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PRACTICE POINTER

Autonomic dysreflexia in spinal cord injury

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What you need to know

- Patients with a spinal cord injury above T6 are at risk of developing autonomic dysreflexia, an uncoordinated autonomic response to a noxious stimulus that occurs below the level of the spinal cord lesion.
- Serious complications can occur if autonomic dysreflexia is not recognised and treated early, including cerebrovascular haemorrhage, seizures, and cardiac arrest
- Bladder or bowel distension are the most common triggers
- Sitting the patient upright and eliminating triggers is the first line management
- Patients and their carers will likely have experience of the condition and should help to guide management, in the acute and long term setting

Autonomic dysreflexia is a potentially life threatening complication of spinal cord injury. It carries a mortality rate of 22%¹ and increases the risk of stroke

by 300% to 400%.² Clinicians working in emergency or urgent care may not see patients with this condition often, but when they do, prompt recognition and treatment are required. This practice pointer gives a brief overview of autonomic dysreflexia and how to identify it.

What is autonomic dysreflexia?

Autonomic dysreflexia is the product of dysregulation of the autonomic system, leading to an uncoordinated response to a noxious stimulus below the level of a spinal cord injury,² usually in individuals with a spinal cord injury above the level of T6 (fig 1). It is three times more prevalent in those with complete spinal cord injury than in those with incomplete injury (91% versus 27%, respectively).³ Autonomic dysreflexia is clinically defined as an acute episode of systolic blood pressure elevated 25 mm Hg or above the patient's normal measurements.² It is usually secondary to a reversible and treatable cause, such as urinary retention, constipation, or infection.

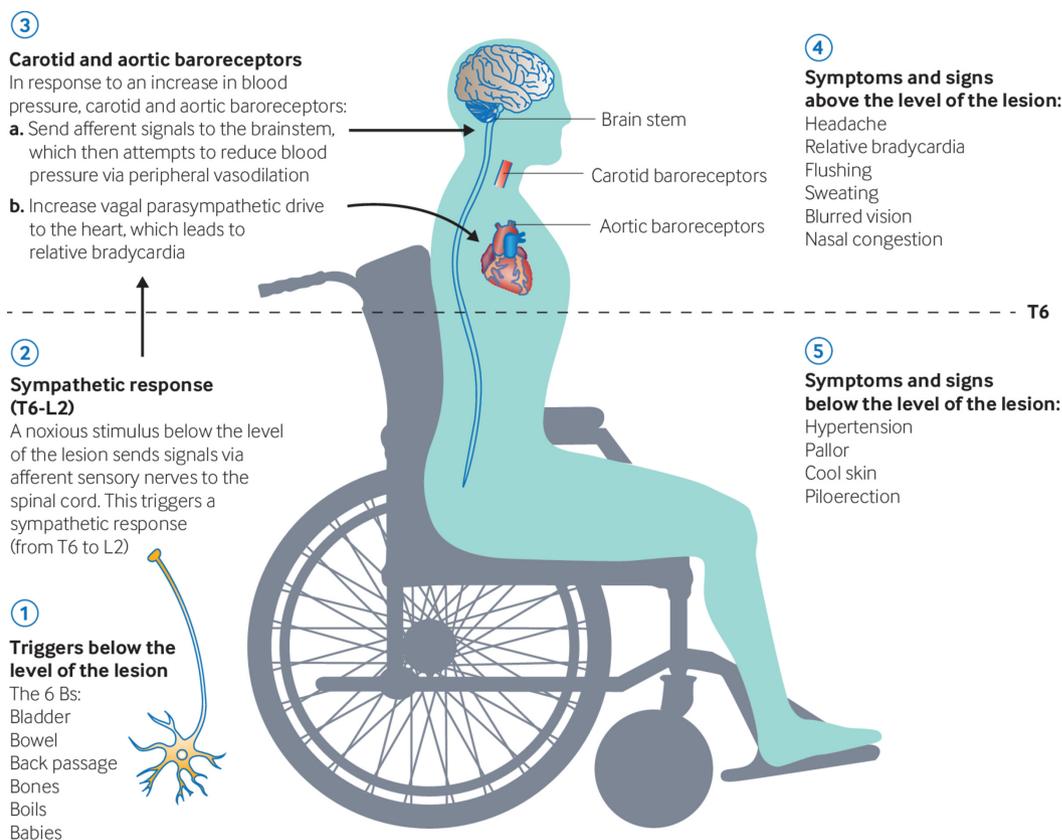


Fig 1 | Signs and symptoms of autonomic dysreflexia in spinal cord injury

How does it occur?

