Autonomic dysreflexia often goes unrecognised in patients with spinal cord injury. Health professionals must be able to recognise when patients are at risk.

Autonomic dysreflexia in spinal cord injury

In this article...
▶ An overview of autonomic dysreflexia
▶ Factors that can trigger autonomic dysreflexia
▶ Management strategies and the treatment pathway

Author Helen Cowan is nurse in older people's care, Order of St John Care Home, Oxford.


Autonomic dysreflexia is a medical emergency occurring after spinal cord injury caused by disruption of the normal autonomic responses to a stimulus below the level of spinal cord lesion. Although it can lead to stroke, convulsions, cardiac arrest and death, health professionals are largely ignorant of the condition and it is frequently misdiagnosed. This article gives an overview of autonomic dysreflexia, along with how it can be diagnosed and treated.

A young patient with tetraplegia arrives in the emergency department with a severe headache, dilated pupils, beads of sweat on their forehead, chest pain, bradycardia and a blood pressure of 280/130. What do you think is happening? Recreational drug use? A hypertensive crisis with a renal, endocrine or neurological cause? Is your immediate response to carry out an electrocardiogram and blood tests? In fact, this life-threatening emergency could be caused by something as simple as a full bladder.

The most serious complication of spinal cord injury (SCI) is autonomic dysreflexia (AD), in which a noxious stimulus below the level of injury, such as a blocked catheter or bowel distension, triggers an episode of extreme hypertension that can lead to stroke, haemorrhage, seizures and death (Wan and Krassioukov, 2014). However, the condition is under-recognised and often not understood outside of specialist SCI centres, which can lead to delayed or inappropriate treatment. In one survey, emergency department staff scored an average of two out of 29 points on a questionnaire to test their knowledge of AD (Jackson and Acland, 2011).

There is a lack of current literature about AD. The condition gained attention during the London 2012 Paralympic Games when the dangerous practice of “boosting” in which athletes self-trigger AD to enhance performance due to the increased blood pressure was described. This has now been banned by the International Paralympic Committee and all athletes are tested for the presence of AD before competing (Blauwet et al, 2013). The Royal College of Nursing (2012) provides some information on the condition and its possible occurrence during digital rectal examination and the digital removal of faeces.

This article outlines the key symptoms of AD, underlying triggers, mechanisms responsible and current management strategies. However, there is still much to learn. For example, although AD is more often seen in patients with a cervical or thoracic spinal cord lesion (typically at the level of T6 or above), and in those with complete lesions (Krassioukov et al, 2009) we do not know why its appearance is so unpredictable – a stimulus that causes an acute hypertensive attack in one patient may have no effect on another (Lindan et al, 1980).

Recognising autonomic dysreflexia

The devastating incidence of gross injuries to the spinal cord during the First and Second World Wars meant AD began to be recognised as a clinical set of symptoms.
The seminal work of Head and Riddoch (1917) revealed that newly injured patients with SCI were displaying episodes of similar symptoms; in one typical patient they noted that:

“Sweat ran together into huge beads which rolled off his face and neck, and moisture could be wrung out of a sponge, passed over his skin, almost as if it had been dipped into water… this excessive sweating was accompanied by a feeling of fullness and discomfort in the head... dilation of the pupils... the pulse tended to become slower, more forcible and somewhat irregular.”

Later work by Guttmann and Whitteridge (1947) noted a characteristic distribution of the observed sweating, with flushing and sweating above the level of SCI, and pale, cool skin and piloerection below the SCI. The systolic blood pressure was seen to 150–250mmHg and the diastolic to rise to 130–150mmHg – this was particularly alarming as the usual resting blood pressure in people with spinal cord lesions is around 20mmHg lower than in able-bodied people (Popa et al, 2010).

One hallmark feature of AD is a sudden-onset, severe, pounding or throbbing headache (Furlan, 2011); less-common manifestations can include aphasia, visual disturbance, convulsions, dyspnoea and even coma (Lindan et al, 1980).

