

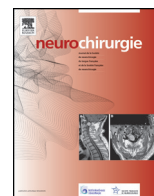


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Update

Acute traumatic central cord syndrome: A comprehensive review



Syndrome centromédullaire aigu post-traumatique : revue systématique

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ABSTRACT

Acute traumatic central cord syndrome (ATCCS) is the most common type of incomplete spinal cord injury, characterized by predominant upper extremity weakness, and less severe sensory and bladder dysfunction. ATCCS is thought to result from post-traumatic centro-medullary hemorrhage and edema, or, as more recently proposed, from a Wallerian degeneration, as a consequence of spinal cord pinching in a narrowed canal. Magnetic Resonance Imaging is the method of choice for diagnosis, showing a typical intramedullary hypersignal on T2 sequences. Non-surgical treatment relies on external cervical immobilization, maintenance of a sufficient systolic blood pressure, and early rehabilitation, and should be reserved for patients suffering from mild ATCCS. Surgical management of ATCCS consists of posterior, anterior or combined approaches, in order to achieve spinal cord decompression, with or without stabilization. The benefits of early surgical decompression in the setting of ATCCS remain controversial due to the lack of clinical randomized trials; recent studies suggest that early surgery (less than 72 hours after trauma) appears to be safe and effective, especially for patients with evidence of focal anatomical cord compression.

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R É S U M É

Le syndrome centromédullaire aigu post-traumatique (SCAPT) est la forme la plus fréquente de lésion médullaire incomplète, caractérisée par la prédominance d'une faiblesse motrice aux membres supérieurs et accompagnée d'une dysfonction sensitive et vésicale moindre. Le SCAPT pourrait résulter d'une hémorragie et d'un œdème intramédullaire ou plutôt, comme récemment démontré, d'une dégénérescence Wallérienne. Dans les deux cas l'origine serait un pincement médullaire aigu chez un patient avec canal cervical étroit. L'IRM reste la technique de référence pour le diagnostic car permet de mettre en évidence le typique hypersignal intramédullaire en séquence T2. Le traitement non-chirurgical comprend l'immobilisation cervicale, le maintien d'une bonne pression sanguine systolique et la réhabilitation précoce, et il devrait être réservé aux patients présentant les formes modérées de SCAPT. Le traitement chirurgical du SCAPT peut se faire par abord antérieur, postérieur ou combiné consistant principalement à faire une décompression de la moelle, sans ou avec une stabilisation vertébrale. Les bénéfices portés par une décompression chirurgicale précoce dans un contexte de SCAPT sont controversés dans la littérature, ceci s'explique par le manque d'essais cliniques randomisés à ce sujet. Les études récentes suggèrent qu'une décompression précoce (moins de 72 heures après le trauma) semble être sans trop de danger et efficace particulièrement pour les patients avec mise en évidence d'une compression médullaire focale.

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1. Introduction

Cervical spinal cord injury may result in different types of syndromes, one of which is the acute traumatic central cord syndrome

(ATCCS). It is the most common acute incomplete cervical spinal cord injury accounting for 70% of all incomplete cervical spinal cord injuries [1]. About 20% of patients with cervical spinal cord injuries present a clinical ATCCS [2,3].

First described by Schneider et al. in 1954, the clinical presentation of ATCCS is characterized by a motor weakness more severe in the upper than in the lower extremities [4,5]. A variable degree of sensory loss below the level of injury is present.

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Typically a bilateral loss of pain and thermal sensation due to damage of the anterior white commissure may occur, mimicking a “cap” distribution over the shoulders and upper extremities. ATCCS may be associated with sphincter dysfunction, if the preganglionic autonomous neurons are impaired. Recent studies propose a new definition stipulating that a score difference of at least 10 points in the American Spinal Injury Association (ASIA) score between the upper and the lower limbs is required to define the injury as ATCCS [6,7].

The aim of this article is to review the physiopathological mechanisms involved in ATCCS, to explain the role of modern imaging techniques, and to provide a review of the literature regarding the management of the ATCCS, focusing on timing of decompression and its impact on clinical outcomes.

2. Search criteria

We conducted a MEDLINE search focused on ATCCS using PubMed, including only English language publications from 1936 to 2013. References cited in the articles were also reviewed to include any other relevant information. Finally, standard spine textbooks were used to supplement this analysis. For pathogenesis, the search headings included the following words: “central cord syndrome pathogenesis”, “hematomyelia”, “edema”, “primary and secondary injury”, “Wallerian degeneration”. The abstracts of relevant citations were reviewed and articles then selected. For neuroimaging, the following search terms were used: “MRI”, “CT scan”, “X-ray”, “intramedullary changes”, “edema”, “spondylosis”. The abstracts were reviewed, and applicable papers further selected. For management of ATCCS, we used standard search terms along with MeSH headings, i.e. “surgical management of ATCCS”, “non-surgical management”, “collar immobilization”, “medical treatment”, “anterior and posterior approaches”, “combined approaches”, “laminectomy”, “laminoplasty”, “ACDF”, “ACCF”. The relevant papers have been inserted and detailed in Tables 1–3. We graded all manuscripts as class III evidence, in the absence of randomized control trials (Table 1). There is a fairly wide range of literature concerning ATCCS, and therefore it was difficult to be completely exhaustive. Some items were not selected because their abstract did not seem relevant for this review or because the findings were similar to other studies previously mentioned.

Table 1

Summary of manuscripts with systematic review of the literature on SCI.
Résumé des articles de type revue de la littérature sur les lésions médullaires.