The noxious stimulus—for example, a distended bladder or bowel, sends signals via afferent sensory nerves to the spinal cord. This triggers a sympathetic response (from T6 to L2) of hypertension, pallor, cool skin, and piloerection,⁴ which is only clinically apparent below the level of the lesion. In response to an increase in blood pressure, carotid and aortic baroreceptors increase vagal parasympathetic drive to the heart, resulting in relative bradycardia. Baroreceptors also send an afferent response to the brainstem, which then attempts to reduce blood pressure through peripheral vasodilation. The patient experiences headache, flushing, sweating, blurred vision, and nasal congestion. These inhibitory vagal and brainstem responses are unable to pass below the level of the spinal cord lesion. The resulting unopposed sympathetic activity below this level can lead to a life threatening rise in blood pressure and its sequelae¹ if the noxious stimulus is not promptly recognised and withdrawn.

What causes it?

The most common cause is over-distension of the bladder, accounting for 75%-85% of episodes.⁵ The “6 Bs” are a reminder of possible triggers to consider in the emergency scenario: bladder (urinary tract infection or retention, stones, or distension caused by catheter blockage), bowel (constipation, impaction), boils (skin damage), bones (fractures), babies (pregnancy, sexual intercourse, breastfeeding), and back passage (haemorrhoid or fissure).⁶

How do you recognise it?

Patients frequently complain of a sudden onset, severe, throbbing headache. They may have flushing and sweating of the skin above the level of their spinal cord lesion, alongside blurred vision and nasal congestion. Sympathetic overdrive causes pallor, cool skin, and piloerection below the level of the lesion.⁴ In one study of 32 patients with life threatening autonomic dysreflexia, 23 presented with cerebrovascular signs (including haemorrhage and seizure), seven with cardiovascular related signs (including arrhythmia and cardiac arrest), and two with pulmonary oedema.¹ Symptoms are not necessarily correlated with the level of blood pressure increase.

People with a spinal cord injury above T6 typically have a systolic blood pressure of between 90 mm Hg and 110 mm Hg,⁷ so may present with symptoms of autonomic dysreflexia and a seemingly normal (or only modestly raised) blood pressure. It is therefore valuable to compare the patient's blood pressure to their baseline measurement when possible.

Ask the patient and their carer(s) about their past experience. Patients are usually experts in their condition and will be able to tell you their own precipitating factors and typical clinical presentations from previous experience.

What is the acute management? (fig 2)

Patients at risk of autonomic dysreflexia usually have a self-management plan, which includes carrying a rescue pack containing the appropriate medications (organised by their specialist team) at all times. However, occasionally a person with spinal cord injury is admitted to hospital for an unrelated condition but develops autonomic dysreflexia, which will require urgent management in the immediate absence of specialist support.

As soon as practically possible, reposition the patient in an upright position and remove any tight clothing. Systematically consider and manage potential triggers. For example, a catheter that is blocked should immediately be repositioned, irrigated, and/or replaced; insert a catheter if acute urinary retention is suspected.

Once a urinary tract stimulus has been ruled out, consider bowel triggers (most commonly constipation) next. Evacuate an overloaded rectum using lignocaine gel and gentle digital stimulation.⁷ This should trigger anal sphincter relaxation and emptying of the rectum without the need for pharmacological intervention, but a suppository can be used if required. Avoid large volume enemas, which may exacerbate autonomic dysreflexia.⁸ Following this first line management, manage the other causes listed above (the 6 Bs).

Monitor blood pressure every 2-5 minutes.⁷ If blood pressure does not return to baseline within 5-10 minutes with the above measures or the systolic blood pressure is above 150 mm Hg, begin medical treatment. Nifedipine 10 mg sublingual or chewed, or glyceryl trinitrate spray (1-2 sprays) are recommended as first line treatments by the Royal College of Physicians and British Society of Rehabilitation Medicine guidance.⁹ Repeat doses every 20-30 minutes as required.

In all cases, monitor the patient's blood pressure for at least two hours after an episode to ensure no rebound hypotension.⁷ Admit the patient (and inform the specialist spinal rehabilitation team) if their condition does not respond to the above treatment, if the cause has not been identified, or if an obstetric complication is suspected.⁷ We recommend referral to intensive care to monitor and manage hypertension if the blood pressure remains poorly controlled despite the measures above, or if end organ damage is present.

