

FACTORS INFLUENCING NEUROLOGICAL RECOVERY IN BURST THORACOLUMBAR FRACTURES

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The association between the thoracolumbar vertebrae fracture pattern, treatment and neurological recovery was estimated. Sixty-three patients with burst fractures at the T11 to L2 vertebral level and associated neurological deficit were evaluated by plain roentgenograms, CT scan and a quantitative neurological examination. The parameters used were percent canal compromise, location of the retropulsed middle column fragment, kyphosis, type of treatment, and neurological recovery. The follow-up varied from 24 to 84 months (mean 44 months). Treatment was conservative in 15 patients and surgical in 48 patients. Posterolateral decompression was carried out in 26 patients.

The severity of the initial paralysis did not correlate with the initial fracture pattern except perhaps for Frankel A cases. Neurological recovery did correlate with the initial kyphosis but not with the amount of canal compromise or the location of the middle column fragment. Neurological recovery did not correlate with decompression. Improvement of paralysis was associated with restoration of the sagittal spine alignment. From the patients with greater than 5° correction of kyphosis the majority improved neurologically. If the correction of the kyphosis was less than 5° the recovery was poor regardless of the method used.

We assume that the initial paralysis in burst fractures with severe kyphosis is partially caused by permanent cord or root damage and partially by neuroapraxia from angulation of the neural structures and their vessels. With reduction of the fracture and correction of the kyphotic deformity, spinal cord, roots, and their vessels become lax, and the chances for neurological recovery increase significantly.

Keywords : thoracolumbar fractures ; kyphosis.

Mots-clés : fractures thoraco-lombaires ; cyphose.

INTRODUCTION

Neurological injury has been reported to occur in 30 to 90% of patients with burst thoracolumbar fractures (2, 4, 15, 27). Multiple studies have examined clinical recovery patterns with paralysis secondary to this type of fracture in general (1, 2, 5, 9, 16, 17, 23, 25, 26). Neurological recovery varied from 50 to 90% in these studies, with either conservative (1, 5, 15, 18, 19) or surgical (9, 12, 13, 14, 21, 23) methods or treatment. However most of these studies 1) look at improvement of paralysis only as a subgroup in a study of various spinal injuries, 2) utilize only one treatment plan, 3) fail to separate the value of stabilization from the value of decompression, 4) evaluate all thoracolumbar fractures and thoracolumbar levels as one, and 5) fail to look at parameters related to the initial alteration of the vertebral anatomy. This paper assesses the parameters influencing neuro-

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logical recovery in a patient population having a specific thoracolumbar fracture pattern. A protocol was constructed to correlate neurological deficit and recovery with specific fracture pattern and treatment on the basis of spinal canal compromise and alteration of the sagittal spine configuration.

PATIENTS AND METHODS

Between 1981 and 1991, 195 patients with burst fractures of the thoracic and lumbar spine were admitted to Athens General Hospital. One hundred and twenty-two of these patients had sustained a fracture, or had their most severe fracture in cases of multiple vertebral fractures, at a level between T11 and L2. Of these 122 fractures 63 (51.6%) were associated with neurological injury. Of these injuries 20 (16.3%) were neurologically complete and 43 (35.2%) incomplete. There were 39 male and 24 female patients aged from 16 to 72 years (mean age 40.7 years) (table I).

The criteria for inclusion in the series were 1) a burst fracture as defined by Denis (7), 2) complete or incomplete paralysis of the cord or the cauda equina or both and, 3) greater than 10% spinal compromise by the retropulsed vertebral middle column or disc fragment. The type of treatment was as follows: Conservative (postural reduction-bracing) in 15 patients, distraction rod instrumentation (Harrington) and fusion in 29 patients, distraction rod instrumentation with segmental wiring (Harrington-Luque) and fusion in 11 patients, and rectangular rod segmental wiring (Hartshill) and fusion in 8 patients. Posterolateral decompression of the neural structures from the retropulsed vertebral body and disc fragments was carried out in 26 patients. Each patient was followed at specific intervals for at least 2 years postoperatively. The follow-up varied from 24 to 84 months (mean 44 months). All patients had adequate data in the preoperative evaluation. In order to determine the fracture pattern three variables were evaluated. The first of them was the percentage of spinal compromise. The second was whether the maximum canal compromise occurred at the level of the ligamentum flavum or where the bone encircles the canal. The third variable was the degree of kyphosis. Additional variables such as the type of treatment, including decompression, were also tested statistically. The kyphosis was measured as the Cobb angle between the intact vertebrae above and below the fractured ones. The maximum canal compromise was measured and compared to the normal diameter of the spinal canal at the level above the fracture, and a percentage of

