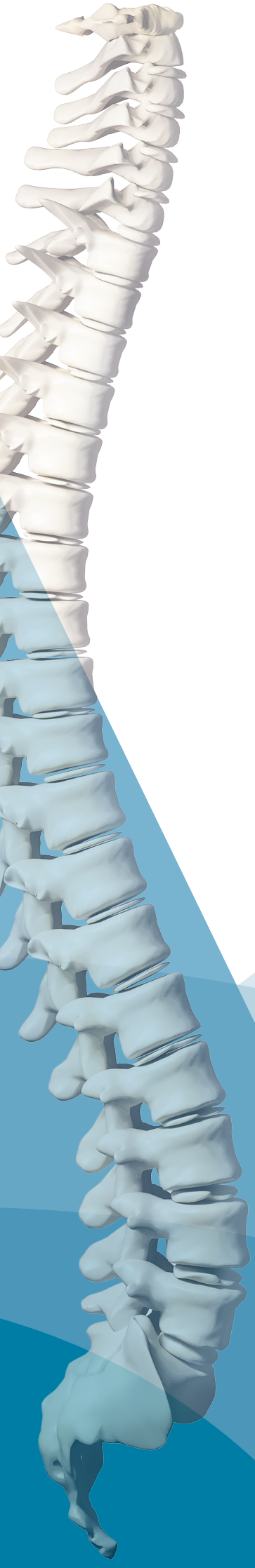

ACI NSW Agency for Clinical Innovation

Treatment of Autonomic Dysreflexia for Adults & Adolescents with Spinal Cord Injuries

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All recommendations are for patients with spinal cord injury as a group. Individual therapeutic decisions must be based on clinical judgment with a detailed knowledge of the individual patient's unique risks and medical history, in conjunction with this resource.

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1. INTRODUCTION

Autonomic dysreflexia, also referred to as autonomic hyperreflexia, is a potentially life-threatening condition, which affects individuals with spinal cord injury (SCI) above the major splanchnic outflow (typically from a lesion at or above the T6 neurological level).[1] It is a condition that many general practitioners and medical and nursing staff in emergency departments have insufficient knowledge about and require further education to rectify this.[2, 3] Delayed diagnosis or untreated autonomic dysreflexia can result in various significant complications, including stroke, seizures, myocardial ischaemia, and death. In a recent review of 32 cases of death or life-threatening complications, the majority (72%) were related to central nervous system (CNS) complications of stroke or seizures, 22% were cardiovascular-related, and two cases (6%) were complicated by pulmonary oedema. Six of the 7 deaths were due to CNS complications, with the other fatality due to pulmonary oedema.[4]

2. PATHOPHYSIOLOGY

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, overactivity of sympathetic ganglia remains uncontrolled due to isolation of the spinal cord below the injury from normal regulation by vasomotor centres in the brainstem (refer to **Figure 1**). Release of substances, such as noradrenaline, cause severe vasoconstriction with skin pallor, piloerection and a sudden rise in blood pressure (BP), which is usually accompanied by a pounding headache.

Parasympathetic activity above the level of SCI occurs when the rise in BP is sensed by baroreceptors in the aortic arch and carotid bodies, resulting in compensatory bradycardia (via the vagus nerve). Flushing due to dilatation of blood vessels, which is probably also responsible for headache, and profuse sweating above the level of injury also occur (via sympathetic inhibitory outflow from vasomotor centres). However, both of these mechanisms are insufficient to satisfactorily control paroxysmal hypertension due to massive sympathetically-mediated vasoconstriction of the splanchnic bed.[5]