Citations	Description of the study	Timing of intervention	Conclusions
Aarabi et al., 2013 [6]	Systematic review of the literature	Undetermined	Surgical decompression particularly if the compression is focal is recommended. Patient age and comorbidities are important factors when considering surgical treatment
Nader et al., 2013 [75]	Systematic review of the literature	Undetermined	Superiority of surgery to conservative treatment for ATCCS. No difference in early versus late surgical management
Aarabi et al., 2008 [8]	Systematic review of the literature	Undetermined	Any type of recommendation for the timing of surgery remains an option
Fehlings et al., 2010 [9]	Systematic review and a 20-questions survey sent to spine surgeons	< 24 h	No consensus between spine surgeons (971) concerning surgical decompression in ATCCS due to spinal stenosis
Fehlings et al., 2006 [10]	Systematic review of the literature	< 24 h or < 72 h	Urgent decompression seems to be recommended in a patient with incomplete tetraplegia or with neurologic deterioration. Decompression within 24 hours may reduce length of intensive care unit stay and medical complications
La Rosa et al., 2004 [11]	Systematic review of literature	< 24 h; > 24 h	Early decompression gives better outcomes

SCI: spinal cord injury.

3. Pathophysiology of ATCCS

3.1. Animal models for ATCCS

There exists preclinical evidence that the amount of compression and its duration time are two factors negatively related to neurological improvement [10,27–29]. Moreover, although evidence in humans is less clear, animal evidence is in favor of early decompression. This is explained by the physiopathological mechanisms involved. In fact, primary and secondary lesions can characterize acute spinal cord injury (SCI). Primary lesions are directly due to the damage caused by the initial compressing trauma. This “initiates a cascade of secondary injury mechanisms, including ischemia, electrolyte derangements, and lipid peroxidation” [10,27]. It is mainly in order to avoid these secondary lesions that medical and surgical support play a fundamental role and also emphasize the importance of the timing of decompression [10,27,30].

3.2. ATCCS in humans

The mechanisms involved in ATCCS, initially reported by Taylor et al. [31], have been updated by Schneider et al. [4,5]. These authors proposed that cervical hyperextension causes acute spinal canal narrowing by anterior protrusion of the ligamentum flavum with subsequent spinal cord pinching. Injury is facilitated in the setting of a stenotic spinal canal primarily caused by degenerative changes in older patients and congenital narrowing in younger patients. Age distribution in ATCCS is bimodal [32–34] with a first peak in patients under 30 years old, suffering from high velocity injury, and a second peak in elderly patients with low velocity trauma in the setting of cervical stenosis [8,32,33].

Schneider et al. initially described the presence of hematomyelia centrally located in the spinal cord post mortem, which led to the destruction of the central cord structures [5]. Nonetheless, more frequently hematomyelia was absent, and the injury was caused by edema alone. This correlates with the autoptic study published by Quencer et al., where only axonal lesions were identified with no hemorrhage [8,35]. The explanation of the disproportionate motor impairment between upper and lower limbs is not clear. Historically, the preferential involvement of the upper limbs has been

Table 2

Evidentiary summary of manuscripts examining the comparison between early surgery, late surgery and conservative management.
Mise en évidence des manuscrits comparant la prise en charge chirurgicale précoce, chirurgicale tardive et conservative.

Citations	Description of the study	Study population	Timing of intervention	Conclusions
Fehlings et al., 2012 STASCIS [12]	Multicenter prospective cohort study	182 patients underwent early surgery and 131 late surgery	< 24 h	Early decompression can be performed safely and is associated with improved neurological outcomes
Aarabi et al., 2011 [13]	Retrospective study of prospectively collected data	211 patients treated for ATCCS	< 24 h 24 h to 48 h > 48 h	Timing of decompression does not play a significant role
Stevens et al., 2010 [14]	Retrospective review	126 patients diagnosed with CCS and 67 received surgery	< 24 h; > 24 h; or second hospitalization	No statistically significant difference in neurologic outcome was identified with regard to timing of surgery
Chen et al., 2009 [15]	Retrospective review	A total of 49 patients with ATCCS who underwent surgical intervention	< 4 days or > 4 days	The improvement in the ASIA motor score was positively correlated with age at injury
Aito et al., 2007 [16]	Retrospective review	82 patients, 45% operated and 55% had conservative treatment	Not specified	Surgical decompression did not affect outcome
Guest et al., 2002 [17]	Retrospective review	50 patients treated surgically	< 24 h or > 24 h	Early surgical decompression is overall in favor of a better recovery, especially if the cause is disc herniation or fracture/dislocation
Papadopoulos et al., 2002 [18]	Prospective. Non randomized study	A total of 91 patients with acute traumatic cervical spinal cord injury	< 24 h	Early decompression is feasible and may significantly improve neurological outcome
Mirza et al., 1999 [19]	Retrospective case series	A total of 43 patients of two different center	< 72 h	Early decompression is feasible and improves neurological recovery
Chen et al., 1998 [20]	Prospective. Non randomized	37 patients with cervical spondylosis, 16 surgery and 21 conservative treatment	< 2 weeks	Surgery is associated with shorter hospital stay and improved neurological recovery
Chen et al., 1997 [21]	Retrospective review	114 patients with ATCCS, 28 surgical and 86 medical treatment	From 6 days to 24 months	Younger patients had better recovery
Bose et al., 1984 [22]	Retrospective study	28 patients, 14 medical therapy alone and 14 surgical and medical	Not assessed	Operative intervention did not produce neurological worsening
Brodkey et al., 1980 [23]	Case reports	7 patients with ATCCS	3 to 6 weeks	All patients improved very rapidly
Schneider et al., 1954 [5]	Review of 14 cases	8 personal cases and 6 from the literature	Not assessed	They do not recommend surgical treatment because most patients improved spontaneously