canal stenosis was estimated. The questions asked were: Does initial paralysis correlate with the three fracture pattern variables? Does neurological recovery correlate with the variables? Does neurological recovery correlate with the type of treatment including both decompression and restoration of the sagittal spine configuration?

Since only few patients underwent postoperative CT scans to study the vertebral canal, no attempt was made to find out whether "blind" decompression by realignment gave adequate anatomic decompression.

RESULTS

To test whether a significant association existed between the variables, we used two appropriate methods. More specifically if one variable was measured on a nominal scale (i.e. attribute) and the other was measured on a ratio scale, the method used was one-way analysis of variance (F-test). Namely, we tested whether the values of the ratio-scale variable differed significantly between the groups identified by the attribute-variable. On the other hand if both variables were attributes (nominal scale) we used the contingency table technique (X^2 -test).

There was a significant association between the initial neurological deficit and the amount of canal compromise of these 63 patients: $F(3, 59) = 3.883$, $P = 0.0133$ (fig. 1). However, it should be noted that the above significant value of the F-test criterion is attributed to the significantly higher mean of the Frankel A group, whereas the means of the other groups are approximately equal (\bar{X} Frankel A = 64, Frankel B = 43, Frankel C = 50.7, and Frankel D = 42.4). There was no correlation between the initial neurological deficit and the degree of the initial posttraumatic kyphosis: $F(3, 59) = 1.212$, $P = 0.313$. There was also no statistical association between the initial neurological deficit and the location of the retropulsed middle column fragment with respect to its proximity to the ligamentum flavum or the lamina. $X^2(3) = 1.08$, $P = 0.782$.

From the total of 63 patients, 37 (59%) improved neurologically and could be upgraded one or more Frankel grades. From the 10 Frankel A patients 2 improved. Perhaps these two patients were not

