction due to unopposed vagus nerve (parasympathetic) activity

- Albuterol (Proventil) 2.5 mg, nebulized solution, q4h and q2h prn +/- ipratropium
- Consider nebulized hypertonic saline and/or mucomyst for thick secretions
- May de-escalate to q8h and q4h prn when bronchospasms **and** secretions are absent x 24 hrs
- Suction therapy
 - Maximum of two passes at each procedure
 - Suctioning pressure to be at 100-200 mmHg
 - Limit insertion to 1/3rd of catheter length of ~15 cm in an adult tracheostomy tube
 - Hyperoxygenate x 1 min with 100% oxygen before and after suctioning
- Bronchoscopy
 - Consider bronchoscopy early if secretion clearance is likely to be ineffective with aggressive pulmonary toilet (as above) alone.
- Abdominal binder in place when OOB at all times.

Ventilated SCI Patients with Tracheostomy

- Pulmonary toilet as above
- Initial aerosol trach collar (ATC) guidelines as follows:
 - FVC < 250 ml, start with 5 minutes Q8h
 - FVC between 250-500 ml, start with 15 minutes Q8h
 - FVC between 500-750 ml, start with 30 minutes Q8h
 - FVC > 1000 ml, start with 60 minutes Q8h
- **Prior to** initiating all ATC trials cough assist will be performed
- **After** completing an ATC trial a FVC is repeated and documented to evaluate patient's level of tolerance and degree of derecruitment
- **After** completing post-trial FVC, IPV is performed when returning the patient to the ventilator



- With initial trials, prevent fatigue by allowing rest periods on ventilator support
- Trach cuff must be deflated for all trials

- Once a patient has reached 24 hrs on ATC, discontinue IPV and continue cough assist for secretion clearance
- Once ATC trials are initiated, consult speech therapy for Passy-Muir valve
 - May apply valve once patient is able to tolerate ATC for > 2 hrs.

Non-Ventilated SCI Patients with no Evidence of Respiratory Compromise/Disease

- Cough Assist Q4h
- Peridex oral rinse 15 ml swish and spit/suction q6h
 - May discontinue when patient is tolerating an oral diet
- Albuterol nebulizers Q4h prn (as above)
 - May discontinue if not needed for > 72 hrs
- IS Q1h, PEP valve Q1h
- Acapela Q4h

Non-Ventilated SCI Patients with History of Respiratory Disease/Increasing Secretions/Worsening Pulmonary Function

- Cough Assist Q4h
- Peridex oral rinse 15 ml swish and spit/suction q6h
 - May discontinue when patient is tolerating an oral diet
- Albuterol nebulizers Q4h (as above)
- Initiate Aggressive Pulmonary Toilet Regimen (“Red Lung”)

Thick Secretions

- Heated Ventilator circuit
- Mucomyst 20% 3 ml nebulized Q4h-Q6h x 24 hrs and re-assess
- Hypertonic saline nebulizers
 - Monitor and evaluate for pulmonary infections
- Consider bronchoscopy and/or BAL

Appendix

Respiratory complications are the most common cause of morbidity and mortality in acute spinal cord injury. It has been shown that 80% of deaths in hospitalized patients with cervical SCI are secondary to pulmonary dysfunction, with the pneumonia the cause in 50% of cases. The development of respiratory complications is directly correlated with the level of injury and degree of injury completeness¹.

The main muscles that contribute to inspiration are the diaphragm, external intercostal muscles, and the accessory muscles which include the scalene, sternocleidomastoid (SCM), trapezius and the pectoralis major. The degree of inspiratory embarrassment suffered by a SCI patient depends on which muscles are affected, which are in turn affected by the level of the injury. The diaphragm is innervated by C3-5 (phrenic nerve). The intercostals are innervated by T1-11. The accessory muscles are variably innervated by the nerve roots from C1-8. In any quadriplegia the intercostals are invariably lost, therefore active expansion of the rib cage and elevation of the ribs is lost. High quadriplegics may also lose the nerve supply to the diaphragm and require diaphragmatic pacing to help come off a mechanical ventilator.

Expiration is normally a passive but forced expiration, namely 'coughing' is active. Although the internal intercostals contribute, the bulk of forced expiration originate in the abdominal muscles, namely the rectus abdominis, transversus abdominis, and the internal and external obliques. The pectoralis major muscle also has a role in expiration. In all quadriplegics and many SCI patients, abdominal muscle function is completely absent.