STASCIS: Surgical Timing in Acute Spinal Cord Injury Study; ATCCS: acute traumatic central cord syndrome.

explained by the somatotopic organization of the corticospinal tract, where fibers for the upper extremities are more centrally located than those for the lower extremities [5,6,8,36]. However, Levi et al. criticized such a somatotopic organization of the corticospinal tract in primates [8,37]. Subsequently, Jimenez et al. published the evidence of a Wallerian degeneration of the corticospinal tract and suggested that the corticospinal tract fibers seem preferentially involved in motor control of upper extremities rather than in that of lower extremities [38].

4. Imaging for ATCCS

Magnetic resonance imaging (MRI) is the examination of choice for the diagnosis of ATCCS, showing intramedullary hypersignal on T2-weighted and STIR sequences consistent with edema as well as

lesions of ligaments and intervertebral discs [39,40]. Aarabi et al. recently observed a positive correlation of admission ASIA motor score and length of parenchymal damage on T2-weighted MRI imaging [6]. MRI is also useful for the assessment of hemorrhage [41,42] and may show the presence of prevertebral hematoma or disruption of posterior ligaments, as a possible indicator of spinal column instability [43]. Conventional MRI has a lower sensitivity than diffusion tensor imaging (DTI) for assessing the correlation between imaging and neurological impairment [41,44–46]. Quantitative DTI and fiber tractography analysis are useful to evaluate, in greater detail, the white matter lesions in the central cord syndrome [46], but this needs to be further developed towards quantification.

Cervical dynamic X-rays play an important role in the radiographic assessment of the eventually associated disco-ligamentous

Table 3
Evidentiary summary of manuscripts focused on conservative management.
Résumé des manuscrits décrivant la prise en charge conservative.

Citations	Description of the study	Study Population	Conclusions
Dvorak et al., 2005 [24]	Retrospective review	70 patients with ATCCS	The functional status correlates with the initial AMS, the level of education, the young age, the absence of spasticity and comorbidities
Newey et al., 2000 [25]	Retrospective study	32 patients were evaluated	Age-related differences in outcome. Spasticity does not appear to cause significant functional problems
Waters et al., 1996 [26]	Prospective multicenter study	19 patients were evaluated	One the average. Patients doubled their initial ASIA motor score at one-year follow-up without surgery
Bosch et al., 1971 [3]	Retrospective review	In a total of 60 patients, there was a subgroup of 42 cases of CCS managed conservatively	Initially an improvement in neurological symptoms was observed but at long-term follow-up (10 years), patients developed spasticity and decreased their functional score
Schneider et al., 1958 [4]	Retrospective review	Two age groups: young patients with fracture dislocation injuries. Older patients with hyperextension injuries	Most patients improve spontaneously. Expectant management seems to be a good treatment

STASCIS: Surgical Timing in Acute Spinal Cord Injury Study; CCS: central cord syndrome; ASIA: American Spinal Injury Association; AMS: Asia Motor Score.

instability, which is either defined according the White and Panjabi criteria or to the more traditional Roy-Camille criteria. The former authors defined cervical instability as follows:

- translational instability: more than 3.5 mm of slippage of the concerned vertebrae;
- rotational instability: more than an 11-degree rotational difference to that of either adjacent vertebra [47,48].

According to the Roy-Camille criteria, the diagnosis of cervical instability relies on the following findings base on cervical standard and dynamics X-rays: increase of interspinous distance, facet dislodgement, discal segmental kyphosis, and anterolisthesis [49].

5. Electrophysiology and ATCCS

The diagnosis of ATCCS is first of all clinical, and then confirmed by neuroimaging. The role of electrophysiological studies, as evoked potentials (i.e. somatosensory evoked potentials (SSEP), motor sensory evoked potentials [MEP]) and electromyography, in ATCCS patients may be useful because of the limiting component of subjectivity using only the standard American Spinal Injury Association (ASIA) classification. Moreover, as the ASIA score has some limitations for detecting modest changes in sensorimotor function, it may be useful to use quantitative measures like SSEP, MEP to achieve a more sensitive and specific approach to detect a neurological deficit and to evaluate neurological improvement after treatment [50–52]. The attenuation of MEPs may be different with the type of spinal cord injury and therefore play a role in diagnosis [52]. For example, in a central cord syndrome, Curt et al. showed that axons devoted to the motor control of the hands are more affected than those devoted to the lower limb [52]. However, some authors realize that these quantitative measures “need to be further evaluated in prospective longitudinal studies” [50,53].

As regards electromyography, this procedure may show signs of denervation in the muscles depending on the anterior horns involved.

6. Management of ATCCS

6.1. Non-surgical treatment

Non-surgical management of a patient with ATCCS consists of a rigid external cervical orthosis, respiratory protection, and maintaining a systolic blood pressure ≥ 90 mmHg to maintain adequate blood supply of the contused spinal cord and limit a secondary injury cascade [6,30,54]. Such non-surgical treatment is usually reserved for patients with mild ATCCS and cervical stenosis, and slight neurological impairment. Schneider et al. reported that functional recovery of these patients is possible; it starts at the lower extremities, followed by recovery of bladder function and finally of the upper extremities [4,5,22,24,33,34,54]. However, this effect is ephemeral for some patients because they develop spasticity and then may decrease their functional score after long-term follow-up (10 years). This phenomenon is called the “chronic central cord syndrome” [3]. The outcome of non-surgically treated ATCCS patients has been analyzed and compared to the outcome in studies where surgical management was performed (Tables 2–3) [3–5,12–21,23–26].