2.1 Common Causes include:

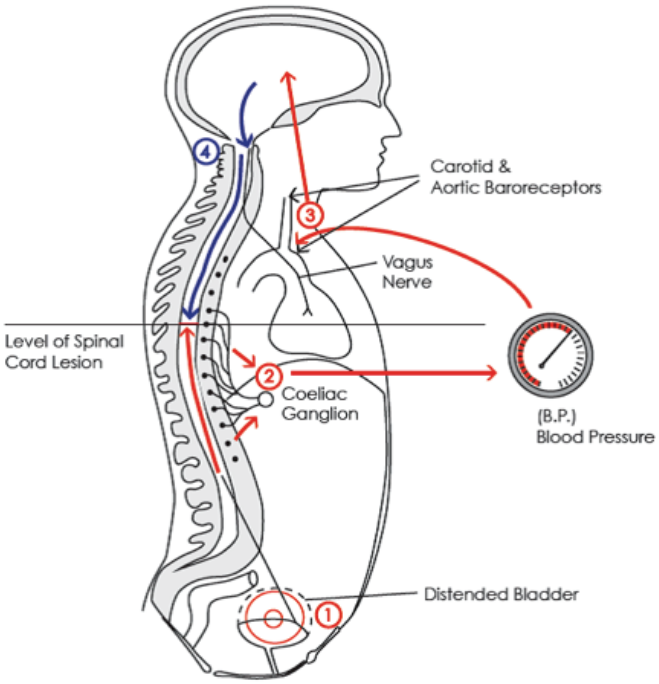
Bladder: Distended or hyperactive bladder, urinary tract infection, bladder or kidney stones, urological procedure or even inserting a catheter.

Bowel: Constipation, faecal impaction, rectosigmoid gaseous distension, rectal irritation (e.g. enema or manual evacuation), haemorrhoids.

Skin: Ingrown toenail, burns, pressure area, tight clothing.

Other: Any irritating stimulus, including fracture, epididymoorchitis, distended stomach, sexual intercourse, labour or severe menstrual cramping. Note that autonomic dysreflexia may be caused iatrogenically by staff clamping a catheter for a procedure such as a bladder ultrasound or kinking catheter by accident in operating theatre during a surgical procedure.[1]

Figure 1: Pathophysiological mechanisms involved with autonomic dysreflexia



1. Strong sensory stimulus from below level of lesion (e.g. distended bladder) conveyed into spinal cord.
2. Signals transmitted up spinal cord initiate massive reflex sympathetic activation, causing widespread vasoconstriction (involving splanchnic blood vessels), and resulting in severe hypertension.
3. Brain detects a sudden rise in BP (via signals from baroreceptors in aortic and carotid vessels carried in the 9th and 10th cranial nerves).
4. Brain responds and attempts to control BP sending descending primary inhibitory impulses from brainstem via spinal cord (which are blocked). Impulses travelling through the vagus nerve cause secondary bradycardia.

2.2 Symptoms and Signs

Common symptoms and signs of autonomic dysreflexia are listed in **Table 1**. Autonomic dysreflexia in a person with SCI can present in a variety of ways and vary in intensity from mild discomfort to a severe, life threatening medical emergency. Typically, the patient will complain of a pounding headache with flushing and profuse sweating above the level of spinal lesion, with or without other symptoms such as nasal congestion (stuffiness), blurred vision, shortness of breath and/or anxiety. Skin pallor and piloerection (goose bumps) can be seen below the level of spinal lesion.

BP is significantly elevated (at least 20 to 40 mmHg above normal resting systolic level or 15 to 20 mmHg above resting systolic level in adolescents).[1, 6] It is important to remember that BP for individuals with high paraplegia or tetraplegia may usually be low, around 90 to 100/60 mmHg while lying down and possibly lower whilst sitting. Therefore, patients with SCI may become symptomatic with BP in the normal range for the general population.

Autonomic dysreflexia is a **medical emergency**! If unrecognised or not treated promptly the BP may rise to dangerously high levels and precipitate intracranial haemorrhage, encephalopathy and seizures, cardiac arrhythmia, or death.

Women with spinal cord injury above the mid thoracic region (T6 level) who are pregnant may experience autonomic dysreflexia as the first sign of the commencement of labour.

Table 1: Common symptoms and signs of autonomic dysreflexia (hyperreflexia)

- 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
- irritability or combative behaviour (in people with limited cognitive and communication skills).

Note: Autonomic dysreflexia can present with a variety of symptoms and can vary in intensity from being asymptomatic or mild discomfort and headache to a life-threatening emergency.

3. TREATMENT

Remember: this is a **medical emergency**, do not leave the patient alone. One person should monitor BP while another provides treatment.

