

Statement on Autonomic Dysreflexia

Issued by	British Association of Spinal Cord Injury Specialists (BASCIS) Multidisciplinary Association of Spinal Cord Injury Professionals (MASCIP) Spinal Injuries Association (SIA)
Date	June 2017
Statement	<p>Autonomic Dysreflexia in Spinal Cord Injured Patients</p> <p>There are a number of spinal reflexes that affect the sympathetic outflow and blood pressure, some of which are useful to people with high spinal cord injury in managing to sit/stay upright. It is the altered, or abnormal reflexes that become prominent in the chronic situation and it is probably because there are a number of these, influenced by a number of factors, by the passage of time and “neuroplasticity” and subject to systemic factors, such as atherosclerosis, that the occurrence and dangers of a condition known as Autonomic Dysreflexia (AD) can be so variable and unpredictable. A ‘track record’ is no indication or guarantee of what is likely to happen.</p> <p>AD is an unpredictable, potentially life-threatening condition whereby there is a sudden, rapid and uncontrolled increase in blood pressure (systolic blood pressure can rise >300 mm Hg). It signifies the paralysed body’s response to a problem that the individual, because of their paralysis, cannot perceive or identify directly, and is triggered by acute pain or some other noxious or non-noxious stimulus experienced below the level of spinal cord injury. Provocative factors are diverse and disparate, but include over-distension of the bladder, urinary tract infection, scrotal compression, pressure ulcer and bowel-related factors.</p> <p>AD occurs in people with spinal cord injury or dysfunction and affects those with neurological lesions (both complete and incomplete) at or above thoracic level T6. The resultant acute relative rise in blood pressure can lead to life-threatening outcomes including cerebral haemorrhage, pulmonary oedema, epileptic seizure, myocardial infarction and even death¹.</p> <p>AD should always be regarded as a MEDICAL EMERGENCY and needs to be addressed immediately^{2,3,4,5}.</p> <p>Mechanism:</p> <p>Body functions involve central and reflex control systems within the brain, spinal cord and nerve structures. Connections to the different parts of the body are organised segmentally from the spinal cord – the sympathetic nervous system connections come from T1 to L2 segments; those to the major blood vessels (in the trunk/abdomen and legs) are from T5 to T12/L2 spinal cord levels.</p> <p>Blood pressure is mediated by modulating (increasing or decreasing) the sympathetic input to the (smooth muscles in the walls of) blood vessels causing constriction (narrowing) or dilation, thereby rapidly adjusting the blood pressure by effectively changing the resistance to blood flow.</p> <p>When the neurological level of injury is T6 or higher, altered reflexes in the spinal cord, below the level of the damage, mean that sympathetic activation, including rises in blood pressure, can result from a variety of inputs. Such sympathetic activation, can be erratic and unpredictable in both occurrence and severity; subject to other things affecting the spinal cord and nervous system and also systemic factors, such as infection. Loss of central control (the baroreceptor reflex) compromises, the body’s ability to compensate for changes in posture or bring down high blood pressure. Baseline blood pressure may also be affected. The sensing part of these baroreceptor connections remains intact – information comes from “pressure sensors” in the circulation, via nerves (IX, X cranial nerves) to the control centre in the brainstem. That control centre can only activate the parasympathetic (vagus) nerve connection to the heart, and none of</p>

the sympathetic, in someone with tetraplegia, or only the upper part of the sympathetic, in someone with a high paraplegia. There is no control centre connection, through the spinal cord, to the lower sympathetic nerve connections or means of modulating the blood pressure (by vasoconstriction or dilation of the abdominal and leg blood vessels).

These responses to the changes brought about by sympathetic over-activity result in the symptoms and signs (e.g. flushing and sweating above the lesion, severe pounding headache) of AD which can be an invaluable warning mechanism for patients to take action and/or seek assistance from care givers. Indicative symptoms vary, however, between patients. Raised blood pressure is the crucial medical concern, severe headache often being the predominant distressing symptom.

