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Management of spinal cord injury in general practice – Part 1

Background

Spinal cord injury (SCI) is a complex disability, often requiring specialised knowledge and expertise to manage multisystem impairments. This topic is often not included in undergraduate medical education and the general practitioner may understandably feel underprepared for managing a patient with SCI.

Objective

This two part article provides an overview of common complications associated with SCI.

Discussion

Common management issues in SCI include neurogenic bladder and bowel dysfunction, autonomic dysreflexia, sexuality, fertility, pressure areas, pain, spasticity, musculoskeletal disorders, neurological complications, general health and psychological wellbeing. Armed with key information, management of the patient with SCI can be systematically addressed in the general practice setting.

■ **The general practitioner plays a vital role in the primary care of people with spinal cord injury. In a previous article we illustrated how practice structures can accommodate people with disabilities, allowing health surveillance/promotion and disease prevention activities through a comprehensive, systematic approach.¹**

Bladder function

Spinal cord injury (SCI) disrupts bladder control, affecting reciprocal coordination between detrusor and bladder neck, proximal urethra and pelvic floor muscles. Bladder management methods include:

- clean intermittent self catheterisation (CISC) with an anticholinergic medication such as oxybutynin hydrochloride (preferred method where sufficient hand function exists)
- drainage by permanent indwelling urethral or suprapubic catheter, or
- reflex voiding with an external collecting device.²

Urological complications are common after SCI, including urinary tract infection (UTI), nephrolithiasis, epididymo-orchitis, urethral stricture/false passage, and rarely, bladder cancer in those with a permanent catheter for more than 15–20 years.³ Conditions affecting the general population should not be forgotten (eg. benign prostatic hypertrophy). Sustained high bladder filling pressures from poorly controlled detrusor hyperactivity and/or reduced bladder compliance may cause upper tract deterioration, hydronephrosis and renal failure. Renal function should therefore be routinely monitored. Urea, electrolytes, creatinine, estimated glomerular filtration rate (eGFR) are recommended annually, with renal tract imaging (ie. ultrasound or intravenous pyelogram) performed every 2 years. Symptoms listed in *Table 1* should alert the GP of underlying pathology requiring expedient action.

Urinary tract infection is very common but symptoms may be altered or absent in a person with SCI. Colonisation and asymptomatic bacteriuria occurs frequently in populations with permanent catheters in situ, particularly with low pathogenic organisms such as *Pseudomonas aeruginosa* or mixed growth. Treatment (when symptomatic) should be based on microurine examination, culture and

Table 1. Urological symptoms that should alert the GP to action

- Increased frequency of UTIs
- Increased or new episodes of incontinence
- Obstructive symptoms – hesitancy, poor stream, frequency (in patients who void spontaneously)
- Lower abdominal discomfort or bladder spasms
- Passing 'grit' or 'stones' in urine
- Difficulty passing an intermittent self catheter
- Penile erosions
- Increased spasticity or episodes of autonomic dysreflexia triggered by bladder fullness or pathology
- Blood in urine, weight loss (especially if the patient is a smoker or has had an indwelling catheter for more than 15–20 years)
- Gradually increasing blood pressure
- Worsening renal blood tests if results known
- Lost to follow up with no recent (within 5 years) urological imaging or testing

sensitivity in a fresh urine specimen taken at new catheter change. Inappropriate, frequent or prolonged antibiotic therapy can predispose to development of antibiotic resistant strains over time. Criteria to assist clinical decision making are shown in *Table 2*. Recurrent UTIs call for review of patient hygiene and catheterisation techniques and consideration of further investigation to exclude bladder calculi, diverticulum or catheter cystitis. Antiseptic medications (eg. Hiprex or cranberry juice/tablets) are not effective.⁴

Gastrointestinal system and bowel management

Bowel dysfunction is common following SCI, secondary to impaired colonic transit time, altered compliance, immobility and medication side effects (eg. anticholinergics, antidepressants, narcotic analgesics).⁵ Problems such as constipation, faecal incontinence or bowel accidents, abdominal distension and pain can significantly impact on social and emotional wellbeing, participation and quality of life.⁶ Diseases such as reflux oesophagitis and gallstones causing repeated complaints of abdominal bloating or nausea are also more prevalent in SCI population, but may be misdiagnosed due to altered sensation. The prevalence of chronic gastrointestinal symptoms