3.1 Initial Steps

- Ask patient and carer if they suspect a cause.
- Elevate the patient's head and lower the legs (this will help lower BP while cause is identified).
- Loosen any constrictive clothing, such as an abdominal binder or compressive stockings.
- Check bladder drainage equipment for kinks or other causes of obstruction to flow, such as clogging of inlet to leg bag or overfull leg bag.
- Monitor BP every 2 to 5 minutes.
- Avoid pressing over the bladder.[6]

3.2 Further Treatment

If symptoms persist or BP remains elevated following the above efforts or a cause cannot be readily identified, pharmacological treatment with a short acting antihypertensive medication should be commenced concurrently with the search for and treatment of the noxious stimulus.[7] There are no studies showing the precise point at which BP becomes dangerous. The Consortium for Spinal Cord Medicine guideline adopts 150 mmHg systolic BP as the value at which pharmacological treatment should be considered when BP remains elevated with a cause either not yet identified or able to be quickly reversed, such as by passing a catheter. This is certainly true when performing the procedure of manual evacuation and autonomic dysreflexia will be exacerbated during prolonged nociceptive stimulus to remove faecal matter.[6] The BP threshold at which medication is given prior to more straightforward procedures, such as catheterisation, may vary (e.g. 170 mmHg) requiring clinical judgement about whether any difficulty or delay is anticipated. The reason for this is that sudden decompression of a large volume of urine in the bladder may cause significant hypotension if the individual has already been given pharmacological agents to decrease BP (refer to Treatment Algorithm).[8]

3.2.1 For a person with an indwelling urethral or suprapubic catheter

- If a blocked catheter is suspected, empty the leg bag and estimate volume. To help determine if bladder is empty or not, consider patient's fluid intake and output earlier that day and normal pattern of drainage. A bladder scan will enable the bladder volume to be estimated objectively.
- If catheter seems blocked, irrigate the bladder GENTLY with no more than 10 to 15mls of sterile normal saline at body temperature.
- If urine does not drain after irrigation, re-catheterise using a generous amount of lubricant containing a local anaesthetic (e.g. lignocaine 2%) gel. In men with an indwelling urethral catheter, remove the catheter, ensure adequate anaesthetic jelly is inserted into urethra and prevented from leaking out by squeezing the tip of the penis for 2 to 3 minutes, to relax external urethral sphincter prior to re-catheterisation. Replace the catheter, drain urine and **be alert for sudden hypotension due to rapid draining of an over-distended bladder** and/or sudden resolution of autonomic dysreflexia.

3.2.2 For a person wearing an external collection device or doing intermittent self-catheterisation

If bladder is distended and patient is unable to void in their usual manner, lubricate the urethra with a generous amount of lignocaine 2% gel, wait 2 to 3 minutes and then pass a catheter to empty the bladder. Drain urine and **be alert for sudden hypotension due to rapid draining of an over-distended bladder** and/or sudden resolution of autonomic dysreflexia. Leave catheter in situ until reason for retention is identified and remedied.

Note that if BP declines after the bladder is empty, the person still requires close observation as the bladder can go into severe contractions causing hypertension

to recur (see section 3.4 Pharmacological Treatment). If the catheter is left in situ, an anticholinergic medication, such as oxybutynin (Ditropan) 5mg tds, can be given. In situations in which an anticholinergic medication is started in a person who normally voids by reflex bladder contraction, an adequate 'washout' period with cessation of medication for at least 12 hours prior to catheter removal may help to avoid possible retention.

3.3 For Faecal Evacuation

If you are sure the bladder is empty and symptoms persist, apply a generous amount of lignocaine gel onto the anal sphincter region and into the rectum. Wait five minutes before gently inserting a finger to remove faecal matter.

Note that BP may rise rapidly during rectal examination with the person positioned in side-lying. Monitor BP manually before commencing and during digital stimulation. If BP increases significantly, cease digital stimulation and only recommence following administration of suitable medication (refer to Treatment Algorithm).[8]

3.4 Pharmacological Treatment

Note: BEFORE ADMINISTERING PHARMACOLOGICAL TREATMENT, ALWAYS CHECK FOR INTERCURRENT AND/OR RECENT USE OF MEDICATION FOR ERECTILE DYSFUNCTION

3.4.1 Oral agents

Glyceryl Trinitrate (GTN)

DO NOT USE GTN SPRAY, TABLETS OR PATCH IF A MEDICATION FOR ERECTILE DYSFUNCTION SUCH AS SILDENAFIL (VIAGRA), VARDENAFIL (LEVITRA) HAS BEEN TAKEN IN LAST 24 HOURS, OR TADALAFIL (CIALIS) HAS BEEN TAKEN WITHIN THE LAST 48 HOURS OR 96 HOURS IN THE ELDERLY (Refer to Manufacturers Product Information for further pharmaceutical information because extremely severe reductions in BP may occur).