Assessment of Risk and Implications for Care:

Autonomic dysreflexia represents a serious, significant and lifelong risk to the health and well-being of susceptible individuals, and occurs in up to 90% of those with a cervical or high thoracic SCI⁶. It can occur at any time following SCI⁷. Little is known about the connection between the severity and level of SCI and the severity of autonomic dysfunction⁷.

The inappropriate activation of the sympathetic nervous system associated with AD occurs several times a day and may even occur asymptotically. Moreover, potential triggers for AD occur when caring for a susceptible SCI person (e.g. during digital stimulation of the rectum for bowel evacuation). This means that the phenomenon of AD is part of a continuum from no symptoms (asymptomatic AD) to high-grade paroxysmal hypertension or 'full-blown' AD⁷. The outcome of management depends on the early recognition of the condition and lowering the blood pressure by removing the stimulus, as patients with low grade or chronic AD can become high grade very quickly^{4,5}.

The risk of a susceptible SCI individual having an episode of AD can never be permanently reduced or removed. At best when the individual's healthcare needs are well-managed (for instance, in a specialist Spinal Cord Injury Centre or with high quality care in the community) the incidence of episodes can be reduced. Even with the best healthcare management regimen in place, AD can, and does, nevertheless occur. Therefore, it is never clinically safe to use the absence of historical episodes of AD as a predictor of future risk and associated healthcare needs. Indeed, when an attack occurs where there is no history of previous episodes, the risk is heightened because the person will lack the experience to identify or deal with the symptoms or cause of the attack.

In an assessment of care needs, therefore, a person's needs should **not** be 'downplayed' on the basis that they have not manifested recent episodes of AD. Absence of prior episodes of full-blown AD should be interpreted as an indication that the person's healthcare needs are being well-managed.

Management considerations:

Management focuses on 'remove the cause' and 'reduce the blood pressure' (sit the person up to induce postural hypotension, and consider use of a short acting hypotensive (blood pressure lowering) medication. It is advised that susceptible individuals carry a prescribed vasodilator drug labelled 'for use in unrelieved autonomic dysreflexia' at ALL TIMES to alleviate hypertensive crisis. Practice varies with some favouring GTN sub-lingual spray and others utilising nifedipine formulations. Most need assistance by a trained care giver to intervene to identify causation and to administer medication: tetraplegics lack requisite manual dexterity, and in both those with paraplegia and tetraplegia the normal level of cognition may be impaired⁸ to an extent that they cannot self-medicate.

Early recognition of signs and symptoms of AD is a major key to immediate and appropriate treatment of this urgent condition^{7,9}. Management must concentrate on identifying causation and removal of the stimulus (lower basal blood pressure means that measured 'high blood pressure' is less relevant than relative high blood pressure – a BP of 20-40 mm Hg above baseline may be a sign of AD. For an individual measurement of BP is not an immediate priority and confirmation of AD or initial investigation should not be deferred until the systolic pressure reaches a specific value). This requires care givers to investigate urgently possible causations (e.g. blocked/twisted catheter, overloaded rectum, trauma to paralysed areas of the body such as in-grown toenail or skin damage from sitting on a foreign object) and to act accordingly.

If resolved, symptoms of AD will generally subside. However, a 'status dysreflexia' (a state of prolonged, heightened susceptibility to AD after an initial attack) may endure. It is, therefore, important to avoid emergence of AD by ensuring a person's needs continue to be well-managed, especially in terms of continence and skin care needs. The maxim of 'prevention is better than cure' applies.