To better understand a potentially favorable spontaneous evolution, some authors have focused on factors predicting the recovery rate. High level of education, young age at injury, higher initial ASIA motor score at admission, absence of comorbidities, and absence of spasticity correlated with a good neurologic recovery [3–5,13,15,21,24,25,33,55]. Song et al. in 2006 reported that the presence of spinal column instability in ATCCS patients predisposes the patient to lower ASIA score at admission [43].

Short-term benefit of conservative treatment may be nullified by the occurrence of mid-term complications in ATCCS patients, such as neuropathic pain and spasticity. Both are potentially disabling conditions, and they are difficult to treat. Neuropathic pain is usually limited to the hands, and it may be treated by specific drugs as gabapentin and lamotrigine [56,57]. Spasticity may be prevented by an adapted physical therapy and treated by specific drugs, such as baclofen, dantrolene, tizanidine and gabapentin [56,58,59]. However, we found that in some of the pre-cited papers, the follow-up of patients is too short [13,22,32,60]. The patients' status could worsen after this period. Moreover, some studies do

not provide sufficient details about the medical treatment administered to patients to prevent spasticity [24,25].

6.2. Surgical treatment

Surgical treatment in ATCCS follows the same principles as for cervical myelopathy. It is indicated for patients with clinically moderate and severe ATCCS and progressive neurological impairment. The best indications for surgery are 1–2 levels of spondylotic changes [17].

6.2.1. The choice of surgical approach

The optimal surgical approach is a matter of debate. Often the choice is made based on the personal experience of the surgeon rather than on a specific algorithm. As a general rule, the ideal surgical approach should target the site of predominant compression of the spinal cord: anterior, posterior, or combined [61]. Several factors may influence this rule. One of them is the extent of the pathology. Usually, if the compression is restricted to one or two levels, the anterior approach is preferred; if more than two levels are involved, the posterior approach may be more advantageous [2,17,62–64]. The sagittal balance of the cervical spine is also an important issue to consider. In cases of cervical kyphosis, where the spinal cord may be stretched over the anterior osteophytes, the combined anterior and posterior approach is suitable, with the anterior approach allowing for decompression and correction of kyphosis, and the posterior approach for stabilization and decompression [65]. In cases of posterior compression and loss of lordosis, posterior stabilization should be added to the decompressive laminectomy. If lordosis is preserved and anterior compression is the problem, and in this case an anterior approach is sufficient. However, in the presence of ossification of the posterior longitudinal ligament an anterior approach is usually contraindicated due to the presence of thigh adhesions between the dura and the ossified posterior ligament, with a high risk of a central spinal fluid leak [66,67].

Evidence of cervical disco-ligamentous instability associated to ATCCS (see criteria above) requires a surgical decompression and stabilization in an emergency situation. This may be achieved either by an anterior approach with cages and plates, or by a posterior approach with lateral mass or pedicle screws and rods.

The fusion rate, the neurological outcome and the complication rate of both anterior (anterior cervical discectomy and fusion [ACDF], anterior cervical corpectomy and fusion [ACCF]) and posterior (laminectomy, laminoplasty) procedures have been compared [68]: current evidence (class III) suggests no difference in the anterior approaches in terms of neurological outcome. In posterior approaches, laminoplasty and laminectomy were equivalent in terms of neurological outcome. However, laminectomy may better preserve range of motion, but may also be associated with late deterioration because of secondary kyphosis [68–70]. Wada et al. reported that no significant difference in neurologic recovery was found between anterior and posterior procedures [71]. Anterior surgery seems to have better clinical outcomes and more complications at the early stage after surgery for multilevel operated patients compared to posterior approaches [72–74].

6.2.2. Timing of decompression

There is evidence in animal models that the degree of compression and its duration are two factors that are negatively related to the neurological improvement [10,27–29]. Nevertheless, surgical decompression in spinal cord injuries has been, for a long time, perceived as contraindicated as surgical manipulation of the “fragile” contused spinal cord was suspected to further damage the cord [4,5,24–26]. Several studies on the other hand reported a better and more rapid neurological recovery after early surgical

decompression [20,21,23]. Moreover, currently, with improved surgical and anesthesiological techniques, surgical decompression in acute medullary lesion has become widely feasible and beneficial in terms of neurological outcome and protection against late progressive neurological deterioration [10–12,14–19,22,43]. The limitations or criticism of some of these studies are the inherent selection bias. The patients were not reviewed in a blinded fashion but retrospectively. For example in the study of Chen et al., there was a tendency to treat younger patients by surgery rather than medically [21,22,60]. In the study of Fehlings et al. in 2012, there are discrepancies between early and late surgery groups where patients with a slightly lower mean age were primarily in the early group [12]. Moreover, as seen in the study of Bose et al. published in 1984, the groups analyzed were not similar in some reported studies [6,15,22,60]. Non-operated patients often present with more spasticity and more late neurological deterioration [3,16,20].