Hence, as the diaphragm relaxes during expiration the flaccid chest wall moves outward, limiting the expiratory reserve volumes to <20% of normal. Because the abdominal musculature is also flaccid, forced expiration is even more severely compromised. This is the reason that tidal volumes and FVCs are significantly higher in the supine position compared to sitting. Moreover, adding an abdominal binder helps not only with hypotension but also increases tidal volume by 16%. The pulmonary mechanics of SCI patients are superior in the supine position when compared to the erect position. This fact is noted in contradistinction to the evidence of improved ventilator associated pneumonia rates with elevation of the head of the bed ². Hence, all patient care should be performed with

individualization of therapies and the weighing of the risks and benefits of individual interventions.

The specific respiratory complications related to spinal cord injury are many and are generally more severe in this patient population. **Atelectasis** may rapidly worsen in these patients as respiratory muscles fatigue, secretions accumulate, and lung compliance decreases. **Hypersecretion** of bronchial mucosa may occur as soon as one hour after quadriplegia. This is caused by unopposed vagal stimulation. For a similar reason, **bronchospasm** develops early and is routinely seen. Hence, these patients should be started early and aggressively on inhaled bronchodilators even if they have no baseline reactive airway disease or have no audible wheezes on physical examination.

Because of the previously mentioned reasons, **respiratory failure** is common in these patients and attempts at early post-operative weaning should be cautious. SCI patients should have a FVC of at least **15 ml/kg of ideal body weight**, have a good cough prior to weaning. Further caution should be exercised in patients who are previous smokers or have premorbid respiratory illnesses, or are older than 45 years of age.

Again, in contrast to routine respiratory failure which is managed via the ARDSnet protocol, there is evidence to support the use of high tidal volume ventilation in SCI patients to prevent atelectasis. Aggressive secretion management should be routine for these patients and may be supplemented with bronchoscopies as needed. The cough assist device can replicate the 'quad cough' maneuver (abdominal thrust or chest squeeze in coordination with the patient's breath) and should be used early and aggressively to assist with secretion management.

If a high cervical spine lesion precludes early and safe extubation tracheostomies should be considered in these patients. Once this is accomplished graduated weaning from the ventilator should be accomplished as elucidated in the details above and in the flowchart. The emphasis should be on preventing these patients from tiring out and de-recruiting. Note that an FVC should be obtained before and after any aerosol trach collar trials and documented to assist in decision making regarding progression of ATC trials and if there is some element atelectasis developing with the trials. Progressing too quickly with long ATC trials predisposed to atelectasis, hypoxia, desaturation and patient decompensation. Progressing too slowly will result in longer times on the ventilator and in the ICU and the numerous

associated risks. A gradual, protocolized progression should help maintain the optimum balance. Please bear in mind that even 5 minutes of ATC at a time can help these patients start to develop some independence and help with some of the anxiety of coming off of respiratory support. Speech therapy should also be consulted early as these patients may often be able to receive inline speaking valves to help with communication even while on a ventilator.

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Gastrointestinal care of Spinal Cord Injury Patients

Start enteral feeds as soon as possible. Prone positioning for spine surgery is a relative contraindication for NPO status in patients who are already INTUBATED and should therefore be held six hours prior to surgery; all others should have tube feeds started. (Please refer to hospital perioperative fasting guidelines for further details.)

GI Prophylaxis

SCI patients are at *high risk* for stress ulcer gastritis and should be routinely placed on prophylaxis on admission:

- Famotidine (Pepcid) 20 mg PO, 1 tab, Q12h
- If patient has been on a proton-pump inhibitor or higher dose H2-blocker prior to admission restart home medications.