Table 2. Guidelines for antibiotic treatment of UTIs⁴

- Antibiotics are indicated if:
 - microscopy showing WCC >100 associated with a pure growth of organisms, OR
 - leucocyte esterase of +++ to ++++ on urine dip stick, AND
 - at least one 'category 1' OR at least 2 'category 2' symptoms, as defined below:

Category 1 symptoms

- Elevated temperature (>38° core or 37.5° per axilla)
- New or worsening symptoms of autonomic dysreflexia

Category 2 symptoms

- Increased frequency or strength of muscle spasms
- Onset of urinary incontinence (eg. leaking around catheter or in between self catheterisation) despite taking usual anticholinergic medications to control detrusor overactivity
- New abdominal discomfort, unexplained by other pathology

Table 3. Key principles in management of neurogenic bowel

- Determine type of bowel impairment by doing a rectal examination. Patients often have loss of voluntary control over defecation and external anal sphincter (EAS). There are generally 2 types of bowel impairment:
 - upper motor neuron (UMN) or reflexic bowel (usually with lesions above T12). There is intact defecation and anocutaneous reflexes and hypertonic EAS with anorectal dyssynergia
 - lower motor neuron (LMN) or areflexic bowel. Patient often has loss of sacral reflexes including defecation and anocutaneous reflex and poor EAS tone
- Develop individualised regular bowel routine (daily or every second day for UMN, or twice per day or daily for LMN). Bowel care should be done at the same time every day with:
 - bowel emptying timed 20–30 minutes after a meal (to utilise gastrocolic reflex)
 - rectal emptying achieved using an enema, suppositories, anal digital stimulation and/or manual evacuation (latter being particularly helpful in LMN type bowel dysfunction)
 - positioning over toilet can assist with stool emptying

increases with time after injury, suggesting that these problems are acquired and potentially preventable.⁷

Bowel regimens should be individualised, taking into consideration the level and extent of neurological impairment, functional status, skin integrity, diet and nutritional factors, the individual's problem solving skills, social situation and lifestyle goals. Principles for establishing or adjusting a bowel program appear in *Table 3*. Bowel function and program should be reviewed regularly as part of an annual cycle of care using a structured questionnaire.¹ Modifications to management are likely to be necessary over time.

Constipation is common and can present with a range of symptoms such as abdominal distension, pain, early satiety, nausea, respiratory compromise from diaphragmatic 'splinting', worsening haemorrhoids

with rectal bleeding, autonomic dysreflexia, diarrhoea (spurious), and bowel accidents. A systematic approach begins with consideration of the patient's premorbid GI function, concurrent conditions, current bowel regimen, medication use and defaecation procedure (time taken for evacuation, frequency, amount and stool consistency, eg. using Bristol Stool Chart). Fundamentals of treatment are adequate hydration, a high fibre diet, regular bowel care, minimisation of constipating medications and appropriate aperients when required (*Table 4*).

Haemorrhoids with per rectal (PR) bleeding are also common,⁸ often worsened by manual procedures, enemas and constipation. Minor symptomatic cases can often be managed with topical therapy or banding. In some, haemorrhoids can act as a trigger for autonomic dysreflexia and can cause recurrent major bleeding,

