



# Postoperative Central Cord Syndrome: Physiologic Decapitation in the PACU

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## Abstract

Central cord syndrome is a form of incomplete spinal cord injury appearing in the anesthesia literature primarily in trauma. Our institution recently managed a rare, life-threatening central cord syndrome following an uneventful anterior cervical discectomy and fusion which uniquely presented as respiratory depression progressing to quadriplegia. This patient's dramatic experience began nearly indistinguishably from more common etiologies of respiratory depression in the post-anesthesia care unit before blossoming swiftly into quadriplegia. We review the details of her presentation and pathophysiology with a message of vigilance to the anesthesia provider. Interpretation of her clinical exam and rapid intervention were key to preventing a lethal outcome. Central cord syndrome should be considered by anesthesiologists in the differential for respiratory depression following cervical spine surgery.

## Keywords

- ▶ hypercapnia
- ▶ spinal cord anatomy
- ▶ spinal cord blood flow

## Introduction

Perioperative central cord syndrome (CCS) is described in the anesthesia literature in scattered case reports following emergent intubations or prolonged cervical extension for surgical positioning in patients with pre-existing cervical stenosis.<sup>1–3</sup> Presentation is classically one of “man in a barrel,” weakness primarily affecting upper extremities, sometimes with lower extremity involvement. Our post-anesthesia care unit (PACU) recently encountered a patient in whom CCS was caused by a cervical epidural hematoma following cervical spine surgery, presenting initially as respiratory depression, which we found no prior reports of. Rapid interpretation of physical exam was the key to the patient's emergent management. We present her presenta-

tion as well as her pathophysiology in a message of vigilance to the anesthesiologist. The patient provided written Health Insurance Portability and Accountability Act authorization for publication.

## Case Report

An otherwise healthy 46-year-old woman with a history of cervical stenosis arrived in our institution's PACU for recovery following an uneventful C3 to 5 anterior cervical discectomy and fusion (ACDF) with C4 corpectomy. Recovery began unremarkably, and she was alert, conversant, and moving all extremities appropriately. Preparations were being made for transfer to the ward when she reported neck pain and received intravenous fentanyl and hydromorphone.

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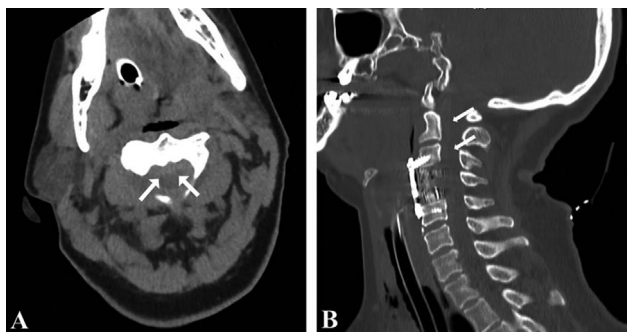
Shortly thereafter, the supervising anesthesiologist was summoned to evaluate shortness of breath. He found the patient dyspneic with shallow respirations and difficulty moving her hands. She appeared somnolent but remained arousable, which improved with naloxone administration and gentle, passive ventilatory assistance via a Jackson Rees circuit and supplemental oxygen. Once more alert, the patient was able to clench her eyes and jaw with good strength. She mouthed words in response to questions appropriately though her voice grew increasingly weak and inaudible. The patient's subsequent neurologic exam then deteriorated rapidly to full paralysis of the upper extremities followed by weakness in the lower extremities and eventual quadriplegia—all while awake and aware!

As preparations were made for re-intubation, an arterial line was placed for hemodynamic management in the setting of a suspected spinal cord pathology. The patient winced with radial artery cannulation but was unable to flinch her arms in recoil. She was induced and intubated for respiratory failure uneventfully via video laryngoscopy and was taken sedated for emergent imaging which showed a fluid collection in the anterior epidural space spanning from C1 to C6 (►Fig. 1). The patient's vital signs were notable for significant systolic hypertension which resolved with ventilatory support and mechanical ventilation and were thus attributed to hypercapnia.

The patient was taken to the operating room for emergent exploration of her surgical site under neuromonitoring. Mean arterial pressure was maintained above 85 mm Hg as surgeons evacuated a cervical epidural hematoma and achieved hemostasis of local venous bleeding. Motor evoked potentials, initially absent in all extremities, gradually returned in the legs and, then, arms. Her initial surgery was ultimately converted to a C2 to 5 ACDF with additional corpectomy at C3, and the patient made a full neurologic recovery with the exception of urinary retention at the time of discharge.

