Neurological recovery after cervical cord decompression for canal stenosis myelopathy

H. Pascal-Moussellard [1], L.-R. Despeignes [1], S. Olindo [1], J.-L. Rouvillain [1], Y. Catonné [1]

[2] Service d’Orthopédie 2C, CHU La Meynard, Fort-de-France, Martinique.
[3] Service de Neurologie 6C, CHU La Meynard, Fort-de-France, Martinique.

ABSTRACT

Purpose of the study
Progressive myelopathy secondary to stenosis of the spinal canal is generally treated by surgery. Results of surgical decompression are generally good but the pattern of neurological recovery has not been studied. We followed a cohort of patients who underwent cervical cord decompression to study the course of neurological recovery.

Material and methods
The study cohort included 39 patients (22 men and 17 women), with a mean age 65.7 years, who underwent surgery between 1998 and 2002 for progressive cervical myelopathy. The same surgeon performed all procedures (23 posterior and 16 anterior approaches). The JOA score and MRI findings were noted. The patients were seen at 1, 3, 6, 12, and 18 months and then annually (JOA score). The Hirabayashi score was used to assess neurological recovery. Two populations were identified (group 1: preoperative JOA score >6, group 2: preoperative JOA score ≤6).

Results
The mean preoperative JOA score was 8.3/17; range, 1–15. Ten patients had a severe JOA score (<6). The mean postoperative JOA score was 13.3 (range, 3–17) at 6 months and remained stable during follow-up. Neurological recovery as assessed with the Hirabayashi technique was 52.5% on average at last follow-up. Neurological gain occurred mostly during the 1st to 3rd months following decompression and remained stable thereafter. Patients with a severe deficit showed the same recovery pattern but stabilized at a lower neurological level. Expressed in JOA points, neurological gain was very similar in the two groups. There was no significant difference between patients who underwent anterior or posterior procedures.

Discussion and conclusion
The pattern of neurological recovery in patients with degenerative cervical disease appears to be rapid during the first 6 months following surgical decompression. The level of recovery then stabilizes, irrespective of the severity of the initial deficit. This study demonstrated that more specific evaluation scales than the JOA score should be developed for assessment and follow-up of these patients.

Key words: Myelopathy, cervical cord decompression, spondylosis.

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INTRODUCTION

The narrowness of the cervical canal can be responsible for cervical cord pain. This narrowness can be primary, related to congenital narrowing of the cervical canal (congenital narrow cervical canal) or secondary. In this case, it is most often of arthritic origin (cervical spondylotic myelopathy). Although many studies have evaluated the quality of neurological recovery after decompression surgery or its stabilization over time [Matsuda et al. (1), Kimura et al. (2), Fouyas et al. (3), Kadanka et al. (4)], we found no reports in the literature describing the pattern of this recovery. The authors report the results of a prospective study investigating 39 patients operated on between 1998 and 2002 for progressive cervical cord myelopathy. This study aimed to assess the speed of neurological recovery, with particular attention paid to severe myelopathies.

MATERIAL AND METHODS

All patients were assessed using a standardized neurological examination designed to identify cervical cord pain. They were evaluated using the Japanese Orthopaedic Association (JOA) score (Table I) [Mazel et al. (5)]. Changes were brought to the subjective information on clumsiness of the upper extremities so that this score could be adapted to Western cultures (Table I).

In all cases, the radiographic workup included frontal and lateral conventional x-rays of the cervical spine, the basis for the Torg index calculations (6); the impairment was considered pathological when below 0.8. The Torg index represents the relation between the anterior-posterior diameter of the cervical canal by the anterior-posterior diameter of the body (measured at its mean point), at a given level.

All patients were examined by MRI, confirming canal narrowness. This examination provided objective evidence of the disappearance of the perimedullary safety margin corresponding to the presence of cerebrospinal fluid (CSF) around the spinal cord and the possible deformation of the spinal cord pressed against the vertebral body in front and the lamina behind. MRI provided visualization of the possible existence of an intramedullary hypersignal in a T2-weighted sequence, a sign of cervical cord pain.

