# Autonomic Dysreflexia

A Life Threatening Emergency

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Contents



**Learning Objectives**

At the completion of this article the reader will be able to:

- Recognize the signs and symptoms of autonomic dysreflexia (AD)
- Recognize AD as a medical emergency
- Know successful intervention strategies to relieve AD
- Understand the pathophysiology underlying AD

Autonomic dysreflexia is a potentially life threatening, uninhibited and exaggerated reflex response by the autonomic nervous system to a noxious stimulus. The condition occurs most commonly in individuals with spinal cord injuries above T6 (Keely, Kewalramani). Advances in on-site and hospital-based emergency care of individuals following spinal cord injury have resulted in markedly reduced mortality with survival rates of 85-90% (Karlsson). As many as 85% of individuals with SCI above T6 experience autonomic dysreflexia (Trop and Bennett). Given these statistics, it is increasingly likely that therapists (physical, occupational and speech, as well as assistants aids and nurses) will treat these individuals and should, therefore, be aware of the signs and symptoms, causes, and management recommendations for this serious condition (Travers).

The first episode of autonomic dysreflexia (AD), also known as autonomic hyperreflexia, usually occurs after the phase of spinal shock and the return of reflex responsiveness (usually hours to weeks following the injury, Comarr and Eltorai). The potential to experience this exaggerated response to noxious stimuli persists throughout the individual's lifetime. AD is caused by a noxious stimulus below the level of the lesion.

**Signs and Symptoms**

An individual experiencing an episode of AD may exhibit one or more of the following symptoms. The more common signs and symptoms are:

- A sudden and significant increase in blood pressure (normal BP for an individual with a cord injury at or above T6 is 90-110 mm Hg systolic and 50-60 mm Hg diastolic). The Consortium for Spinal Cord proposed an increase of 20 to 40 mm Hg above baseline is a sign of AD.
- Pounding headache of sudden onset
- Profuse sweating and skin flushing **above** the lesion level (notably the face, neck, and shoulders)
- Goose bumps (piloerection) **above** the lesion level
- Blurred vision or spots in the visual field
- Nasal congestion
- Cardiac dysrhythmias, premature ventricular contractions, or atrial-ventricular conduction abnormalities

These symptoms may occur individually or in combination. The most common and potentially dangerous sign of AD is hypertension. It is this severely elevated blood pressure that constitutes the medical emergency. Blood pressures may rise as high as 300/220 mm Hg during as episode of AD (Trop and Bennett). Such extreme pressures may result in subarachnoid, intracerebral, renal, or retinal hemorrhage, seizure, myocardial infarction, and possibly death.

**Causes of AD**

AD is caused by a noxious stimulus below the level of the lesion. To effectively resolve the episode, the specific cause must be identified and promptly removed. The most common cause (accounting for approximately 75% of cases) is noxious stimulation of the lower urinary tract (Keely, Kewalramani). The stimulus may be a full bladder, catheterization, urinary tract infection, obstruction or kinked indwelling catheter. Other causes include:

- Bowel distention or impaction (vigorous enema or rectal stimulation)
- Scrotal compression
- Bladder or kidney stones
- Pregnancy, especially labor and delivery
- Deep vein thrombus
- Pressure ulcers (including ingrown toenails)
- Contact with hard or sharp objects
- Constrictive clothing (shoes or appliances)
- Temperature fluctuations
- Insect bites

**Management Recommendations**

Specific, 3-step guidelines have been established by the Consortium for Spinal Cord Medicine to direct immediate intervention for an individual presenting with signs and symptoms of an acute onset of AD. Initially the therapist needs to recognize the constellation of signs and symptoms as AD and be aware that the condition constitutes a medical emergency requiring prompt action. Intervention should include monitoring blood pressure, identifying and eliminating the cause, and notifying all appropriate team members.

Step 1. Determination of Blood Pressure
The first step taken should be the determination of blood pressure and pulse rate. If the blood pressure is elevated and the individual is supine, bring the person to a sitting position. Doing so attempts to employ the effects of orthostatic hypotension to counteract the hypertension. Once in the sitting position, loosen any constrictive clothing or devices (abdominal binders and support stockings). Loosing of orthoses or other spinal stabilizing apparatus may be contraindicated. Blood pressure should be monitored every 2 to 5 minutes until stable (consistent pressure readings for at least three consecutive readings). The pulse should be monitored along with blood pressure because the pulse rate may reflexively slow. As a precaution, blood pressure and pulse rate should be monitored frequently for at least 2 hours following resolution of the episode.