AD can be associated with diverse electrocardiograph changes. In the early work on AD,extrasystoles and an increase in the size of the T and U wave were observed (Guttmann and Whitteridge, 1947), while a more recent case study showed recurrent ventricular fibrillation and cardiac arrest in a patient with C6 tetraplegia (Colachis and Clinchot, 1997). Anecdotal communication within the SCI community reveals too many examples of health professionals focusing entirely on a cardiac diagnosis and management of patients with AD, missing the true cause entirely.

### Triggers for autonomic dysreflexia

The most common contributing factors for AD are bladder and bowel distension; students are usually taught to remember “the 6 Bs” as a crude summary of possible triggers (Sharp et al, 2014). These are bladder, bowels, boils, bones, babies and back passage; they loosely encompass the conditions summarised in Table 1.

AD triggered by rectal stimulation during procedures such as DRF is particularly relevant as many patients with SCI are dependent on this method of elimination. Healthcare staff must be taught how to perform the procedure correctly, and topics covered should include risk assessment, monitoring for signs of AD, consent, dignity and communication (RCN, 2012). Failing to support DRF in these patients can cause faecal loading and impaction, increasing the risk of AD, as well as embarrassment and indignity.

### Underlying mechanisms

Immediately after an SCI, there is a period of “spinal shock” in which all spinal reflexes are lost completely below the level of lesion. Over a matter of weeks or months, these reflexes slowly reappear to some extent; signs of AD often emerge in parallel (Lindan et al, 1980), confirming that AD is caused by some aberration in a spinal reflex arc.

Current understanding suggests the condition is caused by a normal physiologic sympathetic discharge in response to a trigger below the level of SCI that is unopposed by descending neuronal pathways due to the complete transection of the spinal cord; this results in a massive sympathetic outflow causing extreme vasoconstriction (Krassioukov et al, 2009). This would explain the observed hypertensive crisis, ECG changes and pale, cool skin below the SCI in patients with AD.

Meanwhile, peripheral baroreceptors in the aortic arch and carotid artery detect the increase in blood pressure and send signals to the brainstem activating the parasympathetic nervous system; significant bradycardia occurs via the vagus nerve, and vasodilatation is triggered – although only above the level of spinal cord lesion, resulting in the characteristic flushing and sweating observed in the head, neck and upper body (Wan and Krassioukov, 2014).

The sympathetic response below the level of lesion far exceeds the parasympathetic reaction and so hypertension is maintained, leading to severe headache (Furlan, 2011). With lesions below the level of T6, however, AD is rarely seen (Krassioukov et al, 2009).

Understanding the mechanism of AD gives an insight into the design of the autonomic nervous system: normally the two branches are interdependent and precisely tuned, but in SCI the feedback loops between them are interrupted, manifesting paroxysmally and dramatically in episodes of AD. More research is being undertaken to understand the underlying cellular mechanisms.

### Managing autonomic dysreflexia

During an acute episode, it is imperative that medical and nursing staff consider a diagnosis of AD based on the symptoms seen in patients with SCI and act accordingly (Fig 1). Drug therapy is rarely needed – interventions such as bladder and bowel management are usually effective. There is no consensus about the drug of choice. Antihypertensives with short duration and rapid onset of action can be considered – for example, nifedipine, nitrates and sildenafil (Krassioukov et al, 2009) – although care must be taken not to induce severe hypotension. From personal experience, paramedics can be reluctant to administer these as they are not licensed for this use; with this in mind, it is important for patients to be educated and empowered to self-administer the medication. Training and support can be received from specialist spinal injury centres.

Other relevant drugs include:
- Beta-blockers (Pasquina et al, 1998);
- Botulinum toxin injections – administered into the bladder muscle to allow increased bladder capacity (Krassioukov et al, 2009);
- Intrathecal baclofen – to reduce muscle spasms and distension.