Patients were operated on for an association of cervical cord pain symptoms and MRI diagnosis of cervical canal stenosis. There were nine cases of congenital narrow cervical canal and most often cervical canal spondylosis or a combination of the two (arthritic decompensation of a congenital narrow canal).

Patients

The 39 patients were operated on consecutively between 1998 and 2002 by the same surgeon: 22 males (56.4%) and 17 females (43.6%), with a mean age of 65.7 years (range, 38–86 years).

<table>
<thead>
<tr>
<th>I. Motor dysfunction for the upper extremities</th>
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<tbody>
<tr>
<td>0: Unable to feed oneself</td>
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<tr>
<td>1: Unable to use chopsticks but able to eat with a spoon → Unable to cut meat</td>
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<tr>
<td>2: Able to use chopsticks with great difficulty → Able to eat alone with great difficulty</td>
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<tr>
<td>3: Able to use chopsticks with slight difficulty → Able to eat alone with slight difficulty</td>
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<tr>
<td>4: No dysfunction</td>
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<th>II. Motor dysfunction for the lower extremities</th>
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<tbody>
<tr>
<td>0: Unable to walk</td>
</tr>
<tr>
<td>1: Able to walk on flat floor with a walking aid</td>
</tr>
<tr>
<td>2: Able to walk up or down stairs with handrail</td>
</tr>
<tr>
<td>3: Lack of stability</td>
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<tr>
<td>4: No dysfunction</td>
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<th>III. Sensory dysfunction</th>
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<tbody>
<tr>
<td>A: Upper extremities</td>
</tr>
<tr>
<td>0: Severe sensory loss or pain</td>
</tr>
<tr>
<td>1: Moderate sensory loss</td>
</tr>
<tr>
<td>2: No sensory loss</td>
</tr>
<tr>
<td>B. Lower extremities: identical to A</td>
</tr>
<tr>
<td>C. Trunk: identical to A</td>
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<th>IV. Sphincter dysfunction</th>
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<td>0: Unable to micturate voluntarily</td>
</tr>
<tr>
<td>1: Able to micturate with difficulty (retention)</td>
</tr>
<tr>
<td>2: Able to micturate with moderate difficulty (pollakiuria)</td>
</tr>
<tr>
<td>3: Normal micturition</td>
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The usual clinical picture associating, more or less significantly, paresthesias of the hands as well as gait and sphincter problems were the most frequently observed. Pyramidal syndrome was found in all cases on clinical examination.

Particular clinical forms were found, however:
- Two patients presented symptoms of muscular atrophy, predominating in the upper limbs. The diagnosis of
Amyotrophic lateral sclerosis had been ruled out by examination by a neurologist:

- One patient presented isolated impaired urinary sphincter control associated with brisk reflexes at clinical examination.

Ten patients presented a preoperative JOA score of 6 or lower. This score reflects a very severe tetraparesis corresponding to a total loss of autonomy. These patients were operated on because of the deterioration of their symptoms during hospitalization, leading to bedridden invalidity.

Ten patients presented an associated narrow lumbar spinal canal.

The Torg index was pathological by at least one level in 100% of the cases.

Fig. 1. – X-rays after frontal (a) and lateral (b) corporectomy.