- Administer one spray of glyceryl trinitrate (GTN) (400 mcg Nitrolingual Pump Spray), OR if spray is unavailable, place half GTN tablet (300 mcg Anginine) under tongue, or apply one 5 mg/24 hours GTN Transdermal Patch to chest or upper arm (patch should be removed as soon as hypertension resolves).
- Monitor BP; if little or no effect in 5 to 10 minutes, administer a second spray or other half tablet of Anginine under tongue. **Be cautious, particularly with elderly patients**, when administering GTN spray or tablet as sudden hypotension may result, particularly if cause is quickly remedied. If symptomatic hypotension occurs lay the person down and elevate the legs or tilt bed down. Dosage may be titrated by removing the transdermal patch or by spitting out residual spray or tablet with hypotensive effect being shorter lasting.
- Up to 3 doses can be given in 30 minutes (if BP remains elevated or rises rapidly intravenous medication may be indicated - see below).

DO NOT USE SILDENAFIL (VIAGRA), VARDENAFIL (LEVITRA), TADALAFIL (CIALIS) OR OTHER DRUG IN THE PDE 5 INHIBITOR (NITRIC OXIDE ENHANCER) CLASS WITHIN 24 HOURS OF TAKING GTN SPRAY OR TABLETS.

Alternative (Short Acting) Oral Agents

- If GTN spray, tablets or patch are unavailable, or contraindicated due to recent use of medication for erectile dysfunction within the last 24 hours (or 48 to 96 hours if Cialis) and depending on age (see guidelines above), an alternative (short-acting) antihypertensive agent, such as captopril should be used. Captopril, administered sublingually as a 25mg tablet (taking about 3 minutes to dissolve under the tongue), has been shown to effectively lower BP within 15 minutes of administration.[9]

ADVANTAGES OF SUBLINGUAL ADMINISTRATION OF CAPTOPRIL ARE THAT THE DRUG ENTERS THE GENERAL CIRCULATION DIRECTLY, WITH THERAPEUTIC CONCENTRATIONS AND ONSET OF ACTION ACHIEVED MORE RAPIDLY. IN ADDITION, THE PARTIALLY DISSOLVED TABLET MAY BE SPAT OUT IF THERE IS A VERY RAPID REDUCTION IN BP. [10]

NB. Nifedipine capsules (bite and swallow), which were previously used in this situation are no longer available, having been withdrawn due to risks that have been associated with use in the able-bodied population with angina, or hypertension. The pharmacokinetics of nifedipine oral tablets is less ideal for rapid reduction in BP with delayed onset and longer action. [11]

3.4.2 Parenteral Agents

If hypertension is not relieved by GTN or other oral antihypertensive agent, then administration of parenteral antihypertensive medication will be required in an acute hospital setting. Adequate analgesia (e.g. morphine) should be administered where there is a known cause of ongoing nociception until resolved. Where control of the noxious stimulus is difficult, regional epidural anaesthesia may be appropriate.[7, 8]

NB. An acute episode of autonomic dysreflexia can lead to an **increased susceptibility** to further episodes due to excess circulating catecholamines. These may be precipitated by activities which would not normally do so, such as performing muscle stretches, bowel care, or other activities. The patient must be alerted to this possibility and monitored appropriately for 48 to 72 hours. During this time invasive procedures should be kept to a minimum.

IF A CAUSE CAN NOT BE FOUND AND / OR THE PROBLEM REMAINS UNRESOLVED, PLEASE CONTACT THE NEAREST ACUTE SPINAL CORD INJURIES UNIT AND/OR SPINAL CORD INJURIES UNIT PHYSICIAN ON CALL FOR FURTHER ADVICE AND MANAGEMENT.