Early surgical decompression has been recommended in patients with incomplete spinal cord injuries by many authors [6,9–12,18] because of reduced length of hospital and intensive care unit stay and improvement of neurological outcome compared with both delayed and conservative management [6,10–12]. In their systematic review of the literature, Fehlings et al. suggested that an urgent decompression is feasible and recommended in patients presenting with progressive neurological deterioration, because decompression may lead to improved outcomes [10]. Papadopoulos et al. as well as Guest et al. also confirmed the above-mentioned results in their prospective and retrospective studies of 91 and 50 patients, respectively [17,18]. There is no clear definition of “early decompression”, but most studies have arbitrarily determined a timeline of 24 hours for early surgery, although some authors have used 72 hours as a time limit. However, most of the publications cited above do not provide sufficient details on the groups of patient studied and preventive treatment used [17,18,22,24,25,60].

Some other remarks on the various publications mentioned are necessary. First, it should be noted that the etiologies of the lesions are sometimes poorly defined and even mixed in some publications [17,24,43,60]. Thus, we believe that a biased interpretation is possible. In addition, the outcomes used to measure recovery are not always the same between articles, making it difficult to perform a good comparative analysis. The timing of the first neurological examination after injury and from initial examination to follow-up was not specifically mentioned in most studies [18,20,22,26,43]. Furthermore, a non-homogeneous description of the neurological status was made between studies. There is an absence of well-designed and well-executed randomized controlled trials and most of these studies lack appropriate controls and thus are subject to selection biases and confounding variables. This may be explained due to ethical concerns about allocating a deteriorating patient to delayed decompression. Furthermore, some studies have a large number of patients lost to follow-up and sometimes the number of patients with complete data is difficult to obtain, which adds a biased interpretation. Another point is that the majority of published studies do not provide outcome indices to generic health-related quality of life, which could be an important factor as spasticity is negatively correlated with functional status but positively with motor function.

7. Conclusions

ATCCS is a potentially disabling condition due to damage of the central part of the cervical spinal cord. Hematomyelia, edema and Wallerian degeneration are common pathophysiological findings in these patients, explaining the variable neurological deficits.

MRI is the diagnostic imaging of choice, showing typical intramedullary hypersignal on T2WI. Early orthotic (collar) and medical management (volume resuscitation and blood pressure augmentation) are essential to maximize the chances of neurological recovery, by preventing the secondary injury cascade. Non-surgical treatment may be proposed to patients with mild ATCCS. Nevertheless, this treatment may predispose to occurrence of persisting neuropathic pain and spasticity.

Contrary to what historically advocated, early surgical decompression seems indicated especially in patients who exhibit progressive neurological deficits. Controversy persists in the literature and no clear consensus can be proposed because of the lack of prospective controlled studies. However, recent studies of class III evidence suggest that early surgery for ATCCS is safe and effective, especially for patients with focal anatomical cord compression.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