### TABLE 1: AUTONOMIC DYSREFLEXIA: TRIGGERS

<table>
<thead>
<tr>
<th>Category</th>
<th>Triggers</th>
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</thead>
<tbody>
<tr>
<td>Bladder</td>
<td>Bladder spasms or distension</td>
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<td></td>
<td>Catheter irrigation, insertion or blockage</td>
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<td></td>
<td>Cystometry</td>
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<td></td>
<td>UTI or renal stones</td>
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<td>Bowels</td>
<td>Constipation</td>
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<td>Impaction</td>
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<td></td>
<td>Rectal stimulation during manual procedures</td>
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<td></td>
<td>Enema administration</td>
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<td>Boils</td>
<td>Lesions of the skin</td>
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<td>Pressure ulcers</td>
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<td></td>
<td>Ingrowing toenail</td>
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<td></td>
<td>Burns or bites</td>
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<tr>
<td>Bones</td>
<td>Fractures</td>
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<tr>
<td>Babies</td>
<td>Pregnancy, labour, delivery</td>
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<td>Breastfeeding</td>
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<td>Sexual intercourse</td>
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<td>Scrotal compression</td>
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<td>Back passage</td>
<td>Haemorrhoids</td>
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<td></td>
<td>Anal fissures</td>
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<tr>
<td>Other</td>
<td>DVT</td>
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<tr>
<td></td>
<td>Muscle spasms</td>
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<td></td>
<td>Constrictive clothing</td>
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</tbody>
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**DVT** = deep vein thrombosis. **UTI** = urinary tract infection.

spasticity, which is a known trigger for AD (Kofler et al, 2009).
No randomised controlled trials exist in this area, leading to confusion among medical staff regarding the best course of action.
Once the AD episode is resolved it is important for the multidisciplinary team to reflect on possible causes and act to minimise recurrence. If the episode was triggered by constipation or faecal impaction, the patient’s bowel management programme should be reviewed in terms of frequency and whether drugs such as laxatives or local anaesthetic gel could aid DRE. Weight management, smoking cessation and exercise programmes, as well as advice on fluid intake and diet, can all improve general health and bowel habits, lessening the risk of AD (RCN, 2012).

The Multidisciplinary Association of Spinal Cord Injured Professionals produced guidelines to follow when designing individual bowel management programmes. These call for:
» Patient assessment;
» Intervention planning;
» Evaluation of outcomes in a cyclical process (MASCIP, 2012).

Nurses’ knowledge should also be ascertained as training sessions on DRE are often needed. The RCN (2012) guidance on managing lower bowel dysfunction can be used as a framework on which to develop relevant teaching material. Each trust should have a DRE policy and suitably trained staff available (Ness, 2013).

To manage AD long term, it is also necessary to review the patient’s bladder management programme. Ask:
» Is there a role for antibiotic prophylaxis of urinary tract infections?
» Would botulinum toxin injections help to reduce bladder spasms?
» Are routine kidney and bladder scans useful as an outpatient?

These questions are addressed with many others in national guidelines (National Institute for Health and Care Excellence, 2012) and could be considered by the multidisciplinary team.

Other simple steps to managing AD include conducting an occupational therapy review of the patient’s seating position in the wheelchair to prevent pressure ulcers, and regular podiatry appointments to keep toenails healthy.

Conclusion
AD is a serious condition occurring after SCI as a result of deranged autonomic function in response to a trigger in the paralysed part of the body. It is poorly understood but can usually be treated with relative ease once it has been correctly identified.

Educational programmes are urgently needed to raise health professionals’ awareness of AD. Patients with SCI should also be educated so they are not afraid to challenge medical opinion during diagnosis; they can be empowered by carrying a wallet card summarising the main points of AD (O’Shea, 2015).

References

For more on this topic go online...
» Ensuring a patient received appropriate bowel care following spinal cord injury
» Raising awareness of bowel dysfunction in neurology

FIG 1. AUTONOMIC DYSREFLEXIA TREATMENT PATHWAY

AD = autonomic dysreflexia. BP = blood pressure. SCI = spinal cord injury.