Tube Feeding Intolerance

If patient vomits with goal tube feeds consider the following:

- Post-pyloric feeding
- Initiation of pro-kinetic agents
 - Erythromycin 250 mg PO Q8H before meals
 - Metoclopramide (liquid) 5 mg PO Q8h before meals

Bowel Regimen

Spine injured patients are at a higher risk for developing ileus and constipation. Their bowel regimen requirements are more aggressive than the average trauma patient. Options include the following:

- Daily bisacodyl suppository
- Digital ano-rectal stimulation and manual evacuation of stool
- Glycerine suppositories
- Rectal laxative
- Oral laxative

- Pulse water irrigation

Aim for BM every other day

Bowel Training

All SCI patients should be on the following neurogenic bowel therapy medications on admission:

- Commode should be present at bedside.
- Encourage oral liquid and food intake when medically appropriate
- Attempt bowel movements after oral intake to take advantage of gastrocolic reflex
- Establish a singular daily time for bowel training to assist with habituation.
- Sennosides-docusate sodium (Senokot-S) 8.6-50 mg, 1 tab PO Q12h to assist in maintain stool consistency
- Bisacodyl (Dulcolax) 10 mg, rectal suppository, daily as a bowel irritant to maintain colonic stimulation

If your patient does not pass a soft bowel movement and is constipated proceed with the following:

- Digital ano-rectal **stimulation** and manual **evacuation** using 2% xylocaine jelly for a minimum of 20 minutes or until lower bowel empties
- If no effect, insert dulcolax suppository. Allow 1-2 hours for results ☐ Check for stool and manually evacuate as indicated ☐ If no effect, repeat.
- If no effect, consider Enemeez mini-enema.
- If no effect, contact MD

If your patient does not pass a soft bowel movement and is, in fact, incontinent with liquid stool proceed with the following:

- Hold oral and rectal bowel stimulants and proceed with daily digital stimulations and manual evacuations until a balance regimen can be established.
- It often takes 14 days or more for effective bowel training to be accomplished.

If your patient does not have a bowel movement for 72 hours or more proceed with the following:

- Magnesium citrate (Citroma) 296 ml, PO liquid, daily until bowel movement
- MD to assess for further intervention.

Appendix

Patients with spinal cord injury often suffer from neurogenic bowel. Neurogenic bowel syndrome is colonic dysfunction resulting from a lack of central nervous control. After the loss of ability to ambulate, loss of voluntary control of excretory bowel function has been identified as the second most distressing aspect of life following SCI, and is perhaps the most distressing for the newly injured^{1, 2, 3, 4}. Hence, it has been demonstrated that improving bowel/bladder functions for SCI patients is among their most highly rated concerns.

There are two patterns of neurogenic bowel. Upper motor neuron (UMN) and lower motor neuron (LMN) bowel syndromes. These syndromes are summarized by the referenced review article:

The UMN bowel syndrome, or hyperreflexic bowel, is characterized by increased colonic wall and anal tones. Voluntary (cortical) control of the external anal sphincter is disrupted and the sphincter remains tight, thereby promoting retention of stool. The nerve connections between the spinal cord and the colon remain intact, and therefore, there is preserved reflex coordination and stool propulsion. The UMN bowel syndrome is typically associated with constipation and fecal retention at least in part due to external anal sphincter activity. Stool evacuation in these individuals occurs by means of reflex activity caused by a stimulus introduced into the rectum, such as an irritant suppository or digital stimulation.

LMN bowel syndrome, or areflexic bowel, is characterized by the loss of centrally-mediated (spinal cord) peristalsis and slow stool propulsion. LMN bowel syndrome is commonly associated with constipation and a significant risk of incontinence due to the atonic external anal sphincter and lack of control over the levator ani muscle that causes the lumen of the rectum to open.

The above protocol is part of the process to get our SCI patients as close to normal and a routine as possible. Manageable bowel activity is possible and should be a priority starting from patients' ICU admission. Disregard for an appropriate bowel regimen can result in colonic distension and autonomic dysreflexia which can be potentially fatal. Moreover, alternating bouts of constipation and diarrhea result in unnecessary work for nursing staff, inappropriate ordering of tests such as C. diff checks and incorrect diagnoses of ileus. Emphasizing a healthy bowel regimen early, with the help of the stated protocol, can help avert a great deal of distress both for the patients as well as their providers.

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Genitourinary care of Spinal Cord Injury Patients

Prior to discontinuing an indwelling urinary catheter the following criteria should be met:

- Total intake should be minimized. (E.g. discontinue maintenance IV fluids, de-escalate IV antibiotics, etc.)
- Total output should equate to bladder volumes of less than 500 ml every four hours.

Once the indwelling catheter is removed proceed with the following:

Did the patient void spontaneously prior to four hours post-catheter removal?