References

- [1] McKinley W, Santos K, Meade M, Brooke K. Incidence and outcomes of spinal cord injury clinical syndromes. *J Spinal Cord Med* 2007;30(3):215–24 [Epub 2007/08/10].
- [2] Winston F, Eismont FJ. Controversies in the treatment of central cord injuries. *Spine Surg* 2007;19(4):260–71.
- [3] Bosch A, Stauffer ES, Nickel VL. Incomplete traumatic quadriplegia. A ten-year review. *JAMA* 1971;216(3):473–8 [Epub 1971/04/19].
- [4] Schneider RC, Thompson JM, Bebin J. The syndrome of acute central cervical spinal cord injury. *J Neurol Neurosurg Psychiatry* 1958;21(3):216–27 [Epub 1958/08/01].
- [5] Schneider RC, Cherry G, Pantek H. The syndrome of acute central cervical spinal cord injury; with special reference to the mechanisms involved in hyperextension injuries of cervical spine. *J Neurosurg* 1954;11(6):546–77 [Epub 1954/11/01].
- [6] Aarabi B, Hadley MN, Dhall SS, Gelb DE, Hurlbert RJ, Rozzelle CJ, et al. Management of acute traumatic central cord syndrome (ATCCS). *Neurosurgery* 2013;72(Suppl. 2):195–204 [Epub 2013/03/30].
- [7] van Middendorp JJ, Pouw MH, Hayes KC, Williams R, Chhabra HS, Putz C, et al. Diagnostic criteria of traumatic central cord syndrome. Part 2: a questionnaire survey among spine specialists. *Spinal Cord* 2010;48(9):657–63 [Epub 2010/06/30].
- [8] Aarabi B, Koltz M, Ibrahim D. Hyperextension cervical spine injuries and traumatic central cord syndrome. *Neurosurg Focus* 2008;25(5):E9 [Epub 2008/11/05].
- [9] Fehlings MG, Rabin D, Sears W, Cadotte DW, Aarabi B. Current practice in the timing of surgical intervention in spinal cord injury. *Spine* 2010;35(21 Suppl.):S166–73 [Epub 2010/10/15].
- [10] Fehlings MG, Perrin RG. The timing of surgical intervention in the treatment of spinal cord injury: a systematic review of recent clinical evidence. *Spine* 2006;31(11 Suppl.):S28–35 [Discussion S6. Epub 2006/05/11].
- [11] La Rosa G CA, Cardali S, Cacciola F, Tomasello F. Does early decompression improve neurological outcome of spinal cord injured patients? Appraisal of the literature using a meta-analytical approach. *Spinal Cord* 2004;42(9):503–12.
- [12] Fehlings MG, Vaccaro A, Wilson JR, Singh A, D WC, Harrop JS, et al. Early versus delayed decompression for traumatic cervical spinal cord injury: results of the Surgical Timing in Acute Spinal Cord Injury Study (STASCIS). *PloS One* 2012;7(2):e32037 [Epub 2012/03/03].
- [13] Aarabi B, Alexander M, Mirvis SE, Shanmuganathan K, Chesler D, Maulucci C, et al. Predictors of outcome in acute traumatic central cord syndrome due to spinal stenosis. *J Neurosurg Spine* 2011;14(1):122–30 [Epub 2010/12/21].
- [14] Stevens EA, Marsh R, Wilson JA, Sweasey TA, Branch Jr CL, Powers AK. A review of surgical intervention in the setting of traumatic central cord syndrome. *Spine* 2010;10(10):874–80 [Epub 2010/09/28].
- [15] Chen L, Yang H, Yang T, Xu Y, Bao Z, Tang T. Effectiveness of surgical treatment for traumatic central cord syndrome. *J Neurosurg Spine* 2009;10(1):3–8 [Epub 2009/01/06].
- [16] Aito S, D'Andrea M, Werhagen L, Farsetti L, Cappelli S, Bandini B, et al. Neurological and functional outcome in traumatic central cord syndrome. *Spinal Cord* 2007;45(4):292–7 [Epub 2006/06/15].
- [17] Guest J, Eleraky MA, Apostolides PJ, Dickman CA, Sonntag VK. Traumatic central cord syndrome: results of surgical management. *J Neurosurg* 2002;97(1 Suppl.):25–32 [Epub 2002/07/18].
- [18] Papadopoulos SM, Selden NR, Quint DJ, Patel N, Gillespie B, Grube S. Immediate spinal cord decompression for cervical spinal cord injury: feasibility and outcome. *J Trauma* 2002;52(2):323–32 [Epub 2002/02/09].
- [19] Mirza SK, Krengel 3rd WF, Chapman JR, Anderson PA, Bailey JC, Grady MS, et al. Early versus delayed surgery for acute cervical spinal cord injury. *Clin Orthop Relat Res* 1999;359(359):104–14 [Epub 1999/03/17].
- [20] Chen TY, Dickman CA, Eleraky M, Sonntag VK. The role of decompression for acute incomplete cervical spinal cord injury in cervical spondylosis. *Spine* 1998;23(22):2398–403 [Epub 1998/12/04].
- [21] Chen TY, Lee ST, Lui TN, Wong CW, Yeh YS, Tzaan WC, et al. Efficacy of surgical treatment in traumatic central cord syndrome. *Surg Neurol* 1997;48(5):435–40 [Discussion 41. Epub 1997/11/14].
- [22] Bose B, Northrup BE, Osterholm JL, Cotler JM, DiTunno JF. Reanalysis of central cervical cord injury management. *Neurosurgery* 1984;15(3):367–72 [Epub 1984/09/01].
- [23] Brodkey JS, Miller Jr CF, Harmody RM. The syndrome of acute central cervical spinal cord injury revisited. *Surg Neurol* 1980;14(4):251–7 [Epub 1980/10/01].