In the event that causation cannot be identified and alleviated speedily, and hypertension cannot be

controlled, emergency medical assistance should be summoned. Lack of awareness and understanding of AD amongst healthcare workers outside of spinal cord injury centres^{8,10}, including the emergency services², however, means that it is essential that care givers to susceptible individuals have the skills to act as 'first responders' and, if necessary, convey that knowledge when healthcare professionals attend. Repeated episodes of AD, increase in frequency or unclear causes warrant further investigation; however, regular follow-up often identifies incipient trigger factors before patients are aware, and patients should attend Spinal Cord Injury Centre outpatient appointments at appropriate frequencies as part of a strategy for successfully managing the condition and reducing incidence of AD.

Skill and Knowledge Requirements:

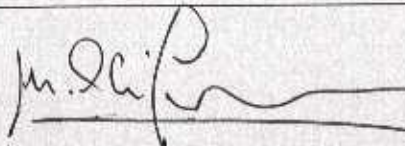

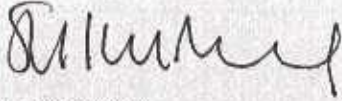
Avoidance and management of AD requires care givers to have knowledge, training and healthcare skills which generally exceed those which can lawfully be provided by social care provision. Care givers require knowledge not only of the condition, but how to recognise incipient AD in that specific individual, and how to resolve matters. Both the severity and constellation of presenting signs vary, but may include one or more of following: pounding headache, blurred vision, anxiety, flushing and diaphoresis above lesion level, piloerection, nasal congestion, vaso-constriction below lesion level, and impaired cognition. Seemingly unremarkable presenting symptoms may, however, disguise serious imminent deterioration⁵. Specific skills including, but not limited to, catheter management and bowel care (including digital rectal checking and removal of faeces) are important.

Susceptible individuals are advised to carry an 'Emergency Medical' card, such as that available from the Spinal Injuries Association: www.spinal.co.uk detailing their treatment for the condition. A Fact-sheet on Autonomic Dysreflexia written by the National Spinal Cord Injuries Centre is available at http://www.spinal.co.uk/userfiles/CHC/NSIC_Autonomic_Dysreflexia_Fact_Sheet_July_2013.pdf to educate both patients and others.

References

1. D. Wan & A.V. Krassioukov. Life-threatening outcomes associated with autonomic dysreflexia: a clinical review. *J Spinal Cord Medicine* 2014; 37(1):2-10.
2. J. Bycroft et al. Autonomic dysreflexia: a medical emergency. *Postgrad Med J* 2005; 81:232-235.
3. J.A. Walker. Autonomic dysreflexia. *Nursing Times*. May 1 2002
4. R. Showkathali & T. Antonios. Autonomic dysreflexia, a medical emergency. *J R Soc Med.* 2007; 100(8):382-383.
5. Vaidyanathan S et al. Autonomic dysreflexia in a tetraplegic patient due to a blocked urethral catheter: spinal cord injury patients with lesions above T6 require prompt treatment of an obstructed urinary catheter to prevent life-threatening complications of autonomic dysreflexia. *Int. J Emergency Medicine* 2012, 5:6
6. A. Krassioukov. Autonomic dysreflexia: current evidence related to unstable arterial blood control among athletes with spinal cord injury. *Clin J Sports Med* 2012; 2(1):39-45.
7. A. Krassioukov et al. Assessment of autonomic dysfunction following spinal cord injury: Rationale for additions to International Standards for Neurological Assessment. *J Rehabilitation Research & Development* 2007; 44(1):103-112.
8. J. Khastgir et al. Recognition and effective management of autonomic dysreflexia in spinal cord injuries. *Expert Opin Pharmacother.* 2007; 8(7):945-56.
9. H. Gunduz & D.F. Binak. Autonomic dysreflexia: an important cardiovascular complication in spinal cord injury patients. *Cardiology J.* 2012; 19(2):215-219.
10. J.C.Furlan. Headache attributed to autonomic dysreflexia: an under-recognised clinical entity. *Neurology* 2011 23; 77(8):792-798.

Signatures

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Useful links

http://www.bascis.org.uk	http://www.masclip.co.uk	http://www.spinal.co.uk
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