Table 4. Advice for management of common bowel problems

Problem	Possible causes	Recommended action/s	Notes
Hard, dry and pebbly stool or no result from bowel care	Medication side effects Poor fluid intake (<1.5 L/day) Insufficient stool softeners and/or bulking agents Decrease in physical activity Change in bowel regimen or lifestyle factors	Review and rationalise medications (eg. narcotic analgesics) Increase fluid intake (≥2 L/day) and review dietary fibre intake Increase softeners or decrease bulking agents; review timing If using physical interventions, review technique. If not, consider adding to assist bowel motion Patient may require disimpaction if hard mass is found on PR Review bowel regimen and change lifestyle factors	An increase in fibre needs to be matched with an increase in fluid intake or the constipation can worsen If the patient has good results one day and minimal on alternate days, bowel pattern may be changing and may be more suited to an alternate bowel program Avoid irritant stimulants if possible
Alternating 'diarrhoea' (spurious) and hard, inspissated stools (indicates impaction)	Irregular bowel regimen (eg. taking stool softeners and/or bulking agents once per day or every second day) ± some or all of the above contributing factors High impaction with spurious diarrhoea Bowel pattern may be changing	Abdominal X-ray may help to confirm diagnosis In addition to the above, ensure aperients are taken on a regular basis (ie. twice per day) Start a stool chart and review if bowel routine is adequate	If abdominal X-ray reveals proximal GI faecal loading, Movicol 8 sachets over a day may be effective Avoid irritant stimulants if possible
Soft, poorly formed stool (loose or diarrhoea)	Excessive laxative use and/or medication side effects Recent antibiotic use Excess fluid intake Unbalanced diet (insufficient insoluble fibres) Too much alcohol and highly spicy food Gastrointestinal illness	Review medications and aperients (consider adding or increasing bulking agents or decrease softeners) Consider stool culture and Clostridium difficile toxin if recently on antibiotics Review fluid and dietary fibre intake May need increase of digital stimulation	Poorly formed stool can be difficult to evacuate completely during bowel care, predisposing to bowel accidents
Bowel accidents	Insufficient time for bowel care or insufficient evacuation Stool too soft Certain irritant enemas or suppositories can cause incontinence or residue later in the day Change in diet Gastrointestinal illness	Allow sufficient time for bowel care Review carer's technique Review aperients and enemas or suppositories used Consider stool culture	

which should prompt consideration of haemorrhoidectomy. The presence of haemorrhoids and rectal trauma in SCI can give a high false positive rate on faecal occult blood testing and this test should not be relied on for screening of colorectal cancer (CRC). There is no definitive evidence to date to suggest that there is an increased risk of CRC in SCI and screening should follow that recommended for the general population.

Autonomic dysreflexia

Autonomic dysreflexia (AD) is a potentially life threatening condition affecting persons with a SCI lesion at or above T6 level.⁹ In these individuals, a nociceptive stimulus (*Table 5*) below SCI level triggers excessive reflex activity of sympathetic nervous system (major splanchnic outflow) causing severe vasoconstriction with rapidly rising, uncontrolled blood pressure (BP). Secondary parasympathetic activity (elevated BP sensed by aortic/carotid baroreceptors) produces vagal activation with bradycardia and blood vessel dilatation above the injury level, insufficient to lower BP.

Table 6 lists common signs and symptoms of AD. Individuals may experience combinations of symptoms peculiar to them, without all of the typical symptoms of the condition. If the condition is unrecognised or not treated promptly, intracranial haemorrhage, encephalopathy and seizures or a cardiac arrhythmia may occur, which can result in death. Remember that BP for people with high paraplegia or tetraplegia is typically low (~90–100/60 mmHg lying down and possibly lower while sitting). Autonomic dysreflexia symptoms may occur within the normal BP range for the general population (ie. 20–40 mmHg above resting systolic level).¹⁰

Autonomic dysreflexia is most commonly related to bladder/urinary tract problems such as distended or severely spastic bladder, urinary tract infection, bladder or renal stones, and epididymo-orchitis; followed by bowel problems such as constipation, rectal distension, enlarged haemorrhoids, and irritation by enema. Other causes may relate to skin (eg. pressure sores, burns, ingrown toenails) or other systems (eg. fracture, distended stomach, labour, severe menstrual cramping, sexual stimulation).

Case study

Bob, 42 years of age, with C5 incomplete tetraplegia from a motor vehicle accident 5 years ago, presents to his GP complaining of an intermittent pounding headache with flushing over his face, sweating and feeling anxious. He also mentions having experienced abdominal cramps. Using the SOS Health questionnaire,¹ the GP elicits the following history. Bob's bladder is managed by suprapubic catheter on free drainage. He reports taking oxybutynin (Ditropan) 2.5 mg twice per day, which he had reduced from 5 mg three times per day due to a dry mouth. He drinks about 2 L/day and a carer changes his catheter every month. He has had three UTIs in the past 6 months (with *Klebsiella* grown on several previous cultures). Over the past 3 weeks Bob has noticed large amounts of urinary debris and occasional blood clots with frequent catheter blockages and some leaking of urine around his SPC site and per urethra. He has abdominal distension and a feeling of fullness and discomfort in his abdomen and is spending up to 2 hours on the toilet for bowel care, with his stool 'dribbling out'. He has a second daily bowel regimen, taking Coloxyl two 120 mg tablets per day as well as 60 mL of pear juice twice per day. He uses Bisalax enemas to initiate evacuation, describing sweating during emptying and prolonged rectal discharge. Bob denies any changes in his medications or dietary intake. He reports that his skin is intact. On examination, Bob's BP is 139/86 mmHg sitting in wheelchair, HR 90 bpm and temperature 37.6°C. He has mild abdominal distension with no tenderness or rigidity on palpation. A PR reveals an empty rectum and prolapsed haemorrhoids which bleed on contact. The SPC site looks moist but there is no skin breakdown or purulent discharge. Urine in the catheter appears cloudy and urine analysis reveals positive leucocytes and nitrites.

