AUTONOMIC DYSREFLEXIA

Autonomic dysreflexia is the term used to describe the autonomic response to painful (noxious) stimuli perceived below the level of lesion. This is a potential complication for all patients with spinal cord lesions above the level of T6. The most common stimulus is a blocked catheter. This problem manifests itself as acute hypertension. Systolic blood pressure can easily exceed 200mmHg. Unresolved it can cause significant complications including stoke, seizures, severe myocardial ischemia and death.

This reflex response is usually suppressed during the period of spinal shock however should still be considered.

The main presenting features of Autonomic Dysreflexia are:

Severe (pounding) headache

Profound vaso-dilation (flushing) above the level of cord lesion and vaso-constr visible even in different skin types

Profuse sweating above the level of cord lesion

In the presence of visible primary symptoms, it is recommended that the initial investigation and treatment of cause should not be delayed through a poorly

Mechanism

Body functions involve central and reflex control systems within the brain, spinal cord and nerve structures. The autonomic nervous system is a regulatory branch of the central nervous system that helps people adapt to changes in their environment acting though its two branches the parasympathetic nervous system and the

regulation by vasomotor centers in the brainstem. Release of substances, such as noradrenaline, cause severe vasoconstriction with skin pallor, pilo-erection and a sudden rise in blood pressure (BP), which is usually accompanied by a pounding headache.

increase in blood pressure they increase the parasympathetic signaling as a compensatory mechanism resulting in bradycardia (via the vagus nerve) and flushing (focal peripheral vasodilation); probably also responsible for headache and profuse sweating above the level of injury (via sympathetic inhibitory outflow from vasomotor centers). However, both these mechanisms are insufficient to satisfactorily control paroxysmal hypertension due to massive sympathetically mediated vasoconstriction of the splanchnic bed.

may be appropriate to remove the blocked catheter completely to allow for possibility of urethral drainage until re-catheterisation is possible.

3. If appropriate, sit the patient up, or tilt the bed head-up, to induce some element of postural hypotension. Do this gradually, as it may actually worsen symptoms and hinder investigation where distended bladder or bowel is the cause.

4. If symptoms remain unresolved after removal of noxious stimulus or if noxious stimulus cannot be identified then administer prescribed proprietary chemical vaso-dilator such as sublingual glyceryl trinitrate (GTN) or sublingual captopril (25mg).

   NB: Nifedipine capsules, which were previously recommended for use in treating Autonomic Dysreflexia have been withdrawn from routine use in UK due to being linked with post-incident hypotensive crises.

5.

References

British Association of Spinal Cord Injury Specialists (BASCIS), Multidisciplinary Association of Spinal Cord Injury Professionals (MASCIP), Spinal Injuries Association (SIA) (2014) *Statement on Autonomic Dysreflexia.* Available at: https://www.spinal.co.uk/wp-content/uploads/2018/06/Statement-on-Autonomic-Dysreflexia-2017.pdf (Accessed July 2020)

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