Step 2. Identification of the Cause
The second step taken should be determination of the cause. Because bladder distention is the most common cause of AD, the excretory status of the bladder must be evaluated. The individual should also be examined carefully for irritation stemming from the clothing, support surface, environment, or treatment response.

If the individual does not have an indwelling catheter, the appropriate healthcare personnel) should perform urinary catheterization. If an indwelling catheter is in place, the system should be carefully examined for occlusion. If the catheter is clamped, release the clamp. Tubing and receptacles should be checked for kinks, twists, folds, and internal blockage. Occlusion of the drain line and adverse response to intervention are the most common causes of AD during therapy treatment sessions. If occlusion is found and removed, the blood pressure should be monitored because a sudden release of urine may result in hypotension. If hypotension does occur, return the individual to the supine position and elevate the legs. If the blood pressure remains elevated in spite of the resolution of bladder distension, the clinician should rule out fecal impaction (the second leading cause of AD). If the cause cannot be

identified and symptoms do not subside, prompt assistance should be sought from the appropriate medical staff. Pharmacologic intervention with antihypertensives may be necessary (Naftchi and Richardson).

Step 3. Notification of Team Members
The episode should be documented in the patient record with the following patient-specific information included:

- Presenting signs and symptoms
- Course of the signs and symptoms
- Intervention

To evaluate the effectiveness of the clinical management of the acute episode, outcome assessment should be performed using the criteria recommended by the Consortium for Spinal Cord Medicine. The four criteria are:

- The cause of the episode was effectively identified.
- The blood pressure was restored to acceptable pre-episode levels (usually 90-110 mm Hg systolic in the sitting position).
- The pulse was restores to acceptable pre-episode levels.
- The episode was resolved with no adverse signs or symptoms (cardiac or cerebral).

When the patient has stabilized following an episode of AD, the cause of the episode should be discussed with the individual, family, and caregivers. Patient and family education should include the signs and symptoms, appropriate interventions, and recognition of the medical urgency of the situation. Discharge materials might include a written emergency plan for AD (including signs and symptoms, suggestions for immediate intervention and possibly a wallet-sized card with the same information, Appendix A).

The best way to prevent AD includes bowel and bladder programs to prevent distension as well as care with catheters, tubes, and collection containers (Morris and Marshall). The use of local anesthetic in lubricating jellies can help prevent noxious stimulation during catheter placement. Routine skin and foot care can help prevent wounds and environmental controls can help regulate body temperature.

Figure 1 details a six-step process by which the noxious stimulus results in an uninhibited sympathetic response.

The first signs and symptoms of autonomic dysreflexia are usually sweating or a headache. Such complaints should raise a “red flag” for the clinician and draw an immediate response. First, check blood pressure to confirm the presence of AD. Then, determine and relieve the noxious stimulus. The most common cause is a distended bladder or bowel. Ask the last time the client was catheterized or had a bowel movement. If the client has a Foley catheter, check to see if the line has become blocked. If the bladder and bowel are cleared, loosen tight clothing below the level of the lesion with attention to potentially noxious seems or creases. If the client has been in one position for prolonged period, change position. Examine the skin for lesions, leg or foot swelling (deep vein thrombosis), evil redness over pressure points (dicubiti), foot disorders (ingrown toenail) or evidence of other trauma. Corrective action removing the stimulus will result in rapid relief of symptoms. If the condition persists, pharmacologic intervention to bring down the blood pressure is essential. A physician or nurse may administer sublingual nifedipine (Procardia 10 mg) to promptly lower blood pressure (Naftchi and Richardson ). The head of the bed can also be raised to enlist orthostatic hypotension to counteract the hypertension.

Because of the extended periods of time therapists spend with clients who have suffered spinal cord injuries, it is likely that sooner or later they will encounter an acute episode of AD. Knowledge of its signs and symptoms and appropriate responses can help prevent dire consequences.

