# "Oh, the Pounding!"

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## L.C.'s Case

L.C., a 30-year-old male C7 paraplegic, caused by a bullet wound two years previously, presents to the ED having passed no urine from his suprapubic catheter for 12 hours. His caregiver tried to flush it without success. An attempt to flush it in the ED failed, and the catheter was removed with the plan to insert a new one. During the attempt to replace the catheter, the patient suddenly developed a pounding headache. He became flushed, diaphoretic, nauseated and his vision became blurred. His blood pressure, which was 110/70 mmHg in triage, then read 210/100 mmHg.

Read on for more on L.C.



### *Questions and Answers*

### 1. *What is going on?*

The differential diagnosis of severe headache and hypertension of sudden onset includes the potential causes listed in Table 1. In a patient who has suffered a high (*i.e.*, > T6) spinal chord injury, a high degree of suspicion for autonomic dysreflexia should be maintained, as the condition has the potential to rapidly lead to serious sequelae.

### 2. *What is the pathophysiology of autonomic dysreflexia?*

Autonomic dysreflexia (AD) may occur in patients with spinal cord lesions at or above the level of T6, and is the result of losing downward central control of a normal spinal reflex. A noxious stimulus below the level of the lesion excites afferent sensory neurons, which initiate the spinal reflex. Within the spinal cord, these signals are transmitted to efferent sympathetic neurons that elicit the generalized sympathetic response, including vasoconstriction and the resulting rise in blood pressure. In a person with an intact spinal cord, these signals would also be sent upwards to the brainstem, which would then mediate the sympathetic response through downward inhibitory pathways, attenuating the surge in blood pressure. These inhibitory pathways are interrupted in patients with a spinal cord injury.

Without this central control to moderate the sympathetic response, vasoconstriction results in hypertension, the severity of which is dictated by the number of vessels involved.

Intact baroreceptors above the level of the lesion respond to hypertension and send signals to the brainstem, which in turn sends out mediating parasympathetic outflow. Parasympathetic outflow results in reflex bradycardia (although tachycardia is also common), and vasodilation above the level of the lesion. Parasympathetic outflow dominates above the level of the lesion, while sympathetic outflow dominates below; this results in the interesting clinical presentation of cool, dry skin below the level of the lesion and flushed, sweaty skin and nasal congestion above.

In patients with lesions below the level of T6, exclusion of the splanchnic vasculature from

**Table 1**

**Differential Diagnoses of Severe Headache and Hypertension**

- Subarachnoid hemorrhage
- Migraine
- Cluster headaches
- Essential hypertension
- Posterior fossa tumours
- Pheochromocytoma
- Abuse of sympathomimetic drugs
- Combination of monoamine oxidase inhibitors and tyramine-containing foods
- Autonomic dysreflexia in paraplegics or quadruplegics
- Toxemia of pregnancy

uncontrolled vasoconstriction results in milder, and therefore less dangerous forms of autonomic dysreflexia.

## 3. What do we need to remember about AD in the ED?

In severe cases, blood pressure may rise to levels of 300/220 mmHg. Bearing in mind that blood pressure is normally decreased after spinal cord injury, even readings of 120/80 mmHg may represent elevated pressure. Blood pressure, therefore, must be compared to the individual's baseline values. Elevated pressure may manifest as bilateral pounding headache, vision changes, malaise and nausea, and if untreated may result in arrhythmia, pulmonary edema, cerebral or subarachnoid hemorrhage, seizures, coma or death.

Autonomic dysreflexia may be triggered by any noxious stimulus occurring below the level of the lesion. Lower urinary tract stimulation is the most common precipitant; this includes bladder distension, infection, instrumentation, stones or testicular torsion. If the patient has a catheter, it should be checked for blockages. Flushing or replacing a catheter may further trigger AD, but may be necessary. Bowel distension is the second most common AD precipitant mainly due to constipation or fecal impaction; however, distension can also be caused by instrumentation, hemorrhoids, and anal fissures.

Additional triggers range from urinary or gastrointestinal tract dysfunction, to cutaneous stimulation through restrictive clothing, pressure sores or ingrown toenails, to musculocutaneous pathology, including fractures and dislocation. Normal bodily functions such as menstruation, labour and delivery, and sexual activity can elicit AD. Even medications, including sympathomimetics, nasal decongestants and misoprostol, may trigger AD.

*Bladder distension and bowel distension due to fecal impactation are the number one and two most common precipitants of autonomic dysreflexia.*

## 4. How do I treat AD in the ED?

As potential sequelae of severe uncontrolled hypertension are very serious, and because the cause may be occult, the most important measure in treatment of AD is early recognition. Elevated BP with any distressful symptoms necessitates an immediate response to potential AD, beginning with sitting the patient upright to

## Back To L.C.

Our patient developed the typical signs and symptoms of autonomic dysreflexia as a result of bladder distention. He was moved from a lying to a sitting position, and manipulation of his catheter was stopped until his hypertension and symptoms were controlled with sublingual nitroglycerin and chewed captopril 25 mg. His blocked indwelling suprapubic catheter was removed and replaced. Unfortunately, stimulation from insertion of the new catheter further aggravated his AD, resulting in a severe pounding headache, blurred vision, nausea, blood pressure of 180/98 mmHg and reflex bradycardia. He was seated upright and treated with sublingual nitro spray, and the procedure was performed as quickly as possible to try and minimize noxious stimulation.

A few minutes after the procedure, he returned to baseline. His urine showed evidence of bacterial growth; this was believed to be in response to colonization rather than acute infection. Considering his rapid recovery after relief of the obstruction, antibiotic treatment was not prescribed, although he was cautioned to return in the event of fever, nausea or recurrence of AD symptoms. The diagnosis of AD and the high potential for recurrence was discussed, and he was advised to remind all health care workers from whom he received treatment that he was at risk.

induce an orthostatic drop in blood pressure. Restrictive clothing and devices should be removed before initiating a more complete search for potential precipitants. If possible, these precipitants should be eliminated; however, it is important to remember that therapeutic actions such as passing or flushing a catheter, or manual bowel disimpaction, may temporarily exacerbate the dysfunctional autonomic response.

While potential precipitants are sought out, blood pressure should be monitored every two to five minutes. A systolic pressure of 150 mmHg or greater necessitates pharmacologic treatment of blood pressure. Since the cause of the patient's hypertension is temporary, the ideal antihypertensive to use should have a rapid onset and short duration. Although no randomized controlled trials dictate treatment, conventional treatments of choice are immediate release nifedipine and nitrates (spray or paste). If these are unsuccessful, additional treatments include sodium nitroprusside, hydralazine, mecamylamine, diazoxide, prazosin, captopril, and clonidine. If the noxious stimulus cannot be removed (*e.g.*, a pressure sore), prophylactic treatment with an α-blocker may be warranted. Admission to the hospital may be required to ensure maintenance of blood pressure control, especially in cases where control proves difficult or where the offending stimulus cannot be removed.

Patients should be educated on how to prevent the recurrence of autonomic dysreflexia. Instruction regarding proper bladder, bowel and skin care is an important part of prevention. Teaching the patient to recognize signs and symptoms of AD allows the patient to be his/her own advocate, and seek help when it is needed. Dx

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Publication Mail Agreement No.: 40063348
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