- Check residual bladder volumes via bladder scanner
 - If residual bladder volumes are greater than 150 ml Q4h x 2, begin intermittent urinary catheterization (straight cath) Q4H.
 - If urine output is <500 ml with each straight cath Q4h x 2, may de-escalate to Q6h
 - If residual bladder volumes are less than 150 ml q4h x 2, routine assessment of bladder volumes may be discontinued

Did the patient fail to spontaneously void at the 4 hour mark?

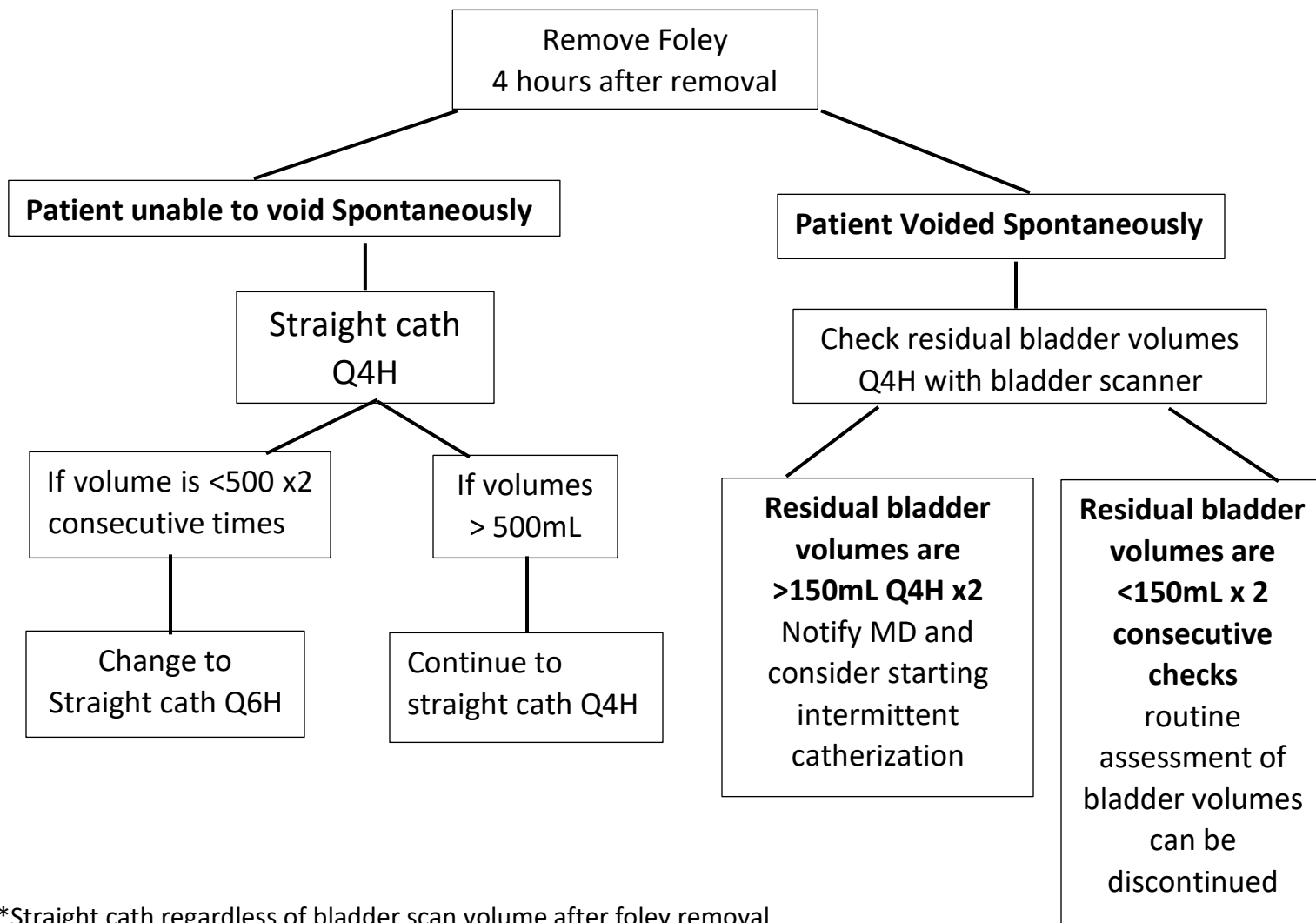
- Straight cath the patient at the 4 hours mark.
 - If urine output is greater than 500 ml, straight cath again at the 4 hour mark.
 - If urine output is greater than 500 ml, straight cath again at the 4 hour mark after previous straight cath.
 - If urine output **remains greater than 500 ml** at the fourth straight cath, insert indwelling urinary catheter.
 - If urine output is **less than 500 ml Q4h x 2 consecutive times**, straight cath Q6h while monitoring bladder volumes