- Royal North Shore Hospital
  Main Switch (02) 9926 7111
- Prince of Wales Hospital
  Main Switch (02) 9382 2222
- Sydney Children's Hospital
  Main switch (02) 9382 1111
- The Children's Hospital at Westmead
  Main Switch (02) 9845 0000

TABLE 2: Summary of recommended treatment/interventions with accompanying rationale derived from Clinical Practice Guidelines for "Acute Management of Autonomic Dysreflexia", Consortium for Spinal Cord Medicine, Paralysed Veterans of America, 2001

Action/Intervention	Rationale
Check blood pressure (BP) with **manual sphygmomanometer**. If BP is elevated: • Sit patient as **upright** as possible. • **Remove all tight clothing** including abdominal binders.	Assess if BP is elevated (NB. Automatic BP measuring devices, e.g. Dynamap, are too slow to detect rapid rises in BP). Encourages pooling of blood in lower extremities and abdomen to decrease venous return, reduce BP and buffer rises in BP.
Obtain assistance from other staff member.	Two people are required to monitor and treat patient.
Monitor BP and pulse rate every 2 to 5 minutes until episode has completely resolved.	BP will continue to rise, often rapidly, until successfully treated. It is essential to continue monitoring BP closely during all interventions and until the episode has resolved with BP returning to normal resting level.
Perform thorough survey of the patient to determine cause of autonomic dysreflexia. **Beginning with urinary system**: If catheter is in situ: • Check entire urinary drainage system for kinks, folds, blockages. • If problem such as kink identified, rectify immediately. • If catheter appears to be blocked attempt to unblock catheter by pulling back on the syringe. If block persists, gently irrigate catheter with 10 to 15 mls of normal saline at body temperature. • If catheter is not draining, remove and **replace the catheter**. OR • If catheter is not in situ **insert urinary catheter**. NB. **Prior to inserting catheter instil 2% lignocaine gel** (if readily available) into the urethra and wait 2 to 5minutes if possible. ** Be alert for sudden hypotension due to rapid draining of bladder and/or sudden resolution of autonomic dysreflexia. • If the *catheter* is draining and BP remains elevated continue to next step.	• Episode of autonomic dysreflexia will not resolve until cause is identified and rectified. • The urinary tract, particularly bladder distension, is the commonest cause of autonomic dysreflexia. • Obstruction of urine outflow may result in bladder over distension. • Allowing unobstructed flow of urine may resolve episode. • Irrigation may remove blockage. Large volume of fluid instilled in bladder may further exacerbate autonomic dysreflexia. • Existing catheter may be blocked with sediment or blood. It is essential to decompress bladder. • New catheter should run freely. • Insertion of catheter may exacerbate autonomic dysreflexia. Lignocaine gel may decrease sensory input and relax sphincter for catheterisation. • Sudden decompression of a large volume of urine, whilst expected to normalise BP, may cause hypotension, particularly if the person has already been given pharmacological agents to decrease BP. • Clinical evidence suggests urinary system is not the cause of autonomic dysreflexia.