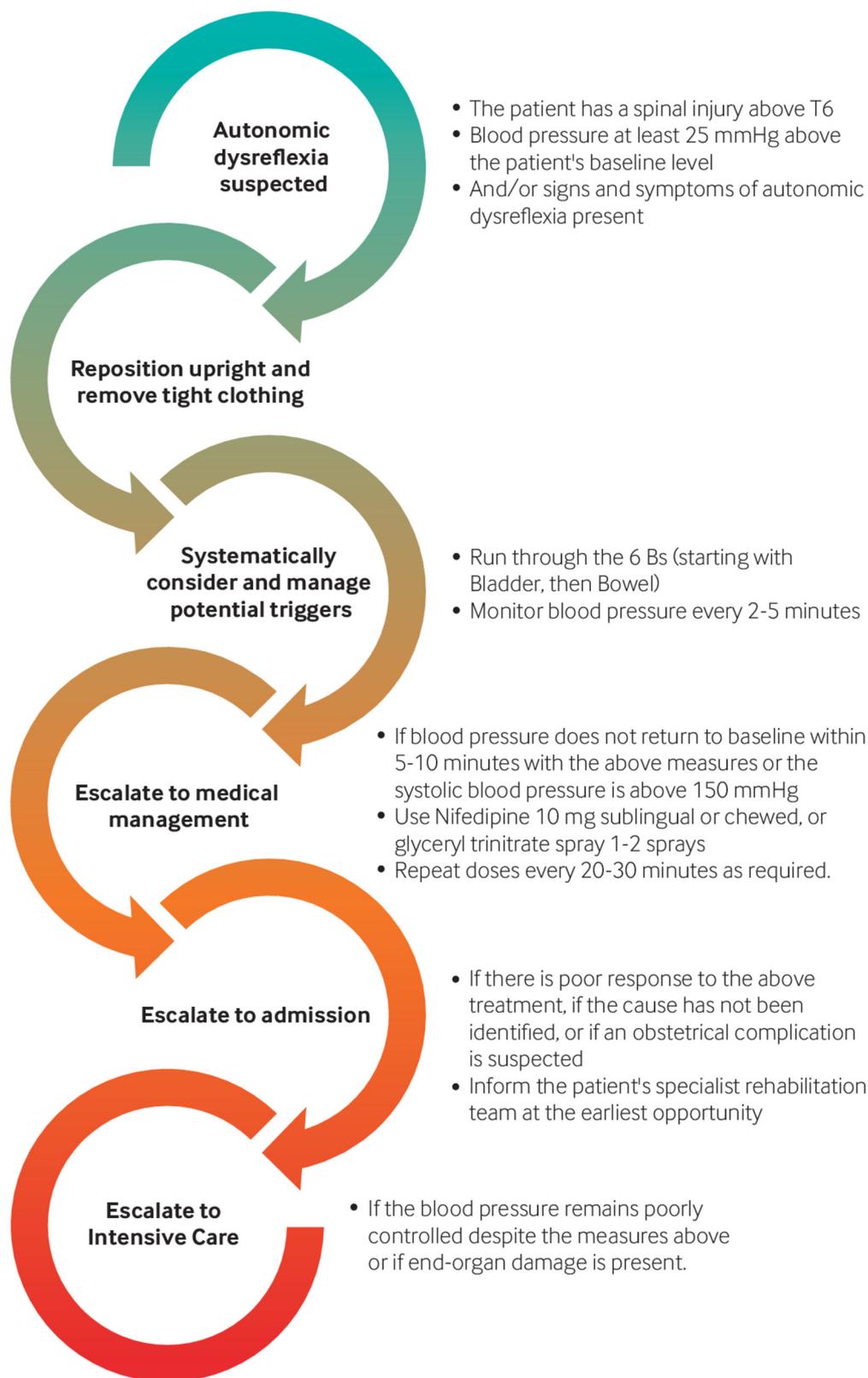


Fig 2 | Management of acute autonomic dysreflexia

Ongoing care

The long term management of patients with spinal cord injuries is overseen by a specialist spinal rehabilitation multidisciplinary team,

with the patient at the centre of care. If the patient experiences recurrent episodes of autonomic dysreflexia, arrange specialist review to investigate possible triggers and provide ongoing support. This may include blood tests, a detailed bowel and bladder

assessment (including abdominal radiography, and ultrasound of the abdomen and renal tract), and a magnetic resonance imaging scan of the spine to rule out the presence of a progressive syrinx. Further multidisciplinary input should include an occupational therapy review to provide pressure relief in a wheelchair, and education for the patient and their carers on how to avoid triggers, recognise the early signs, and treat autonomic dysreflexia.

Education into practice

- How is autonomic dysreflexia taught in your clinical environment?
- Do you consider autonomic dysreflexia in a patient with spinal cord injury presenting with headache?

How patients and carers were involved in the creation of this article

- Author Helen Cowan's husband has experienced episodes of autonomic dysreflexia. He is a member of the Spinal Injuries Association.

How this article was made

We conducted a literature search using PubMed and Medline from inception to July 2020. The key words "spinal cord injury" or "spinal injury" and "autonomic dysreflexia" were used. Institutional approval was not required for this study. The language was limited to English.

Competing interests *The BMJ* has judged that there are no disqualifying financial ties to commercial companies. The authors declare the following other interests: none.

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