Fig. 2. – MRI after corporectomy. a) Sagittal T2-weighted image. b) axial T2-weighted image.
MRI demonstrated the cervical cord pain as an intramedullary hypersignal in T2-weighted images in 16 cases (41%). Cervical cord decompression was performed by the anterior or posterior approach depending on the seat of the compression (anterior or posterior), the number of levels to decompress (the posterior approach was preferred when the decompression concerned more than two contiguous levels), and the existence of spinal balance impairment (kyphosis, instability). Anterior approach decompressions (16 patients) required corporectomy (one level 13 times, two levels twice) or discectomy (once at C7–T1). The corporectomy involved C4 eight times, C5 eight times, and C6 once. An autologous iliac graft associated with osteosynthesis with a screw-plate was systematically done (Figs. 1a, 1b, 2a and 2b). Posterior decompressions consisted of laminectomy or laminoplasty. Laminoplasty was carried out by making a bilateral trough at the lamina–articular process junction, then opening the posterior arch along the median line. Twenty-three patients were operated on via the posterior approach: 21 laminoplasties (Fig. 3a–c) extending from C3 to C7 and two segment laminectomies of two levels. Postoperative drainage was set up following both approaches.

Patients were monitored systematically at 1, 3, 6, 12, and 18 months and then every year after surgery and were evaluated using the JOA score. Neurological recovery was estimated by the recovery rate defined by Hirabayashi et al. (7), calculated according to the formula: 

$$\text{Recovery rate} = \left( \frac{\text{postoperative JOA} - \text{preoperative JOA}}{17 - \text{preoperative JOA}} \right) \times 100.$$ 

None of the patients were lost to follow-up, lasting a mean of 14 months (range, 9–65 months). Two patients died in the year following surgery, one of whom presented severe preoperative tetraparesis (JOA = 2/17).

RESULTS

Early complications

A suffocating hematoma in a patient operated on via the anterior approach required immediate attention. A hematorrhachis responsible for neurological aggravation after laminoplasty in a hypertensive patient required emergency reoperation, which resulted in rapid recovery of the initial

**Fig. 3.** – MRI after laminoplasty. a) sagittal T2-weighted image. b) Axial T2-weighted image.

**Fig. 4.** – Progression of JOA score over 24 months.
neurological deficit. Both patients who underwent segment laminectomy presented secondary hyperpathia of the upper limbs, which did not require reoperation.

**JOA score over time**

Preoperatively, the mean JOA score was 8.3/17, ranging from 1 to 15. The mean postoperative JOA score averaged at 13.3 (range, 3–17) between the first and third months after surgery, then leveled off until 2 years after surgery (Fig. 4).

The mean neurological recovery rate, calculated using Hirabayashi’s technique, was 52.5% at the end of follow-up. Only one patient’s condition worsened at the end of follow-up, at 2 years and 4 months (going from preoperative JOA of 13 to 9 at the end of follow-up). This deterioration was for the most part related to problems with walking attributable to decompensation of a severe narrow lumbar canal associated with cervical stenosis. This patient’s JOA score was 14/17 at the 1st postoperative month, then 12/17 at the 3rd and 6th months, slowly deteriorating to 9 at 2 years of follow-up. This 79-year-old patient did not wish to undergo surgery for the narrow lumbar canal. Excluding this patient, the mean neurological recovery rate was 57.5%.

**Neurological recovery in relation to the severity of the initial deficit (Fig. 2)**

We compared the neurological recovery obtained after decompression in patients with a preoperative JOA score higher than 6 (29 patients) (group 1) and those presenting a preoperative JOA score lower than 6 (10 patients) (group 2).

In group 1, the preoperative JOA score was 10.5 and reached 14.8 at 1 year after surgery. The mean gain was 4 points on the JOA scale and the neurological recovery rate was 60%. The curve representing JOA score progression over time (Fig. 5) showed that the mean score went from 10.5 before operation to 13.9 between month 1 and month 3, then only from 14.3 to 14.8 between month 6 and 1 year after surgery. Therefore, the neurological gain was accomplished between the 1st and 3rd postoperative months and leveled off to only progress slightly beyond that point.

Ten patients (25.6%) presented a preoperative JOA score below 6 (group 2). This impairment, predominately on motor items (upper and lower limbs) of the JOA score, reflected the bedridden state in these patients. This neurological deficit with progressive loss of autonomy was the motivating factor for hospitalization and in these patients operated on for the deterioration of their neurological status during hospitalization.