Table continues on page 10

Table continued from page 9

Action/Intervention	Rationale
If BP continues to be elevated, suspect faecal impaction as the cause: • Instil a generous amount of local anaesthetic (e.g. Lignocaine) gel into the rectum. • Wait approximately 5 minutes before performing gentle per rectum (PR) examination. • If BP is above 150 mmHg, consider pharmacological management to lower and stabilise BP prior to PR and/or faecal dis-impaction. • If necessary perform manual evacuation using generously lubricated gloved finger - gently remove any stool which is present. • If symptoms of autonomic dysreflexia worsen, STOP manual removal immediately, administer oral antihypertensive agent and instil additional Lignocaine. Wait for 20 minutes and repeat manual evacuation. If rectal examination reveals no stool in the rectum, continue to next step.	• Faecal impaction is the second most common cause of autonomic dysreflexia. • Local anaesthetic will be required, even if patient has no or markedly reduced sensation, since PR check and/ or manual removal of faeces may exacerbate autonomic dysreflexia. • Wait to allow action of Lignocaine. • Medication will help to control precipitous rises in BP. Clinical judgement is essential when making decisions about pharmacological management, taking into account the actual BP level and how rapidly BP is changing. • If cause is faecal impaction, autonomic dysreflexia will not resolve until removal of stool. • Digital stimulation may exacerbate autonomic dysreflexia.
Commence systematic survey of patient for other causes of autonomic dysreflexia, which may include following: • pressure area • post-operative irritation or pain • ingrown toe nail • burn • fracture. If cause with persisting nociception is found, administer adequate analgesia. If no cause can be found and symptoms persist obtain assistance from Spinal Unit Consultant.	Autonomic dysreflexia will not resolve without finding and remediating the underlying cause. **Medical assistance is required** for additional treatment and control of symptoms – patient will need a thorough physical examination and may require radiological studies and intravenous pharmacological intervention to prevent complications such as stroke.
Episode is considered to be resolved when: • Cause of autonomic dysreflexia has been identified. • BP restored to normal level for individual. • Pulse returned to normal rate. • Patient is comfortable with no signs of autonomic dysreflexia. **Following resolution of episode:** • Monitor patient (BP and pulse) for 4 hours post episode. • Document episode, cause and treatment in notes.	Some 'hyperactivity' of the sympathetic nervous system may be experienced. Patient should be monitored for any exacerbation or reappearance of symptoms, so they can be quickly and appropriately treated.

Table continues on page 11

Table continued from page 10

Action/Intervention	Rationale
- Educate patient, carers, significant others.	Additional education may be required to help recognition of symptoms, treatment and strategies to avoid further episodes as much as possible. It is important to also alert the patient and carers to the possibility of increased susceptibility to further episodes over following few days.

4. QUIZ

Q1. Which statement below is TRUE regarding autonomic dysreflexia?

a) It is a reflex activity that happens during spinal shock;
b) It is unlikely to occur in an individual with paraplegia;
c) It is due to the inability of the strong sensory stimulus below the level of lesion to reach the spinal cord;
d) Sympathetic activity below the level of the lesion results in flushing and profuse sweating;
e) Parasympathetic activity above the level of the lesion results in bradycardia.

Q2. All the following are potential triggers for autonomic dysreflexia EXCEPT:

a) Fracture;
b) Ingrown toenail;
c) High BP;
d) Sexual intercourse;
e) Urinary tract infection

Q3. All the following are initial steps in managing autonomic dysreflexia EXCEPT:

a) Loosen constrictive devices such as compressive stockings;
b) Sit the patient upright and lower the legs;
c) Check the BP;
d) Apply pressure over the bladder to relieve any possible obstruction;
e) Ask the patient or caregiver what he or she thinks the trigger might be.

Q4. Regarding pharmacological management of autonomic dysreflexia, which statement below is TRUE?

a) Short acting agents such as nitrate spray, tablet or patch are first line;
b) Medications used for erectile dysfunction can assist in lowering the BP;
c) Nifedipine oral tablets are ideal for rapid reduction in BP;
d) Long acting agents can be repeated at regular intervals if the BP remains elevated;
e) Use of sublingual captopril is not recommended due to high risk of serious adverse effects.

Q5. All the following are known complications of autonomic dysreflexia EXCEPT:

a) Myocardial ischaemia;
b) Intra cerebral haemorrhage;
c) Intra-abdominal haemorrhage;
d) Seizure;
e) Cardiac arrhythmia.

QUIZ ANSWERS:

Q1. E - Parasympathetic activity above the level of the lesion via the intact vagus nerve results in bradycardia.
Q2. C - High BP is a sign of autonomic dysreflexia.
Q3. D - Applying pressure over the bladder can aggravate autonomic dysreflexia.
Q4. A - Based on current guidelines, nitrates (in various forms) are the first line agents in managing autonomic dysreflexia.
Q5. C - Intra-abdominal haemorrhage has never been reported as a complication of autonomic dysreflexia.

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6. ADDITIONAL RESOURCES

- Health Safety Alert: http://www0.health.nsw.gov.au/resources/quality/sabs/SN_014_10_pdf.asp (Accessed January 2014).
- ACI clinical practice guidelines for management of bladder, bowel, skin and other secondary conditions: http://www.aci.health.nsw.gov.au/networks/spinal-cord-injury/resources (Accessed January 2014).