The GP's provisional diagnosis is AD precipitated by bladder distension caused by intermittent catheter blockages related to recurrent UTIs, as well as possible bowel impaction. The GP also considers reduced bladder compliance and/or detrusor hyper-reflexia following reduction of anticholinergic medication.

The GP discusses a management plan with Bob. He changes the suprapubic catheter and sends a fresh urine specimen for culture before starting appropriate antibiotic treatment (considering *Klebsiella pneumoniae* to be the most likely organism). He advises Bob to increase his fluid intake to 3 L/day, drinking consistently throughout the day. The GP gives him a prescription for Nitrolingual Spray and an Autonomic Dysreflexia Emergency Treatment Card. He also arranges for the community or practice nurse to educate Bob and his carers further on what to do in case another episode occurs.

The GP suggests that Bob reduce current 'softeners' and increase the fibre in his diet or add a bulking agent such as Metamucil or Normafibre to firm the consistency of his stools (checking abdominal X-ray first to exclude faecal impaction with overflow). The GP recommends a bowel chart to monitor stool consistency changes making further necessary adjustments after 3–5 bowel cycles. Bisalax can cause local irritation to the rectum and may be the cause of the discharge. Microlax enemas or glycerine suppositories are a gentler alternative in combination with digital stimulation and abdominal massage techniques to assist with facilitating the defecation reflex.

Results of investigations (catheter specimen of urine [CSU], blood tests, renal tract ultrasound and abdominal X-ray) reveal *Klebsiella pneumoniae* and a 1 cm bladder calculus. The GP refers Bob to an urologist for a cystoscopy and opinion about performing a videourodynamic study. The urologist performs a cystoscopy, removing the stone, and finds areas of reactive catheter cystitis requiring diathermy. Videourodynamics on oxybutynin 2.5 mg twice per day reveals high detrusor pressures with detrusor hyper-reflexia. The GP suggests oxybutynin be increased to 5 mg three times per day, but reminds Bob that this may cause constipation and increased aperients may be needed.

Table 5. Common precipitants of autonomic dysreflexia

- Bladder – urinary tract infection, epididymo-orchitis, bladder distension, renal tract calculus, urological procedures
- Bowel – constipation, rectal irritation, haemorrhoids
- Skin – pressure area, burn, ingrown toenail, tight clothing or stockings
- Gastrointestinal – biliary colic, appendicitis, other causes of acute abdomen
- Obstetric and gynaecological – onset of labour, severe menstrual cramping
- Musculoskeletal – fracture

Table 6. Common symptoms and signs of autonomic dysreflexia

- Sudden hypertension
- Pounding headache
- Bradycardia
- Flushing/blotching of skin above spinal injury level
- Profuse sweating above spinal injury level
- Skin pallor and piloerection below spinal injury level
- Chills without fever
- Nasal congestion
- Blurred vision (dilatation of pupils)
- Shortness of breath, sense of apprehension or anxiety

If AD is suspected, get help and do not leave the patient alone. Sit the person upright (to buffer rise in BP), loosen tight clothing, compression stockings and abdominal binders. Ask if they suspect a cause (usually due to bladder or bowel problems). Identify the cause (eg. check bladder drainage equipment for kinks, obstruction to flow of urine into bag). Monitor BP every 2–5 minutes. Gentle irrigation (with ~30 mL saline) can be tried to unblock a nondraining catheter.² If unsuccessful or bladder is managed by CISC or reflex voiding, catheterise the patient using a generous amount of lubricant containing local anaesthetic gel (eg. 2% lignocaine jelly). Similarly, if constipation is suspected, check the rectum for faecal material and evacuate the contents after anaesthetic gel is inserted for several minutes.⁷ If BP is >150 mmHg, drug treatment should be commenced.