Table I

No	Age	Level	Type	Treat	Fu	Kyphiu	Kyphfu	Frankin	Frankfu	Bladin	Bladfu	Ambin	Ambfu	Comprin	Comprfu	Decompr	Improv	Impru	Difkyph	Difky10	
A	B	C	D	E	F	G	H	I	J	K	L	M	N	O	P	Q	R	S	T	U	
1	16	2	2	1	26	3	3	4	4	1	1	1	1	40	40	0	0	0	0	0	1
2	56	4	1	2	65	25	15	4	5	1	1	1	1	80	N.A.	0	1	1	10	2	
3	33	3	1	2	72	18	22	3	4	1	1	1	1	40	40	1	1	1	-4	1	
4	44	3	2	2	30	10	5	2	4	0	1	0	1	50	5	1	2	1	5	1	
5	31	1	1	2	32	40	10	3	5	1	1	0	1	25	25	0	2	1	30	2	
6	47	3	2	2	41	32	24	4	5	1	1	1	1	45	0	1	1	1	8	1	
7	62	3	2	2	74	14	8	2	3	0	0	0	0	55	N.A.	0	1	1	6	1	
8	31	2	2	2	28	28	30	4	4	1	1	1	1	60	30	0	0	0	-2	1	
9	42	2	2	2	41	29	16	2	5	0	1	0	1	25	15	0	3	1	13	2	
10	25	3	2	2	44	20	10	3	5	0	1	0	1	70	35	1	2	1	10	2	
11	39	2	1	1	56	16	10	4	4	0	0	1	1	35	35	0	0	0	6	1	
12	41	2	1	2	73	36	25	4	5	1	1	1	1	20	N.A.	0	1	1	11	2	
13	35	4	2	2	40	17	9	4	5	0	1	1	1	65	N.A.	1	1	1	8	1	
14	52	2	2	2	25	18	3	3	3	1	1	0	0	80	0	1	0	0	15	2	
15	42	4	2	2	84	13	5	1	2	0	0	0	0	70	60	1	1	1	8	1	
16	38	4	1	2	36	12	5	2	3	1	1	0	0	65	45	0	1	1	7	1	
17	63	2	2	2	28	16	15	4	4	0	0	1	1	60	0	1	0	0	1	1	
18	28	3	1	2	33	13	15	1	1	0	0	0	0	90	10	1	0	0	<-2	1	
19	72	1	2	1	48	24	18	3	4	1	1	0	1	60	N.A.	0	1	1	6	1	
20	49	3	2	2	41	32	7	3	5	0	1	0	1	25	N.A.	0	2	1	25	2	
21	25	4	1	2	37	36	10	2	4	0	1	0	1	30	30	0	2	1	26	2	
22	55	4	2	2	45	24	15	4	5	1	1	1	1	80	30	1	1	1	9	1	
23	29	3	2	2	30	11	7	4	4	0	1	1	1	30	30	0	0	0	4	1	
24	61	3	1	1	80	35	28	4	4	1	1	1	1	50	50	0	0	0	7	1	
25	31	2	2	2	44	27	16	2	5	0	1	0	1	25	N.A.	0	3	1	11	2	
26	51	3	1	2	31	32	5	3	5	0	0	1	1	70	N.A.	1	2	1	27	2	
27	28	2	2	2	50	20	12	1	1	0	0	0	0	45	10	1	0	0	8	1	
28	48	3	2	2	41	32	25	4	5	1	1	1	1	45	40	0	1	1	7	1	
29	32	4	1	1	81	12	19	4	5	0	0	1	1	40	N.A.	0	1	1	-7	1	
30	44	2	1	2	37	25	0	2	5	0	1	0	1	20	20	0	3	1	25	2	
31	34	3	2	2	72	35	28	4	4	0	0	1	1	35	35	0	0	0	7	1	
32	17	4	2	1	29	5	7	4	5	1	1	1	1	15	15	0	1	1	-2	1	

33	51	3	1	2	65	21	12	2	4	0	1	0	1	1	60	10	1	2	1	9	1
34	28	3	2	2	26	10	20	4	4	1	1	1	1	1	35	35	0	0	0	-10	1
35	62	3	2	2	48	15	8	2	3	0	0	0	0	0	60	20	1	1	1	7	1
36	25	3	1	2	25	20	5	3	5	1	1	0	1	1	50	10	1	2	1	15	2
37	58	1	1	2	72	18	17	4	5	1	1	1	1	1	20	N.A.	0	1	1	1	1
38	23	2	1	2	35	25	3	2	4	0	1	0	1	1	20	15	0	2	1	22	2
39	27	2	2	2	39	23	20	1	1	0	0	0	0	0	50	20	1	0	0	3	1
40	38	2	1	1	36	16	10	4	5	1	1	1	1	1	25	20	0	1	1	6	1
41	51	4	1	1	49	7	9	3	3	0	0	0	0	0	75	75	0	0	0	-2	1
42	19	1	1	2	44	22	3	3	4	0	1	0	1	1	35	0	1	1	1	19	2
43	32	3	1	1	31	19	15	4	4	1	1	1	1	1	40	40	0	0	0	4	1
44	51	3	1	2	69	20	25	2	3	0	0	0	0	0	60	10	1	1	1	-5	1
45	42	4	2	1	71	13	15	2	2	0	0	0	0	0	50	45	0	0	0	-2	1
46	60	1	1	1	27	12	10	1	1	0	0	0	0	0	30	30	0	0	0	2	1
47	49	1	1	2	45	21	19	2	2	0	0	0	0	0	50	10	1	0	0	2	1
48	62	2	2	1	29	23	25	3	3	0	0	0	0	0	35	35	0	0	0	-2	1
49	33	2	2	2	28	14	15	4	4	1	1	1	1	1	60	20	1	0	0	-1	1
50	48	2	2	2	48	19	20	3	3	0	0	0	0	0	45	40	0	0	0	-1	1
51	44	3	1	2	38	25	5	2	4	0	0	0	1	1	45	45	0	2	1	20	2
52	26	4	2	1	55	7	9	4	4	1	1	1	1	1	35	40	0	0	0	-2	1
53	58	2	1	2	24	7	3	1	1	0	0	0	0	0	80	10	1	0	0	4	1
54	31	4	2	2	27	13	2	1	2	0	0	0	0	0	70	30	1	1	1	11	2
55	37	2	2	2	49	22	31	1	1	0	0	0	0	0	45	45	0	0	0	-9	1
56	31	2	2	2	30	40	5	2	5	0	1	0	1	1	30	30	0	3	1	35	2
57	31	2	1	2	25	20	12	3	4	0	1	0	1	1	40	N.A.	0	1	1	8	1
58	70	3	1	1	37	20	25	1	1	0	0	0	0	0	70	70	0	0	0	-5	1
59	39	3	1	2	49	15	0	1	1	0	0	0	0	0	90	N.A.	1	0	0	15	2
60	40	2	1	2	36	35	25	4	5	1	1	0	1	1	35	0	1	1	1	10	2
61	32	1	2	1	24	40	30	4	5	1	1	1	1	1	25	25	0	1	1	10	2
62	40	3	1	2	39	25	0	3	5	0	1	0	1	0	50	0	1	2	1	25	2
63	47	1	2	2	45	11	5	3	3	0	0	0	60	10	10	1	0	0	6	1	

