

# Value of early attention to spinal compression syndromes

In a consecutive series of 125 patients with spinal compression syndromes, neurological regression was evaluated in relation to the time interval from (a) initial symptoms (patient delay), (b) diagnostic recognition (diagnostic delay), and (c) decision on treatment (therapeutic delay) until the time of definitive treatment. Patients with benign lesions showed a statistically significant negative correlation between the duration of (a) + (b) + (c) and the therapeutic result. In cancer patients there was a tendency for rapid progression of the disease to give a poorer result than in patients in whom the course was more insidious. In all, 77 patients improved after a short therapeutic delay (14 h), while in the remaining 48 the status was unchanged or worse after a long delay (34 h); this is a highly significant difference.

*Key words:* cauda equina syndrome; paraplegia; spinal cord; tetraplegia, treatment.

Compression of the spinal cord and cauda equina always involves a serious risk of greatly disabling sequelae. The reported prognosis differs widely, depending on etiology, but mainly on the failure to make a correct diagnosis before the compression syndrome is suddenly a reality (Andersen 1975, Dahlström et al. 1979, Gorter 1978, Hattori 1976, Laasonen et al. 1979, Livingston & Perrin 1978, Nather & Bose 1982, Spännare 1978, Törmä 1957). Presumably, the prognosis can be decisively improved by starting treatment before the patients have become paralysed. Several authors (Dahlström et al. 1979, Livingston & Perrin 1978) have reported more than 1 year's delay after the appearance of solitary, unfortunately most often not particularly alarming symptoms, until massive paralysis sets in. The causes of this therapeutic delay and deficient diagnosis have not been thoroughly elucidated. Diagnostic doubts may be due to the rare occurrence of the condition (Gorter 1978), but also to a widely different etiology with varying symptoms and signs, e.g. trauma, tumors, metastases, infection, infarcts, and degenerative diseases. The influence of therapeutic delay in non-traumatic spinal compression syndrome was analyzed with a view to irreversible changes; the causes of such therapeutic delay are discussed.

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## Patients and methods

From January 1975 to December 1979, a consecutive series of 125 patients with spinal compression syndromes of different severity and etiology (Table 1) was treated in our Department. Seventy-two patients suffered from malignant diseases (44 men, 28 women), ranging in age from 19 to 78 (median 51) years. Benign etiology was found in 53 patients (26 men, 27

Table 1. Type of lesion with and without bone involvement

Group	Type of lesion	Number	+/- bone involvement
Malignant	Metastases (lung 14, breast 7, colon 7, prostate 6, hypernephroma 4, other 10)	48	62/10
	Lymphoreticulosis	22	
	Astrocytoma	2	
Benign	Spondylosis	16	16/37
	Herniated disc	8	
	Myelitis	6	
	Neurinoma	6	
	Meningeoma	7	
	Other intradural lesions (ependymoma, epidermoid, teratoma, abscess)	10	
	Total	125	

Table 2. Percentage of symptoms at onset of the spinal compression syndrome (in per cent)

	Malignant		Benign	
	+ bone involv.	- bone involv.	+ bone involv.	- bone involv.
Pain	70	80	31	47
Paresis	34	30	56	23
Sphincter dysfunction	18	10	6	9
Sensory loss	52	57	17	46

women) with 57 years as the median age (range 6–89 years). In the malignant group metastases, Hodgkin's disease, and myelomatosis predominated. Clinical and radiographic signs of bone involvement were found in 62 patients. Most of the patients with benign diseases had intraspinal affections of soft tissues, and only patients with spondylosis had bony involvement. Eight patients had cauda equina syndrome following following disc herniation.

Eleven patients of the total series had infranuclear 2nd neuron palsy.

Bony involvement was recorded to ascertain whether treatment was instituted sooner if the primary roentgenographs showed abnormal bone structure.

The median follow-up period was 3.9 (1–5.8) years. At the time of follow-up 64 patients had died, but detailed information could be obtained from their case records. All 61 surviving patients were examined by the authors. Treatment consisted of decompressive laminectomy in 96 patients with myelographic block and in partial excision (40 patients) or total excision (31 patients) of tumor in 89 patients with tumors. Conservative treatment was applied in 29 cases, 18 of whom were cancer patients in a poor general condition, and the remaining 11 had various degenerative and infectious diseases. The distribution of the four most important initial symptoms was analyzed to elucidate their influence in instituting treatment (Table 2). The causes of treatment delay were divided into three types: (a) *Patient delay* was the interval from the appearance of the initial signs of spinal compression until the first medical contact. (b) *Diagnostic delay* was the interval from the first medical contact until the development of the spinal compression syndrome. (c) *Therapeutic delay* was the interval from the time when the compression syndrome was diagnosed until treatment was instituted – and this included the general slowness of the hospital system, i.e. the time which invariably elapses after admission, even in cases where a correct diagnosis has been made.

The degree of neurological deficit was assessed by a

slight modification of Frankel's classification system developed from Stoke-Mandeville (Frankel et al. 1969), group A (= total paralysis and sensory loss) being assessed at 0 point, increasing by 25 point-intervals to group E (full recovery), which means 100 points. Non-parametric statistical analysis was performed, with a 5 per cent level of significance.

