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.

The most serious complication of SCI in autonomic dysreflexia (AD) is a noxious stimulus below the level of injury, such as a blocked catheter or bowel distension, that triggers an episode of extreme hypertension that can lead to stroke, hemorrhage, seizures and death (Wan and Krasnowsky, 2014). However, the condition is under-recognized 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 those with complete lesions (Krasnowsky 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 (Linden et al, 1985).

RECOGNISING AUTONOMIC DYSREFLEXIA
One major feature of AD is a sudden onset, severe, pounding or throbbing headache (Furlin, 2011). Other common manifestations can include apnea (inability to write or speak), visual disturbances, convulsions, dyspnea (sudden difficulty in breathing) and even coma (Linden et al, 1985).

TRIGGERS FOR AD
The most common contributing factors for AD are bladder and bowel distension. Students are usually taught to remember the “six B’s” as a summary of possible triggers (Sharp et al, 2014). These are bladder, bowel, bones, back pain and back passage; they loosely encompass the conditions summarized in Fig. 1.

AD triggered by rectal stimulation during procedures such as digital removal of feces (DFR) 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 (ICSN, 2013). Failing to support DFR in these patients can cause fecal 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. Once a minor stimulus, reflexes gradually reappear to some extent. Signs of AD often remain nappier (Linden et al, 1985), confirming that AD is caused by some obstruction 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 the SCI, that is unopposed by decerebration nervous pathways due to the complete transection of the spinal cord. This results in a massive sympathetic outflow causing extreme vasconstriction (narrowing of blood vessels), (Krasnowsky et al, 2009). This would explain the observed hypertensive crisis, ECG change and pale, cool skin below the SCI in patients with AD.

MEANWHILE, paraspinal baroreceptors in the aortic arch and carotid artery detect the increase in blood pressure and send signals to the brainstem activating the sympathetic nervous system. Significant baroreceptors occur 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 (Allen and Krasnowsky, 2017). The increase in bloodflow can raise the level of lesion far exceeds the parasympathetic reaction and so hypertension is maintained, leading to severe headache (Furlin, 2011). With lesions below the level of T5, however, AD is rarely seen (Krasnowsky 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. Antispasmodics with short duration and rapid onset of action can be considered. For example, midazolam, nitroprusside and sodium nitrite (Krasnowsky et al, 2009) — although care must be taken not to induce severe hypotension. From personal experience, paracetamol can be administered to reduce anxiety, 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 (Papazyan et al, 1998)
- butylamine toxin injections – administered into the bladder muscle to allow increased bladder capacity (Krasnowsky et al, 2009)
- intrathecal baclofen – to reduce muscle spasticity, which is known on trigger for AD (Keller et al, 2005).

No randomized 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 successful cases and act to minimise recurrences. If the episode was triggered by constipation or fecal impaction, the patient’s bowel management programme should be reviewed in terms of frequency and whether drugs such as laxatives or local anesthetic gel could aid DFR. 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 (ICSN, 2012).

The Multidisciplinary Association of Spinal Cord Injured Professionals (MASCOP) produced guidelines to follow when designing individual bowel management programmes. These call for:
- patient assessment
- intervention planning
- evaluation of outcomes in a cyclical process (MASCOP, 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 butylamine 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 should be considered by an individual’s multidisciplinary team.

Other amelie 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 damaged 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 professional 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, 22-25. Visit www.nursingtimes.net for more information.