A : Case ; B : Age ; C : Level (1-T11, 2-T12, 3-L1, 4-L2) ; D : Type (retropulsed fragment level at 1-ligamentum flavum, 2-site where bone encircles the canal) ; E : Treatment (1 - conservative, 2 - surgical) ; F : Follow-Up (Months) ; G : Initial Kyphosis (Degrees) ; H : Follow-Up Kyphosis (Degrees) ; I : Initial Neurological Deficit (1-Frankel A, 2-Frankel B, 3-Frankel C, 4-Frankel D, 5-Frankel E) ; J : Follow-Up Neurological Deficit ; K : Bladder Sphincter Initially (0 - Paralyzed, 1 - Functioning) ; L : Bladder Sphincter Follow-Up ; M : Ambulation Initially (0 - No, 1 - Yes) ; N : Ambulation Follow-Up ; O : Initial Canal Compromise (Percent) ; P : Follow-Up Canal Compromise ; Q : Decompression (0 - Performed, 1 - Not Performed) ; R : Improvement of at least one Frankel grade (0 - No, 1 - Yes) ; S : Level of Improvement (Frankel Grades) ; T : Improvement of Kyphosis (Degrees) ; U : Correction of Kyphosis (1 - Less than 10°, 2 - greater than or equal to 10°).

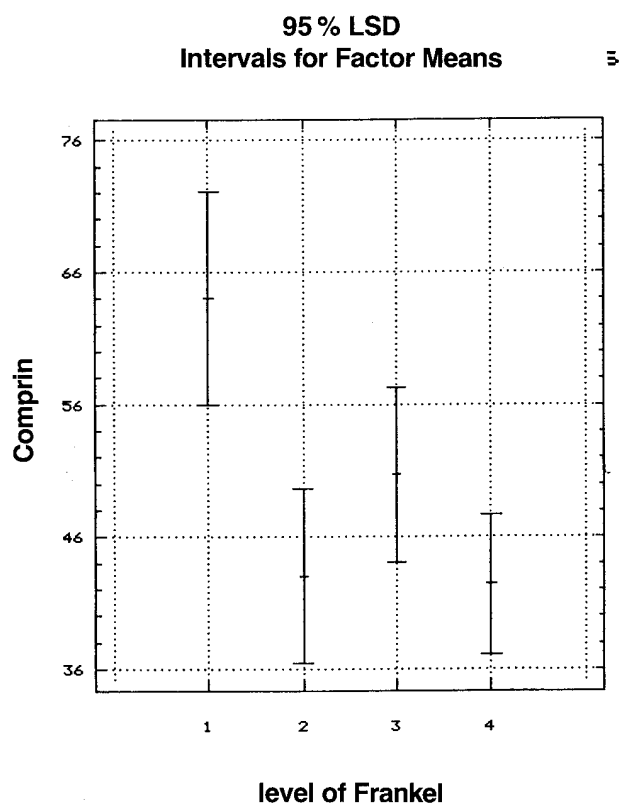


Fig. 1. — Comprin : Initial canal compromise (percent). Frankel : Initial neurological deficit (1 - Frankel A, 2 - Frankel B, 3 - Frankel C, 4 - Frankel D).