- [24] Dvorak MF, Fisher CG, Hoekema J, Boyd M, Noonan V, Wing PC, et al. Factors predicting motor recovery and functional outcome after traumatic central cord syndrome: a long-term follow-up. *Spine* 2005;30(20):2303–11 [Epub 2005/10/18].
- [25] Newey ML, Sen PK, Fraser RD. The long-term outcome after central cord syndrome: a study of the natural history. *J Bone Joint Surg Br* 2000;82(6):851–5 [Epub 2000/09/16].
- [26] Waters RL, Adkins RH, Sie IH, Yakura JS. Motor recovery following spinal cord injury associated with cervical spondylosis: a collaborative study. *Spinal Cord* 1996;34(12):711–5 [Epub 1996/12/01].
- [27] Fehlings MG. Timing of surgery for acute spinal cord injury: from basic science to clinical application. *Essentials of spinal cord injury: basic research to clinical practice*. New York: Thieme; 2012. p. 265–90.
- [28] Guha A, Tator CH, Endrenyi L, Piper I. Decompression of the spinal cord improves recovery after acute experimental spinal cord compression injury. *Paraplegia* 1987;25(4):324–39 [Epub 1987/08/01].
- [29] Kobrine AI, Evans DE, Rizzoli HV. Experimental acute balloon compression of the spinal cord. Factors affecting disappearance and return of the spinal evoked response. *J Neurosurg* 1979;51(6):841–5 [Epub 1979/12/01].
- [30] Smith HE, Albert TJ. *Management of central cord syndrome. Essentials of spinal cord injury: basic research to clinical practice*. New York: Thieme; 2012. p. 329–36.
- [31] Taylor AR. The mechanism of injury to the spinal cord in the neck without damage to vertebral column. *J Bone Joint Surg Br* 1951;33-B(4):543–7 [Epub 1951/11/01].
- [32] Harrop JS, Sharan A, Ratliff J. Central cord injury: pathophysiology, management, and outcomes. *Spine J* 2006;6(6 Suppl.):198S–206S [Epub 2006/11/14].
- [33] Ishida Y, Tominaga T. Predictors of neurologic recovery in acute central cervical cord injury with only upper extremity impairment. *Spine* 2002;27(15):1652–8 [Discussion 8. Epub 2002/08/07].
- [34] Roth EJ, Lawler MH, Yarkony GM. Traumatic central cord syndrome: clinical features and functional outcomes. *Arch Phys Med Rehabil* 1990;71(1):18–23 [Epub 1990/01/01].
- [35] Quencer RM, Bunge RP, Egnor M, Green BA, Puckett W, Naidich TP, et al. Acute traumatic central cord syndrome: MRI-pathological correlations. *Neuroradiology* 1992;34(2):85–94 [Epub 1992/01/01].
- [36] Foerster O. *Symptomatologie der Erkrankungen des rückenmarks und seiner Wurzeln*. Handb Neurol 1936;5:83.
- [37] Levi AD, Tator CH, Bunge RP. Clinical syndromes associated with disproportionate weakness of the upper versus the lower extremities after cervical spinal cord injury. *Neurosurgery* 1996;38(1):179–83 [Discussion 83–5. Epub 1996/01/01].
- [38] Jimenez O, Marcillo A, Levi AD. A histopathological analysis of the human cervical spinal cord in patients with acute traumatic central cord syndrome. *Spinal Cord* 2000;38(9):532–7 [Epub 2000/10/18].
- [39] Miyajiri F, Furlan JC, Aarabi B, Arnold PM, Fehlings MG. Acute cervical traumatic spinal cord injury: MR imaging findings correlated with neurologic outcome – prospective study with 100 consecutive patients. *Radiology* 2007;243(3):820–7 [Epub 2007/04/14].
- [40] Scholtes F, Adriaensens P, Storme L, Buss A, Kakulas BA, Gelan J, et al. Correlation of postmortem 9.4 tesla magnetic resonance imaging and immunohistopathology of the human thoracic spinal cord 7 months after traumatic cervical spine injury. *Neurosurgery* 2006;59(3):671–8 [Discussion 8. Epub 2006/09/07].
- [41] Collignon F, Martin D, Lenelle J, Stevenaert A. Acute traumatic central cord syndrome: magnetic resonance imaging and clinical observations. *J Neurosurg* 2002;96(1 Suppl.):29–33 [Epub 2002/01/25].
- [42] Schaefer DM, Flanders A, Northrup BE, Doan HT, Osterholm JL. Magnetic resonance imaging of acute cervical spine trauma. Correlation with severity of neurologic injury. *Spine* 1989;14(10):1090–5 [Epub 1989/10/01].
- [43] Song J, Mizuno J, Inoue T, Nakagawa H. Clinical evaluation of traumatic central cord syndrome: emphasis on clinical significance of prevertebral hyperintensity, cord compression, and intramedullary high-signal intensity on magnetic resonance imaging. *Surg Neurol* 2006;65(2):117–23 [Epub 2006/01/24].
- [44] Petersen JA, Wilm BJ, von Meyenburg J, Schubert M, Seifert B, Najafi Y, et al. Chronic cervical spinal cord injury: DTI correlates with clinical and electrophysiological measures. *J Neurotrauma* 2012;29(8):1556–66 [Epub 2011/12/14].
- [45] Cheran S, Shanmuganathan K, Zhuo J, Mirvis SE, Aarabi B, Alexander MT, et al. Correlation of MR diffusion tensor imaging parameters with ASIA motor scores in hemorrhagic and nonhemorrhagic acute spinal cord injury. *J Neurotrauma* 2011;28(9):1881–92 [Epub 2011/08/31].