Glyceryl trinitrate (GTN) can be given sublingually as a spray (Nitrolingual) or half to one tablet (Anginine) or transdermally (5 mg patch), repeated every 5–10 minutes up to three times. If GTN is unavailable or contraindicated, give nifedipine (10 mg tablet crushed, mixed with water and swallowed).¹¹ In severe cases with a persisting noxious stimulus, parenteral treatment or epidural anaesthesia may be necessary. Close monitoring after the episode is recommended. A MedicAlert bracelet may be considered along with carrying GTN and an Autonomic Dysreflexia Medical Emergency Card to alert medical personnel of this condition.

Warning: do not use GTN spray/tablets if phosphodiesterase (PDE5) inhibitors such as sildenafil (Viagra), vardenafil (Levitra) have been taken in the previous 24 hours or tadalafil (Cialis) within last 48 hours.

Conclusion

Secondary prevention and management of conditions arising from SCI require specialised knowledge. Common diseases also occur, but may not present typically because of altered sensation, therefore calling for a high degree of suspicion. The GP is ideally placed to deliver first line care and support for people with SCI.

Resources

- Clinical information booklets, listed below, are available at www.ciap.health.nsw.gov.au by following the specialties link:
 - Middleton J. Management of the neurogenic bladder for adults with spinal cord injuries
 - Stolzenhein G. Management of the neuropathic bowel for adults with spinal cord injuries
 - Middleton J. Treatment of autonomic dysreflexia for adults with spinal cord injuries: a medical emergency
- Jannings W, Temblett J, Cairns G, Pryor J. Solving common bowel problems. A resource tool for persons with spinal cord injury. Rehabilitation Nursing Research & Development Unit 2002. Available from the Continence Foundation – telephone 1800 330 066
- Autonomic Dysreflexia Medical Emergency Card. Telephone SSCIS 02 9808 9666.

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References

1. Mann L, Middleton JW, Leong G. Fitting disability into practice: focus on spinal cord injury. *Aust Fam Physician* 2007;36:1039–42.
2. Consortium for Spinal Cord Medicine Clinical Practice Guidelines. Bladder management in adults with spinal cord injury: a clinical practice guideline for healthcare providers. *Paralyzed Veterans of America*, 2006.
3. Groah SL, Weitzenkamp D, Lammertse DP, et al. Excess risk of bladder cancer in spinal cord injury: Evidence for an association between indwelling catheter use and bladder cancer. *Arch Phys Med Rehabil* 2002;83:346–57.
4. Lee BB, Haran MJ, Hunt LM, et al. Spinal-injured neuropathic bladder antiseptics (SINBA) trial. *Spinal Cord* 2007;45:542–50.
5. Lynch AC, Antony A, Dobbs BR, Frizelle FA. Bowel dysfunction following spinal cord injury. *Spinal Cord* 2001;39:193–203.
6. Rajendran SK, Reiser JR, Bauman W, Zhang RL, Gordon SK, Korsten MA. Gastrointestinal transit after spinal cord injury: effect of cisapride. *Am J Gastroenterol* 1992;87:1614–7.
7. Consortium for Spinal Cord Medicine Clinical Practice Guidelines. Neurogenic bowel management in adults with spinal cord injury. *Paralyzed Veterans of America*, 1998.
8. Stone JM, Nino-Murcia M, Wolfe VA, et al. Chronic gastrointestinal problems in spinal cord injury patients: a prospective analysis. *Am J Gastroenterol* 1990;85:1114–9.
9. Cole TM, Kottke FJ, Olson M, et al. Alterations of cardiovascular control in high spinal myelomalacia. *Arch Phys Med Rehabil* 1967;48:359–68.
10. Consortium for Spinal Cord Medicine Clinical Practice Guidelines. Acute management of autonomic dysreflexia: individuals with spinal cord injury presenting to healthcare facilities. 2nd edn. *Paralyzed Veterans of America*, 2001.
11. Braddom RL, Rocco JF. Autonomic dysreflexia: a survey of current treatment. *Am J Phys Med Rehabil* 1991;70:234–41.