When able, times for urinary catheter removal and subsequent straight caths should be standardized and scheduled to prevent delays, urinary retention and the

development of autonomic dysreflexia as well as to prevent significant variations in bladder volumes.

SCI Patient Bladder Catheter Removal Protocol

Remove foley after urine output <2000mL in a 24-hour period



*Straight cath regardless of bladder scan volume after foley removal

**If volumes 500+ with Q6H caths then go back to Q4H and follow algorithm

References

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Skin care of Spinal Cord Injury Patients

- Spinal cord injured patient are at high risk for DVTs and skin breakdown
 - Goals of skin protection are to prevent skin breakdown and pressure ulcers, as well as maintain a high degree of mobility

Skin Breakdown Prevention Guidelines

- Remove Philadelphia collar and replace with Aspen collar ASAP
- Minimize number of sheets under patient
- Moisturize dry skin Q12h
- Apply Mepilex to sacrum and heels for pressure site protection
 - Reassess q8h
 - Change q3days
- Consider PRAFO orthotic or prevalon boot to protect heel and prevent foot drop
 - If foot orthotic is used apply only a single orthotic and alternate between feet
- Turn patient q2h. Patient should alternately be positioned right-side down, left-side down, and supine
 - Using turning clock to schedule turns
 - Utilize pillows, wedges, sundance positioning pillow and/or sundance tortoise positioning device as needed to achieve the following goals
 - Positioned 30-45 degrees on the side to offload ipsilateral gluteal prominence and shoulder blade
 - Protect opposite greater trochanter
 - Maintain knees in a flexed position and protect all pressure points (eg. Pillows between knees and ankles)
- Patient is to be out of bed to chair q8h for no more than two hours at a time.
 - While patient is OOB pressure offload by weight shifting q15-30 min

- This will also include reclining patient fully for 60 seconds at least every 30 minutes
 - If patient's upper extremities are active educate patient immediately to assist with pressure offloading body
 - Patient will be OOB to Tilt-In-Space chair. No overlay necessary
 - If Tilt-In-Space chair is unavailable patient will be OOB to chair with chair overlay in place
 - ACE wrap to bilateral lower extremities and abdominal binder in place when OOB
- Maxi-slide is to be used for all repositionings.

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Analgesic care of Spinal Cord Injury Patients

Monitor pain using a pain visual analog score.

Consider non-narcotic pain control regimens including the following:

- Acetaminophen (Tylenol or Ofirmev) 1g PO or IV, Q6h
- Cyclobenzaprine (Flexeril) 10 mg, PO, Q8h
- Methocarbamol (Robaxin) 1,500 mg, PO, Q6h

Neuropathic Pain

Many SCI patients on the trauma service will have concomitant bony injuries of the spine as well as injuries elsewhere. Consider both somatic and neuropathic pain control when designing a regimen. For neuropathic pain control, consider the following:

- Pregabalin 75 mg, PO, Q12h
 - May take approximately two days to show benefit
 - Titrate to 150 mg PO, Q12h over one week as needed
 - Maximum dose of 300 mg PO, Q12h after 2-3 weeks
 - Taper over at least one week
 - Adjust for renal dysfunction (discuss with your pharmacist)
- Gabapentin 100 mg, PO, Q8h x 24h, then 200 mg, PO Q8h x 24h, then 300mg, PO Q8h
 - May titrate to a maximum dose of 2400 mg/d over 2-3 weeks
- Amitriptyline 25 mg PO, Qhs
 - May titrate to a maximum dose of 100 mg over one week
 - Consider in patients who are being treated concomitantly for depression

Spasticity

Spasticity should be maximally controlled for patient comfort and improve tolerance of physical/occupational therapy. Consider treatment with the following:

- Baclofen 5 mg, PO, Q8h
 - Increase by 5 mg/dose every three days to a maximum of 80 mg/day
 - Consider Q6h regimen if needed
 - Benefit may not start until day 3, with full effect likely at 5-10 days
 - Do not **abruptly** stop the drug (black box warning; potential for death)
- Tizanidine 2 mg, PO, Q8h
 - Increase 2 mg/dose every 1-4 days with a maximum dose of 36 mg/day
 - Slowly taper while monitoring for signs of withdrawal (tachycardia)
 - Benefit usually starts in 1-2 hours
- Diazepam 2 mg, PO, Q8h
 - May increase dose upto 10 mg Q6h as needed
 - Generally used as an adjunct
 - Benefit is generally rapid; does not last as long as alternatives
 - Do not stop abruptly; taper medication when appropriate

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Endocrine Care of Spinal Cord Injury Patients

Clinical trials have suggested that initiating high dose glucocorticoids in acute, non-penetrating SCI has been associated with improved neurologic outcomes. Any initiation of steroids must be cleared through **both** the spine surgeon and trauma surgeon involved in the care of the patient.

- If the spine surgeon of record determines that there is a potential benefit, glucocorticoids may be considered for a spinal cord injury patient who does not have any of the following exclusion criteria:
 - >8 hours from onset of injury
 - < 18 years of age
 - Penetrating mechanism of injury
 - Open fractures
 - Immunosuppression
 - Diabetes Mellitus
 - Pulmonary injury
 - Abdominal injury
 - Significant aspiration
 - COPD
- If a patient is determined to require glucocorticoids as pharmacologic neuroprotection the following is initiated:
 - Methylprednisolone (Solumedrol) 30 mg/kg, IV, bolus infusion over 15 minutes, followed by
 - Methylprednisolone (Solumedrol) 124 mg/kg, IV, drip infusion divided over 23 hours.
- Significant side effects of high-dose steroid medications include infections and sepsis, and wound complications
 - To minimize the wound healing effects of steroids consider starting vitamin A supplementation
 - Vitamin A (Aquasol A) 10-15,000 IU, PO, Daily for 7 days

Appendix

The use of high dose steroids in the treatment of spinal cord injury patients is highly contentious and controversial. Animal experiments determined that the administration of glucocorticoids after a spinal cord injury reduced edema, prevented intravascular potassium depletion, and improved neurologic recovery.¹ Subsequently the NASCIS II and NSCIS III randomized controlled trials were conducted^{2,3}. These trials suggested a **modest improvement in motor function** but no long term functional improvement.

Several experts, however, believe the beneficial effect of methylprednisolone are minimal with only marginal improvements in motor function and the infectious complications can be severe.⁴ Hence, several society guidelines have suggested that steroid administration be altogether avoided in spinal cord injury, or be considered an *option* and not a *standard*.^{5, 6, 7, 8, 9}

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John Michael Moore Trauma Center

Hematologic Care of Spinal Cord Injury Patients

Initiate early chemical and mechanical VTE prophylaxis.

- All spinal cord injury patients should immediately have sequential compression devices (SCDs) placed for mechanical VTE prophylaxis unless a specific contraindication exists.
- Initiate chemical VTE prophylaxis with enoxaparin on admission if no surgical intervention is performed, or at **48 hours post-op** unless a specific contraindication exists.
 - **Low Molecular Weight Heparin Dosing Schema:**
 - BMI < 19: Consult pharmacy
 - BMI 19-24: Enoxaparin 30 mg SQ Q12h
 - BMI 25-29: Enoxaparin 0.5 mg/kg adjusted body weight SQ Q12h
 - BMI > 30: Enoxaparin 0.5 mg/kg total body weight SQ Q12h
- IVC filters should not be used unless **both mechanical AND chemical prophylaxis** can not be used for VTE prophylaxis.
- If mobility is expected to remain impaired for > 2 weeks, discharge the patient with a home enoxaparin bridge to warfarin (INR goal 2-3) for at least 3 months.

Treatment (i.e. therapeutic anticoagulation) of DVTs in spinal cord injury patients should be individualized. In the post-operative spine patient, full anticoagulation may be started after a clear and timely discussion with the operating surgeon.

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