- [46] Chang Y, Jung TD, Yoo DS, Hyun JK. Diffusion tensor imaging and fiber tractography of patients with cervical spinal cord injury. *J Neurotrauma* 2010;27(11):2033–40 [Epub 2010/09/09].
- [47] Wang B, Liu H, Wang H, Zhou D. Segmental instability in cervical spondylotic myelopathy with severe disc degeneration. *Spine* 2006;31(12):1327–31 [Epub 2006/05/25].
- [48] White 3rd AA, Johnson RM, Panjabi MM, Southwick WO. Biomechanical analysis of clinical stability in the cervical spine. *Clin Orthop Relat Res* 1975;109(109):85–96 [Epub 1975/01/11].
- [49] Roy-Camille R, Saillant G, Berteaux D, Bissierie M. Severe strains of the cervical spine operated on by a posterior approach (author's transl). *Rev Chir Orthop Reparatrice Appar Mot* 1978;64(8):677–84 [Epub 1978/12/01. Entorses graves du rachis cervicale. Traitement par voie posterieure].
- [50] Boakye M, Harkema S, Ellaway PH, Skelly AC. Quantitative testing in spinal cord injury: overview of reliability and predictive validity. *J Neurosurg Spine* 2012;17(1 Suppl.):141–50 [Epub 2012/09/19].
- [51] Ellaway PH, Kuppuswamy A, Balasubramaniam AV, Maksimovic R, Gall A, Craggs MD, et al. Development of quantitative and sensitive assessments of physiological and functional outcome during recovery from spinal cord injury: a clinical initiative. *Brain Res Bull* 2011;84(4–5):343–57 [Epub 2010/08/24].
- [52] Curt A, Ellaway PH. Clinical neurophysiology in the prognosis and monitoring of traumatic spinal cord injury. *Handb Clin Neurol* 2012;109:63–75 [Epub 2012/10/27].
- [53] Alexander MS, Anderson KD, Biering-Sorensen F, Blight AR, Brannon R, Bryce TN, et al. Outcome measures in spinal cord injury: recent assessments and recommendations for future directions. *Spinal Cord* 2009;47(8):582–91 [Epub 2009/04/22].
- [54] Vale FL, Burns J, Jackson AB, Hadley MN. Combined medical and surgical treatment after acute spinal cord injury: results of a prospective pilot study to assess the merits of aggressive medical resuscitation and blood pressure management. *J Neurosurg* 1997;87(2):239–46 [Epub 1997/08/01].
- [55] Dahdaleh NS, Lawton CD, El Ahmadih TY, Nixon AT, El Tecle NE, Oh S, et al. Evidence-based management of central cord syndrome. *Neurosurg Focus* 2013;35(1):E6 [Epub 2013/07/03].
- [56] Nicholson BD. Evaluation and treatment of central pain syndromes. *Neurology* 2004;62(5 Suppl. 2):S30–6 [Epub 2004/03/10].
- [57] Gruenthal M, Mueller M, Olson WL, Priebe MM, Sherwood AM, Olson WH. Gabapentin for the treatment of spasticity in patients with spinal cord injury. *Spinal Cord* 1997;35(10):686–9 [Epub 1997/11/05].
- [58] Chang YJ, Liang JN, Hsu MJ, Lien HY, Fang CY, Lin CH. Effects of continuous passive motion on reversing the adapted spinal circuit in humans with chronic spinal cord injury. *Arch Phys Med Rehabil* 2013;94(5):822–8, <http://dx.doi.org/10.1016/j.apmr.2012.11.035> [Epub 2012 Dec 3].
- [59] Tai Q, Kirshblum S, Chen B, Millis S, Johnston M, DeLisa JA. Gabapentin in the treatment of neuropathic pain after spinal cord injury: a prospective, randomized, double-blind, crossover trial. *J Spinal Cord Med* 2002;25(2):100–5 [Epub 2002/07/26].
- [60] Duh MS, Shepard MJ, Wilberger JE, Bracken MB. The effectiveness of surgery on the treatment of acute spinal cord injury and its relation to pharmacological treatment. *Neurosurgery* 1994;35(2):240–8 [Discussion 8–9. Epub 1994/08/01].
- [61] Laus M, Pignatti G, Tigani D, Alfonso C, Giunti A. Anterior decompression and plate fixation in fracture dislocations of the lower cervical spine. *Eur Spine J* 1993;2(2):82–8 [Epub 1993/08/01].
- [62] Edwards 2nd CC, Heller JG, Murakami H. Corpectomy versus laminoplasty for multilevel cervical myelopathy: an independent matched-cohort analysis. *Spine* 2002;27(11):1168–75 [Epub 2002/06/05].
- [63] Dai L, Jia L. Central cord injury complicating acute cervical disc herniation in trauma. *Spine* 2000;25(3):331–5 [Discussion 6. Epub 2000/03/07].
- [64] DiAngelo DJ, Foley KT, Vossel KA, Rampersaud YR, Jansen TH. Anterior cervical plating reverses load transfer through multilevel strut-grafts. *Spine* 2000;25(7):783–95 [Epub 2000/04/06].
- [65] Mummaneni PV, Haid RW, Rodts Jr GE. Combined ventral and dorsal surgery for myelopathy and myeloradiculopathy. *Neurosurgery* 2007;60(1 Suppl. 1):S82–9 [Epub 2007/01/06].
- [66] Lei T, Shen Y, Wang LF, Cao JM, Ding WY, Ma QH. Cerebrospinal fluid leakage during anterior approach cervical spine surgery for severe ossification of the posterior longitudinal ligament: prevention and treatment. *Orthop Surg* 2012;4(4):247–52 [Epub 2012/10/31].
- [67] Li H, Dai LY. A systematic review of complications in cervical spine surgery for ossification of the posterior longitudinal ligament. *Spine J* 2011;11(11):1049–57 [Epub 2011/10/22].
- [68] Mummaneni PV, Kaiser MG, Matz PG, Anderson PA, Groff MW, Heary RF, et al. Cervical surgical techniques for the treatment of cervical spondylotic myelopathy. *J Neurosurg Spine* 2009;11(2):130–41 [Epub 2009/09/23].
- [69] Cabraja M, Oezdemir S, Koeppen D, Kroppenstedt S. Anterior cervical discectomy and fusion: comparison of titanium and polyetheretherketone cages. *BMC Musculoskelet Disord* 2012;13:172 [Epub 2012/09/18].
- [70] Cabraja M, Abbushi A, Koeppen D, Kroppenstedt S, Woiciechowsky C. Comparison between anterior and posterior decompression with instrumentation for cervical spondylotic myelopathy: sagittal alignment and clinical outcome. *Neurosurg Focus* 2010;28(3):E15 [Epub 2010/03/03].
- [71] Wada E, Suzuki S, Kanazawa A, Matsuoka T, Miyamoto S, Yonenobu K. Subtotal corpectomy versus laminoplasty for multilevel cervical spondylotic myelopathy: a long-term follow-up study over 10 years. *Spine* 2001;26(13):1443–7 [Discussion 8. Epub 2001/07/18].
- [72] Hirai T, Okawa A, Arai Y, Takahashi M, Kawabata S, Kato T, et al. Middle-term results of a prospective comparative study of anterior decompression with fusion and posterior decompression with laminoplasty for the treatment of cervical spondylotic myelopathy. *Spine* 2011;36(23):1940–7 [Epub 2011/02/04].
- [73] Liu T, Xu W, Cheng T, Yang HL. Anterior versus posterior surgery for multilevel cervical myelopathy, which one is better? A systematic review. *Eur Spine J* 2011;20(2):224–35 [Epub 2010/06/29].
- [74] Riew KD, Sethi NS, Devney J, Goette K, Choi K. Complications of buttress plate stabilization of cervical corpectomy. *Spine* 1999;24(22):2404–10 [Epub 1999/12/10].
- [75] Dahdaleh NS, Lawton CD, El Ahmadih TY, Nixon AT, El Tecle NE, Sanders OH, et al. Evidence-based management of central cord syndrome. *Neurosurgical Focus* 2013;35(1):E6 [Online publication date: 1-Jul-2013].