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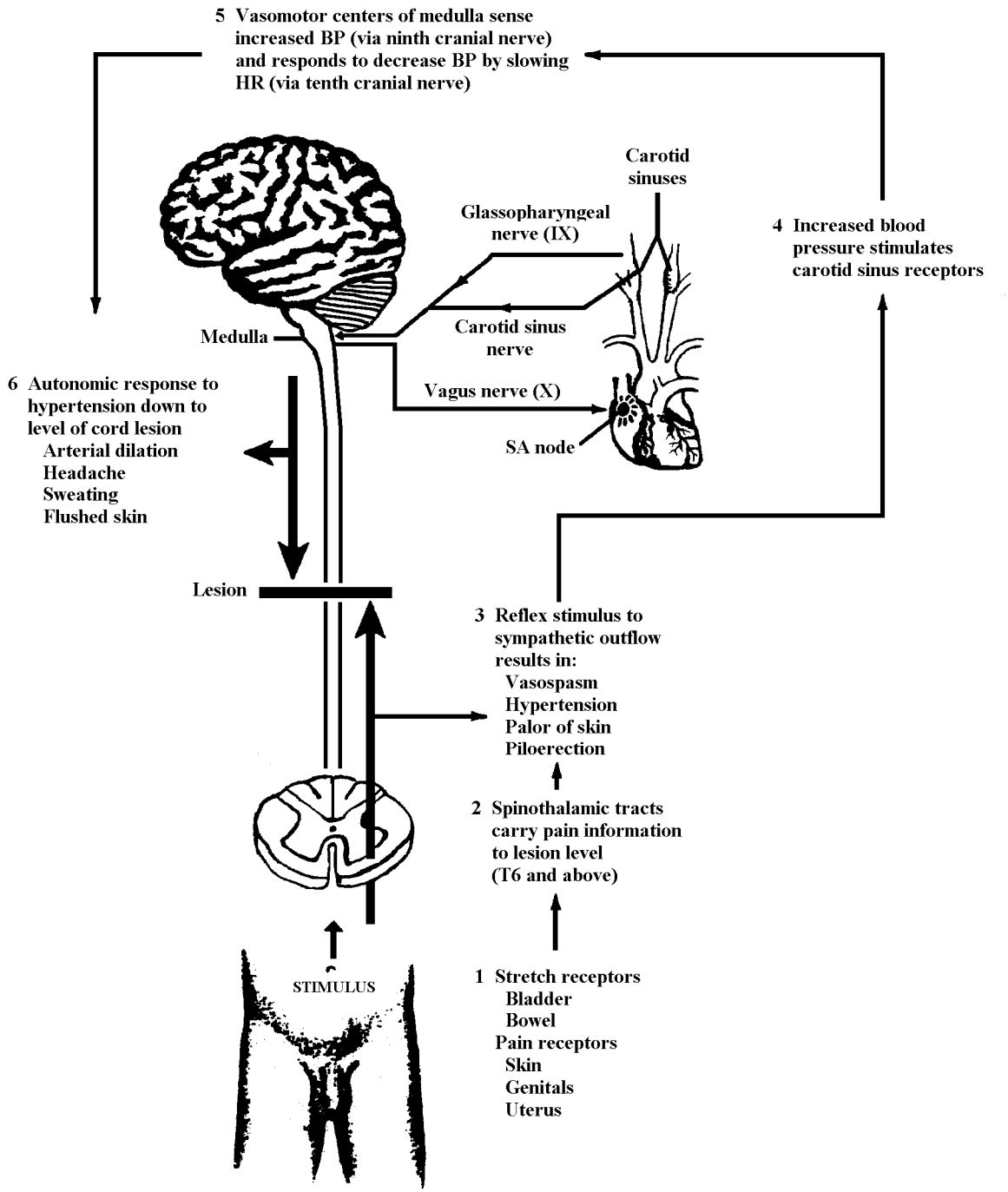