The mean preoperative JOA score of these patients varied from 3.4 (range, 1–6) for a mean postoperative score of 9.6 (range, 3–16) at 1 year after surgery. The mean gain was 6.2 on the JOA scale. The neurological gain was 45.6%.

The study of the course of the neurological recovery over time of these patients shows (as for the patients with a preoperative JOA score over 6) that the recovery took place between the 1st and 3rd postoperative months (mean JOA = 10.2 at 1 month and 10.3 at 3 months). The result leveled off beyond the 3rd month after surgery (Fig. 5) (mean JOA = 9.8 at 6 months and 9.6 at 12 months).

**Comparison between anterior and posterior approach (Fig. 6)**

These two populations were comparable in our series in terms of age, sex, and severity of neurological deficit (preoperative JOA). There was no significant difference in terms of neurological recovery (postoperative JOA and neurological recovery rate) between patients operated on by the anterior approach and those treated by the posterior approach.
approach. In addition, the curves depicting progression over time of mean neurological recovery in these two populations are nearly superimposable (Fig. 6).

**DISCUSSION**

**Progression of neurological recovery**

Many publications have evaluated the more or less long-term results of cervical cord decompression surgery in myelopathy [Fager (8), Gorter (9), Epstein *et al.* (10), Herkowitz (11), Matsunaga *et al.* (12), Satomi *et al.* (13), Kohno *et al.* (14), Emery *et al.* (15)]. We found no studies evaluating the speed of this recovery. Seichi *et al.* (16) showed that the JOA score remains stable after 1 year postoperative, and deteriorates slightly after 10 years. For Matsuda *et al.* (1), the JOA score can deteriorate beyond 5 years after surgery in elderly patients.

The overall analysis of our series showed an initially rapid neurological recovery during the first 3 months after surgery. Subsequently, the result obtained at 3 months after surgery only improved very slightly, with the curve nearly leveled off, with patients progressing little beyond the 6th month. This recovery process describes an analogue curve, whatever the severity of the initial deficit. On average, patients presenting a very severe initial deficit show the same recovery process as patients with less severe impairment.

Therefore, the fact that neurological recovery after decompression surgery that begins early and rapidly progresses during the first 3 months after surgery should be motivation to set up a specific multidisciplinary neurologi-
Secondary hyperpathia

Secondary hyperpathia can be attributed to a conflict between the cranial extremity of the lamina underlying the laminectomized levels and the posterior spinal cords.

This explanation means establishing a preoperative plan that takes into account not only the levels to decompress, but also the balance of the cervical spinal column [Suda et al. (20), Guigui et al. (21), Sodeyama et al. (22)]. The preoperative plan should not only consider the potential pre- and postoperative instabilities of the cervical spine [Guigui et al. (21)], but also plan for the postoperative position of the spinal cord after decompression. Thus, in decompressions treated by the posterior approach, the displacement of the spinal cord will be greater as a function of how much the cervical spine is in lordosis. Laminectomy should therefore involve the laminae that may conflict with the spinal cord during its displacement [Sodeyama et al. (22)]. This displacement of the spinal cord has also been questioned in the etiology of radicular involvement, by the effect of traction on the nerve roots, in particular C5, after decompression via the posterior approach [Chiba et al. (23)].

CONCLUSION

Recovery from cervical spondylitic neurological deficit occurs rapidly following decompression surgery and stabilizes thereafter. Rehabilitation should begin early to optimize this neurological recovery. Severe deficit seems to follow the same process and levels off at a lower neurological level. Exploration and follow-up of these patients requires refining more specific assessment scales than the JOA score, using quantifiable and reliable criteria. For now, evaluation of neurological recovery should preferentially take into account the absolute JOA score rather than the Hirabayashi neurological recovery rate, which is poorly adapted to evaluating patient progression.

References


