AUTONOMIC DYSREFLEXIA IN SCI

Autonomic dysreflexia is a medical emergency occurring after SCI, caused by disruption of the normal autonomic responses to a stimulus below the level of spinal cord lesion. Even though it can be a potentially fatal condition, healthcare professionals are largely unaware 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.

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5 KEY POINTS

1. In patients with an SCI, a stimulus such as bladder or bowel distension can lead to a dangerous increase in blood pressure. It can result in stroke, cardiac arrest and death
2. This hypertensive crisis is due to interruption of the autonomic nervous system
3. It is associated with sweating and flushing in the head and upper regions, and with bradycardia (low pulse rate)
4. It can be easily managed by removing the stimulus, such as a blocked catheter
5. There is poor awareness of this condition among nurses.

The most serious complication of 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).

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).

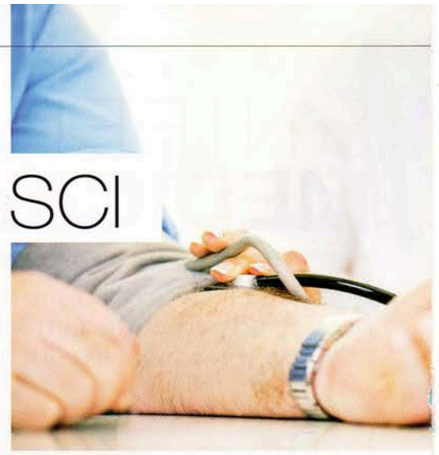
RECOGNISING AUTONOMIC DYSREFLEXIA

One hallmark feature of AD is a sudden onset, severe, pounding or throbbing headache (Furlan, 2011). Less-common manifestations can include aphasia (inability to write or speak), visual disturbance, convulsions, dyspnoea (sudden difficulty in breathing) and even coma (Lindan et al.,1980).

TRIGGERS FOR AD

The most common contributing factors for AD are bladder and bowel distension. Students are usually taught to remember 'the six 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 Fig 1.

AD triggered by rectal stimulation during procedures such as digital removal of faeces (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.

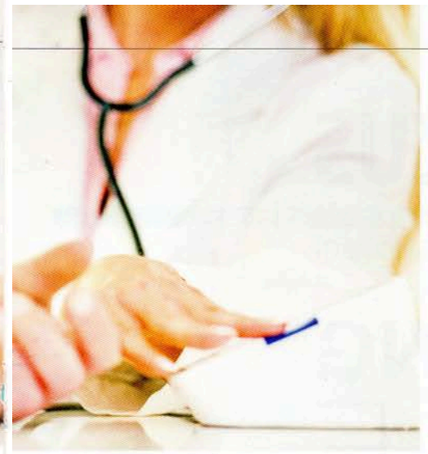
Current understanding suggests the condition is caused by a normal physiologic sympathetic discharge in response to a trigger below the level of SCI, that is unopposed by descending neuronal pathways due to the complete transection of the spinal cord. This results in a massive sympathetic outflow causing extreme vasoconstriction (narrowing of blood vessels), (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 (widening of blood vessels) 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).

MANAGING AD

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 2). 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 SCI 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 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 DRF. 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 (MASCIP) 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).**

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 an individual's multidisciplinary team.

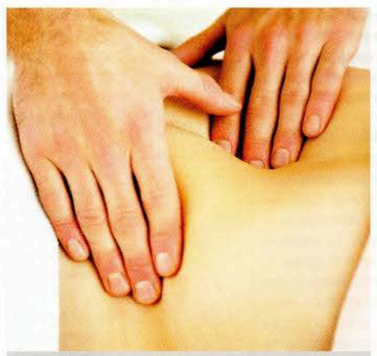
Other simple steps to managing AD include conducting an occupational therapy review of the individual's seating position in the wheelchair to prevent pressure ulcers, and regular podiatry appointments to keep toenails healthy.

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. SCI people 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.

Article references on request.

● The full version of this article appears in Nursing Times; 111: 44, 22-23. Visit **www.nursingtimes.net** for more information.



AUTONOMIC DYSREFLEXIA: TRIGGERS

FIG 1

BLADDER
- Bladder spasms or distension
- Catheter irrigation, insertion or blockage
- Cystometry
- UTI or renal stone

BOWELS
- Constipation
- Impaction
- Rectal stimulation during manual procedures
- Enema administration

BOILS
- Lesions of the skin
- Pressure ulcers
- Ingrowing toenail
- Burns or bites

BONES
- Fractures

BABIES
- Pregnancy, labour, delivery
- Breastfeeding
- Sexual intercourse
- Scrotal compression

BACK PASSAGE
- Haemorrhoids
- Anal fissures

OTHER
- DVT
- Muscle spasms
- Constrictive clothing

AUTONOMIC DYSREFLEXIA TREATMENT PATHWAY

FIG 2

Sit the patient upright and drop their feet over the side of the bed to allow orthostatic hypotension due to the loss of peripheral vasoconstriction in SCI (Krassioukov et al, 2009)

Loosen or remove any form of clothing that might be restrictive

Reassess heart rate and BP every two to five minutes to check whether they are returning to normal. Remember the resting BP in patients with SCI can be lower than that in non-disabled patients (Popa et al, 2010)

Investigate and remove possible triggers for AD, starting with possible bladder and bowel obstruction

If hypertension persists, consider drug therapy

DVT = deep vein thrombosis.
UTI = urinary tract infection
Sources: Furlan (2011), Jackson and Acland (2011), Dakhil-Jerew et al (2008), Lindan et al (1980)