Autonomic Dysreflexia

Also known as Hyperreflexia

It is: an over- activity of the Autonomic Nervous System causing an abrupt onset of excessively high blood pressure. This is characterised by a sudden, severe headache.

Autonomic Dysreflexia (A.D) can develop suddenly, is potentially life threatening and is a medical emergency. If not treated promptly and correctly it may lead to cerebral haemorrhage, acute myocardial infarction and even death.

Persons at risk: Those with a spinal cord injury at T6 and above.

Autonomic Nervous System
Provides automatic control of vital bodily functions.
Consists of:
Sympathetic nerves (localised between the 1st thoracic and 1st lumbar segments of the spinal cord – thoracolumbar outflow). These nerves generally excite the body e.g. increase the heart rate and blood pressure.

Parasympathetic nerves (localised near cranial nerve roots in the brain stem and between the 2nd and 4th sacral segments of the cord – cranial and sacral outflow). These nerves generally calm the body down e.g. by decreasing the heart rate and blood pressure.

A.D. is caused by a variety of stimuli, creating an exaggerated response of the sympathetic nervous system due to lack of control from higher centres. The condition is precipitated by specific noxious stimuli from localised areas below the level of injury, mostly from the abdominopelvic region. The most frequent source of stimuli is an over-distended bladder.

What happens:

1. Local stimuli enter the cord and ascend to the level of cord injury, where communication to the brain is interrupted. An enormous sympathetic response is activated.

2. The result: blood vessel spasticity in the abdominal and pelvic organs and vasculature of the skin. This spasticity causes vasoconstriction to an area so rich in blood supply that the body's blood pressure rises quickly.

3. The increased blood pressure is sensed by nerve endings located in the aorta and carotid sinus.

4. The parasympathetic nervous system is activated and to compensate lowers the blood pressure by slowing the heart rate and attempting to dilate all blood vessels. Some impulses may be effective, as normal parasympathetic outflow is preserved, but other impulses are blocked by the cord injury, thus preventing communication with the lower thoracic and sacral autonomic outflow.

5. As messages sent from the parasympathetic nervous system cannot travel past the area where the cord is damaged, the blood vessels in the internal organs and in other parts of the body do not widen to decrease the blood pressure.

6. The result: Overactive sympathetic vasodilation above the level of injury and the blood pressure remains elevated until the noxious stimuli is removed (Zejdlik, 1992).

<u>Causes of A.D:</u>

Bladder – Over distension/irritation
Urinary tract infection
Urinary retention
Blocked catheter/kinked tubing
Overfilled collection bag
Non-compliance with intermittent catheterisation programme
Calculi

Bowel - Over distension/irritation
Rectal stimulation
Constipation/impaction
Haemorrhoids or anal fissures

Skin - Any direct irritant below the level of injury (e.g. prolonged

pressure or object in shoe or chair, bruise etc.
Pressure sores
Ingrown toenails
Burns (e.g. scolds, sunburn)
Tight or restrictive clothing or pressure to skin from sitting on creased clothing
Sitting on testicles

Sexual Activity – Over stimulation during sexual activity (stimuli to the pelvic region which would ordinarily be painful if sensation were present)
Pregnancy/labour

Other - Heterotopic ossification
Acute abdominal conditions (gastric ulcer, colitis, peritonitis)
Skeletal fractures
Instrumentation (e.g. to the bladder)

Signs and Symptoms of A.D.

Headache (due to elevated blood pressure) of a pounding, severe nature
Elevated blood pressure (compare with baseline observations, a BP of 140/90 may be considered high for some)
Profuse sweating
Flushed face
Blotchiness of skin above the level of spinal cord injury
Goose pimples
Nasal stuffiness
Apprehension/Agitation
Bradycardia
Cold, clammy skin below the level of injury

To care for people with A.D:
Recognise immediately the onset of this condition and initiate measures to lower the blood pressure.
Remove or control stimuli and prevent dangerous or fatal complications.
Prevent recurring episodes.

Treatment:

If up in chair return the patient to bed
Elevate the head of the bed or hold the patient in the sitting position to lower the blood pressure – postural hypotension
Monitor the blood pressure closely
Remove the causative stimuli

1. Check the bladder for over-distension
2. If the patient is on an intermittent catheterisation programme, catheterise immediately
3. If the patient already has a catheter, check it is draining. Look for kinks in the tubing, plugged connections, or a full leg bag.
4. Change the catheter <u>without hesitation</u> if no obvious obstruction. Do not attempt washouts. Use anaesthetic gel for lubrication

If A.D. persists, using lubrication, gently check the lower bowel for stool and gradually try and remove it. If symptoms persist <u>stop procedure</u> and administer anaesthetic gel. Wait 10 minutes and then resume cautiously if symptoms have subsided.

If the bladder or bowel do not seem to be the course, check for:
A pressure sore
Ingrown toenail
Fractured bone
Constrictive clothing/shoes
Anything else that may be causing painful stimuli

<u>Emergency Medications</u>

Give sublingual nifedipine 10 mgs if there is a delay identifying the cause and the patients' blood pressure is not decreasing. Repeat after 15 minutes if necessary.

Other Medications which may be used are, for e.g.
Nitro-glycerine and Captopril.
Others may be recommended, depending on hospital policies.
A Doctor must prescribe all medications.

If the blood pressure still does not return to normal, intravenous ganglionic blocking agents will be required.
(Labetalol 5mg/min. up to 20mg)

Monitor the patients' B/P and Pulse regularly for the next 24 hours. The autonomic nervous system tends to remain unstable for a while when the body's reaction has been severe. If the B/P is labile, treatment for a few days with Nifedipine Retard may be considered.

Provide psychological support

Consider the causes of A.D. for each patient and initiate management to prevent further episodes if possible. Especially provide the optimal bladder and bowel care:

During intermittent catheterisation programme ensure a desirable balance between intake and output. Urinary output for each catheterisation should not exceed 500mls.

When the patient has an indwelling catheter, promote unobstructed, gravity-assisted drainage.

Take measures to prevent urinary infection, reflux and calculi formation (e.g. 2.5 to 3 litre input, vitamin C)

Observe the post-operative patient closely

Provide regular and reliable bowel programme.

Provide patient and family education on A.D. and indeed, all aspects of spinal cord injury and the consequential care that is required.
A small emergency card with a simple explanation about A.D. should be provided and emergency medications prescribed for when discharged. It is also safer to teach a member of the family how to catheterise, if possible.

Zejdlik. Cynthia
Management of Spinal Cord Injury. 1992 Jones and Bartlett Publishers, Boston

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