Early Versus Late Decompression for Central Cord Syndrome

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I. Definitions and Epidemiology
   a. First described by Schneider et al in 1954.[1] Most common pattern of cervical cord injury (9% of traumatic SCI), characterized by relatively greater weakness, sensory loss, and loss of fine motor control in upper extremities versus lower extremities.
   b. ASIA motor score for upper extremities at least 10 points less than lower extremities.
   c. Upper extremity and trunk pain and paresthesias are typically described as tingling, burning, aching. Related to laminar pattern of corticospinal and spinothalamic tracts, medial position of cervical elements relative to lumbar/sacral. Mechanism of injury often involves forceful neck extension. Bimodal distribution with respect to age. Higher energy injuries among younger males. Lower energy injuries among patients age > 50 due spondylotic stenosis. Cord concussion/contusion, axoplasmic stasis and tissue edema versus shear injury and hemorrhage. Central cord hemorrhage associated with less favorable prognosis. Autopsy often shows selective axonal disruption of lateral column white matter with preservation of gray matter. Most commonly involves mid-to lower cervical spine.

II. Natural History
   a. Favorable prognosis in terms of significant functional recovery, but neurological recovery typically incomplete. Majority maintain or recover independent ambulation and continence. Characteristic progression of recovery beginning with lower extremities, followed by bladder, upper extremities, and hands. Prognostic factors include age, associated comorbidities, and injury severity.
   b. Highest rate of improvement in age < 50. Chronic sphincter dysfunction associated with age > 70.[2] Poor outcome in age > 70.[3]

III. Patient Evaluation
   a. Physical Exam
      i. Sensory level, including dull/sharp or two-point discrimination, temperature, proprioception, including perineum/perianal.
      ii. Strength grading of representative muscle groups for cervical and lumbosacral nerve roots.
      iii. Reflexes often absent/depressed in early stage, hyperreflexia and clonus in later stage with muscle spasticity.
      iv. Fine motor coordination
      v. Balance and gait
   b. Radiology
      i. Plain radiography
         1. Flexion/extension views?
      ii. Advanced imaging
1. Identify severity, anatomic location and tissue source of any ongoing spinal cord impingement.
2. Greater axial extent of cord signal changes may be associated with greater initial impairment.[4]

iii. Common spinal injury patterns in younger patients
   1. Fracture-dislocation
   2. Burst fracture

c. Bladder Function

IV. Patient Management
   a. Hard collar
   b. Bedrest
   c. ICU admission
   d. Maintain MAP 85-90 mmHg
   e. Traction?
   f. Corticosteroids? (Recommended by AANS website, updated March 2015)
   g. Thromboembolism prophylaxis
   h. Special considerations
      i. Autonomic dysreflexia: hypertensive response to excessive bladder distension or otherwise unperceived noxious stimuli. Requires diligent monitoring of skin, bowel/bladder function.
      ii. Neurogenic bladder
         1. Adequate function typically returns within 6 months
         2. >50% regain normal bladder function
      iii. Spasticity
         1. Lioresal (baclofen)
      iv. Neuropathic pain
         1. Carbamazepine, gabapentin

V. Surgery
   a. No generally accepted standards or guidelines for treatment
   b. No prospective or randomized studies
   c. Expert opinion recommending surgery in selected cases.[5-7]
   d. One guideline in AHRQ National Guideline Clearinghouse
      i. Management of acute traumatic central cord syndrome (ATCGS). In: Guidelines for the management of acute cervical spine and spinal cord injuries[8]
      iii. Based largely on systematic review of 29 selected articles. “On occasion, the assessed quality of the study design was so contentious and the conclusions so uncertain that the guideline authors assigned a lower medical evidence classification than might have been expected without such a detailed review.”
      iv. Only Level III recommendations (based on case series with poor reference standards/expert opinion):
1. ICU management, particularly for patients with severe neurological deficits
2. Medical management, including cardiac, hemodynamic, and respiratory monitoring, and maintenance of MAP 85-90 mmHg for first week after injury to improve spinal cord perfusion
3. Surgical decompression of compressed spinal cord, particularly if compression is focal and anterior
e. US Nationwide Inpatient Sample data: Percentage of patients with central cord syndrome undergoing surgery increased between 2000 (14.8%) and 2009 (30.5%). Lower mortality rate among surgically treated patients compared to nonoperative treatment (2% vs. 2.7%) and lower pulmonary embolism rate (0.5% vs. 1.2%).[9]
f. Anterior cervical decompression and fusion most common surgery.[10]
g. Recent literature review provides class III evidence in favor of surgical treatment compared to conservative care.[11]

VI. Case for Early Decompression
a. Indication in setting of clear neurological deterioration.
b. Relative indication in patients unable to comply with/tolerate activity restrictions and conservative management.
c. Traditional bias against surgery derives from original clinical series by Schneider in 1954 prior to development of CT/MRI.[1]
d. Advent of advanced imaging allows visualization of disc herniations/degree of stenosis.
e. Ongoing high-grade stenosis/cord compression may be source of progressive cord injury.

VII. Case for Late Decompression
a. Acutely injured spinal cord may be more vulnerable to intraoperative injury.
b. Many patients experience spontaneous neurological improvement with early conservative management up to 6 weeks.
c. Surgery when recovery plateaus in setting of ongoing cord compression or spinal instability.
d. Literature review suggests early surgery associated with more recovery at < 1 year follow-up but comparable outcomes at > 1 year follow-up.[12]

VIII. Postoperative Management
a. Physical therapy to promote “activity dependent plasticity” through repetitive task-specific sensory input.[13]
b. Occupational therapy
   i. Hand splinting to prevent contracture and maintain functional positioning
   ii. Targeted strengthening of weak upper extremity muscles may be facilitated through surface EMG biofeedback.

IX. Conclusion
a. No strong clinical evidence to support surgical treatment over nonoperative management in general
b. Widespread expert opinion support among spine surgeons for selective surgical treatment

c. No strong clinical evidence to support early versus delayed surgical treatment

d. Early surgical decompression may be recommended for patient with active neurological deterioration, especially in setting of high-grade ongoing cord compression/stenosis from acute disc herniation.

e. Delayed surgical decompression may be recommended for patient experiencing spontaneous neurological recovery, especially in setting of low-grade cord compression/stenosis from chronic spondylosis. Reasonable to delay surgery until neurological recovery plateaus, patient is clinically stable, and optimal surgical team/environment can be prepared.