Figure 1 depicts the six-step process by which a noxious stimulus causes autonomic dysreflexia. First, the noxious stimulus is sensed by nociceptors (stretch receptors in the bowel and bladder) or pain receptors in the lower quarter. Second, these receptors synapse with fibers that ascend in the dorsolateral system (spinothalamic tract) to the level of the lesion. Third, the interrupted dorsolateral system reflexively "spills over" to the sympathetic outflow in the thoracic region causing generalized vasoconstriction, hypertension, palor, and piloerection below the injury level. Fourth, the increased blood pressure stimulates the carotid sinus receptors. Fifth, the glassopharyngeal nerve (CN IX) senses the increased blood pressure and informs the vasomotor center in the medulla that stimulates the vagus nerve (CN X) to decrease pressure by slowing heart rate (bradycardia). Sixth, the hypothalamic response to the hypertension produces arterial dilation, flushed skin, headache and sweating above the injury level. Below the injury level, the hypertension proceeds unchecked because the injury prevents the supraspinal response from checking the sympathetic response below the level of injury.

## Review Questions

1. The most common cause of autonomic dysreflexia is:
   A. pregnancy especially labor and delivery.
   B. an ingrown toenail.
   C. rapid fluctuations in environmental temperature.
   D. noxious stimulation of the lower urinary tract.

2. Which effect of autonomic dysreflexia constitutes the medical emergency?
   A. pounding headache
   B. hypertension
   C. bradycardia
   D. sweating above the level of injury

3. How do you know when you have found the cause of autonomic dysreflexia?
   A. rapid resolution of signs and symptoms
   B. lab tests become negative
   C. the individual will be able to tell you
   D. sweating begins below the level of injury

4. What combination of signs and symptoms constitute a “red flag” for the clinician working with an individual with a spinal cord injury above T6?
   A. Incontinence and anger
   B. Pain and goose bumps above the level of injury
   C. Fatigue and skin flushing above the level of injury
   D. Pounding headache and sweating above the level of injury

5. What pathophysiologic mechanism underlies autonomic dysreflexia?
   A. Noxious stimuli from below the injury level caused a reflex increase in parasympathetic output from above the level of injury.
   B. The spinal cord injury prevents the normal vasodilation response to increasing blood pressure from occurring below the level of injury.
   C. The inability to perceive pain below the injury causes the nervous system to over react to noxious stimuli from below the injury level.
   D. The spinal cord injury prevents noxious stimuli from below the injury from being responded to until they become life threatening.

## Answers to Review Questions

1. D
2. B
3. A
4. D
5. B

## Appendix A

A wallet-sized card detailing the signs, symptoms and treatment for autonomic dysreflexia that can be downloaded and printed for distribution to patients to carry with them after discharge from rehabilitation.

Instructions: print this page then cut to fit mounting signs and sumptoms and examination on the front of the card and treatment on the back. The card may be laminated to increase durability.

**AUTOMOMIC DYSREFLEXIA**
A Life Threatening Emergency

**COMMON SIGNS AND SYMPTOMS MAY INCLUDE**:

| | |
|---|---|
| HYPERTENSION | CHILLS WITHOUT FEVER |
| POUNDING HEADACHE | SWEATING ABOVE LEVEL OF INJURY |
| BRADYCARDIA | SKIN FLUSH ABOVE LEVEL OF INJURY |
| SEIZURES | GOOSE BUMPS ABOVE LEVEL OF INJURY |

**EXAMINATION**

Look for noxious stimuli below level of injury
Check bladder: for distension or infection
Check Bowel: for impaction
Check skin: remove constrictive clothing, examine for pressure ulcers, insect bites, sharp or hard objects, ingrown toenails, temperature changes
Males:genitals pinched, condom too tight, reflexogenic erection
Females: menstrual cramping, uterine contractions, vaginitis **OVER**

**TREATMENT**

1. Treat elevated blood pressure until cause is found and eliminated
2. Medications commonly used for elevated blood pressures:
   - Nifedipine 10 mg capsule. May repeat in 20-30 minutes if blood pressure has dot decreased. Observe for possible hypotension.
   - Hyperstat 300 mg IV push and monitor blood pressure every 3 minutes – watch for hypotensive crisis.
3. Anesthetize noxious stimuli, if necessary, before removal.
4. Monitor blood pressure frequently.

**Autonomic dysreflexia can lead to seizures, stroke, or death.**

## Appendix B

An 8 1/2” x 14” poster, suitable for downloading and displaying in rehabilitation clinics, describes the most common signs, symptoms, examination, and treatment of autonomic dysreflexia.