## Discussion

This patient's presentation is one of CCS with life-threatening respiratory depression, paraplegia of upper extremities fol-

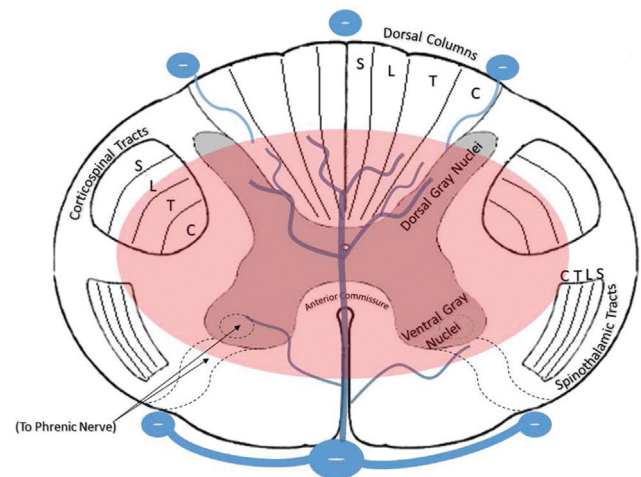


**Fig. 1** Representative slices of the patient's emergent CT, demonstrating a fluid collection in the anterior epidural space (indicated by white arrows). Artifact from newly placed surgical hardware limit the visibility of the lesion. An image of the lesion from the "bone window" is included.

lowed by quadriplegia with sparing of cranial nerves as well as pain sensation in affected limbs. Had rapid recognition and supportive measures not been immediately available, this patient's outcome may have been devastating. One can imagine how terrifying her experience must have been as she was essentially physiologically decapitated. Reviewing basic spinal cord anatomy and physiology aids in understanding this woman's presentation.

We interpret the rapid onset (and resolution) of the patient's syndrome as suggestive of an obstructive vascular etiology,<sup>4</sup> as explained below. The spinal cord is perfused by paired posterior spinal arteries and a single anterior spinal artery. Venous blood exits the spinal cord via small, penetrating intramedullary venules which merge into anterior and posterior spinal veins running longitudinally along the cord's surface. The anterior medial spinal vein drains venous blood from the center of the cord and thus impedance of its drainage can specifically result in central spinal cord ischemia via edematous inhibition of blood flow. Epidural space compression may further impair spinal cord blood flow via the obstruction of the epidural venous plexus, which receives venous blood from radicular veins exiting neuroforamen after draining the aforementioned surface veins.<sup>4,5</sup>

We suspect that this patient's expansive cervical anterior epidural hematoma obstructed drainage of the anterior medial spinal vein and epidural venous plexus, resulting in edematous impedance to blood flow within the central spinal cord (►Fig. 2). Within her affected C3 to 5 levels, the resulting distribution of ischemia compromised centrally located ventral gray nuclei as well as exiting ventral nerve roots, resulting initially in a lower motor neuron-type



**Fig. 2** Artist's rendition of this patient's cervical spinal cord lesion. Pertinent spinal cord tracts and structures appear labeled with their somatotopic organization indicated (C, cervical; T, thoracic; L, lumbar; S, sacral). The area in red represents the cord lesion from focal ischemic injury, presumably following the obstruction of the depicted spinal venous system. Note that the affected area includes ventral gray nuclei and projections to the phrenic nerve, producing the patient's presenting respiratory depression from a central motor lesion. The involvement of the cervical corticospinal tracts with spinothalamic tract sparing explains the subsequent "man in the barrel" motor paralysis classic to a central cord syndrome, which preceded her ultimate quadriplegia.

diaphragmatic paralysis. Ensuing hypercapneic respiratory failure, likely compounded by opioid administration, superimposed carbon dioxide narcosis. This explains her initial depressed level of consciousness improved by naloxone and ventilator support.

As the area of ischemia expanded outward, loss of the descending upper motor neurons (UMNs) of the corticospinal tracts resulted in sequential UMN-type paralysis of the upper and, then, lower limbs according to the somatotopic arrangement of the tracts (medially located cervical neurons and laterally located sacral neurons). Hematoma evacuation alleviated venous compression, normalizing cord perfusion, and neurological function was restored. Sparing of laterally located, ascending sensory neurons of the spinothalamic tracts explains our patient's preserved perception of pain (with arterial cannulation) and distinguishes her syndrome from either a complete cord transection or an anterior spinal artery syndrome in both of which the spinothalamic tracts are compromised. The patient's presentation with bilateral deficits distinguishes her case from a Brown-Sequard (cord hemitranssection) syndrome.<sup>1,6</sup>

This patient's mechanism of injury is distinct from the classical description by Schneider in 1954 of guillotine-like axial compression of the cervical spinal cord by inward tenting of dorsal ligamentum flavum during cervical hyperextension—the cause of CCS within the trauma literature.<sup>1,7–10</sup> Fortunately, our patient's presentation occurred while still in a closely monitored PACU. The rapidity of onset coupled with the patient's inability to speak or call for help would have very possibly led to a fatal outcome. We urge anesthesiologists to consider CCS as part of their differential for respiratory depression in patients having undergone cervical spine surgery.

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#### Conflict of Interest

None declared.

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