## Results

In patients with malignant disease, pain was more often an initial symptom than in patients with benign disease. In patients with benign bone involvement (i.e. spondylosis) initial sensory loss was significantly less frequent than in the other groups. A clear-cut sensory level was present more often in cancer patients with bone lesions, but the sensory loss was on the whole correlated well to the grade of impairment of motor function in each group. Other alarming symptoms – paralysis and sphincter disturbances – were equally distributed among the groups.

In patients with spondylosis an average of 6 weeks of progressing symptoms and signs had elapsed before they consulted a doctor, and thus they had a significantly longer patient delay than the other groups (Table 3). Diagnostic delay was significantly longer also among the patients with spondylosis than among patients with primary cancer or disc herniation. Therapeutic delay was only significantly longer among patients with metastases.

Patients exhibiting evident abnormalities on plain films of the spine had a significantly longer therapeutic delay than patients with

Table 3. Delay of treatment (in days)

	Patient's delay	Diagnostic delay	Therapeut. delay	Total delay
<b>Malignant</b>				
Metastases	11	8	6*	25
Lymphoretic.	8	10	1	19
Astrocytoma	9	17	1/4	26
<b>Benign</b>				
Spondylosis	42*	70*	1	113*
Disc	1	13	3/4	15
Tumor	5	17	2	24
Other	3	34	1	38

\*  $p < 0.05$ .

Table 4. The significance of bone involvement for the therapeutic delay

	Delay in days	
	Median	Range
+ Bone involvement	3	1/6-10
- Bone involvement	1/2*	1/4-26

\*  $p < 0.05$ .

normal radiography of the spine (Table 4). Even patients with marked destructions of the pedicles were exposed to therapeutic delay.

Figure 1 sets out the correlation between the treatment delay and the neurological regression in terms of an increased Frankel score. Only patients in the benign group showed a significant correlation which was negative. In cancer patients there was a tendency for rapid progression to give a poorer result than an insidious course.

In patients who improved, the therapeutic delay was highly significantly briefer than in patients whose status was unchanged or worse (Table 5).

Of the cancer group 46/58 patients remained paralysed versus only 2/10 of the patients with benign conditions.

In the total material, there was no difference in neurological improvement between patients

Table 5. The results of treatment and the therapeutic delay

	Therapeutic delay in hours	No.
Improved	14	77
Unchanged/worse	34*	48

\*  $p < 0.01$ .

with 1st and 2nd neuron paralysis, the improvement being 71/114 and 6/11, respectively.

## Discussion

Of the cancer patients in the present material, 70–80 per cent had pain as the initial symptom, corresponding to the incidence reported in several series (Gilbert et al. 1978, Livingston & Perrin 1978, Nather & Bose 1982, Young et al. 1980). Other alarming initial symptoms were equally distributed in the groups, so that the difference in therapeutic delay cannot be explained on the basis of a different initial symptom. Patients with spondylosis had the longest total delay, in conformity with experimental studies which have shown that slow extradural compression does not result in a symptomatic decrease in the arterial oxygen saturation of the cord until about half of the spinal canal has become occluded (Hukuda & Amano 1980). In an experimental study of cervical spondylotic myelopathy, spinal cord blood flow was measured using isotope labeled microspheres (Nakamura et al. 1981). In delayed paralysis animals, spinal cord flow was within normal limits. In cats with acute spinal cord compression, the blood flow decreased significantly 15 min after spinal cord compression was induced.

In a study on the blood flow in the dorso-lateral funiculus of traumatized thoracic spinal cords in cats, Senter & Venes (1978) found that ischemia arose on a level with the injury, but that posttraumatically there was an interval of more than 1 h during which the blood supply was preserved. Therefore, early treatment of spinal cord compression seems rational and is recommended by most authors (Boland et al. 1982, Choudhury & Taylor 1980, Dahlström

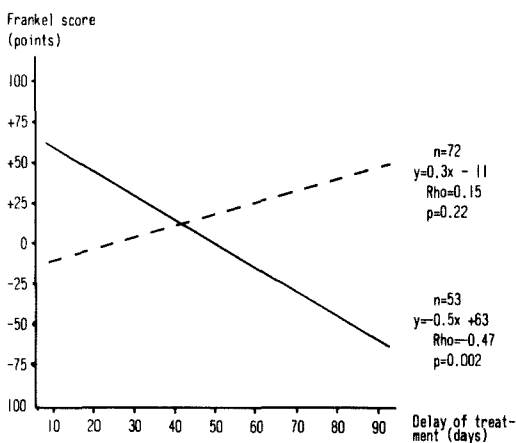


Figure 1. Paired values of delay of treatment and neurological regression in terms of a change in Frankel score. + = improvement. - = deterioration. The lines were determined by linear regression analysis. ---- Malignant. — Benign.

et al. 1979, Gorter 1978, Hattori 1976, Nather & Bose 1982). Indeed, the best results in the present material were found in patients treated at an early stage of the course.

It would be reasonable to imagine that patients with evidently abnormal findings on plain X-rays of the spine would be referred for treatment earlier when the use of their limbs started to fail. However, this was not so, as such patients had a therapeutic delay five times that of patients with normal radiographs of the spine. Surprisingly, the longest therapeutic delay was found in patients with painful, malignant bony lesions. As analysis of the admission procedure did not disclose a reluctance to treat cancer patients, deficient diagnostic recognition is the only explanation for therapeutic delay.

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