This figure shows the 95% confidence intervals of the group means. Note that only the mean of the Frankel A group is significantly different from the other three group means.

true Frankel A's but B's or C's and were wrongly graded A during the initial spine shock. From the 40 patients with conus medullaris injury, 15 demonstrated neurogenic bowel and bladder recovery. There was no correlation between neurological recovery and the initial amount of canal compromise : $F(1.61) = 3.04$, $P = 0.086$. Since P is less than 10% there is some indication of a possible association of the two variables. As with the initial neurological deficit, neurological recovery did not correlate with the type of fracture with respect to the location of the middle column retropulsed vertebral fragment. There was a direct correlation between neurological recovery and initial fracture pattern with respect to kyphosis : $F(1.61) = 9.02$, $P = 0.004$. Looking separately at cases with less than or equal to 15° of kyphosis we noted no

correlation : $F(1.18) = 0.963$, $P = 0.356$. For the group with greater than 15° of kyphosis, there is association only at the 7% level : $F(1.41) = 3.509$, $P = 0.068$. The initial kyphosis correlated directly with the level of neurological improvement on the Frankel scale : $F(3.59) = 4.966$, $P = 0.004$ (fig. 2).

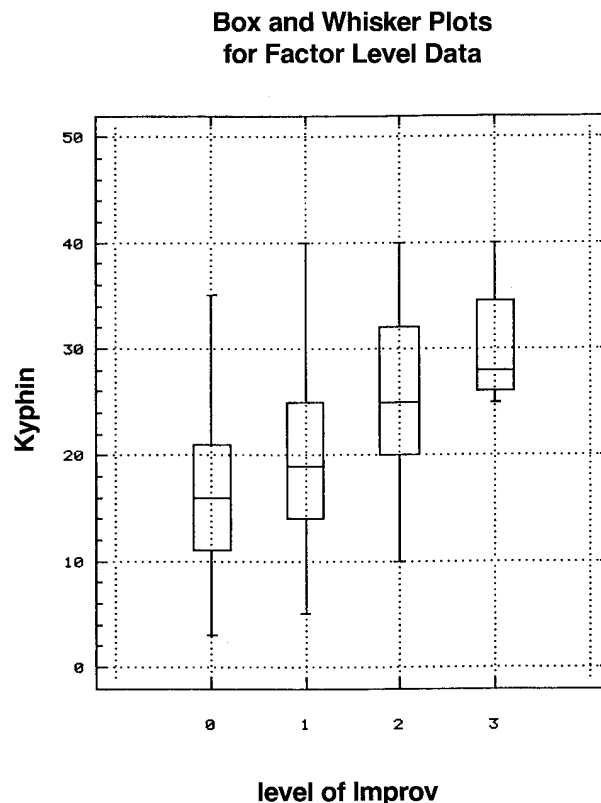


Fig. 2. — Kyphin : Initial kyphosis (degrees). Level of Improv : Improvement on the Frankel scale (levels).

To demonstrate the differences in initial kyphosis among various levels of improvement on the Frankel scale, we used the box and whisker plots which show the distribution of the individual observations with respect to quartiles.

Surgical treatment influences significantly the frequency of neurological recovery. From the 15 patients with conservative treatment only 5 improved, while from the 48 patients with surgical treatment 32 improved : $X^2(1) = 5.239$, $P = 0.022$. Neurological recovery did not correlate with the variable of decompression : $X^2(1) = 0.144$, $P = 0.704$. No patients lost function after decompression. In order to assess the value of

reduction we looked at the variable correction of kyphosis, and we noted a highly significant association between this variable and neurological improvement : $F(1,61) = 20.511$, $P = 0.0004$. We also noted a highly significant association between correction of kyphosis and level of improvement on the Frankel scale : $F(3,59) = 22.812$, $P = 0.00002$ (fig. 3).

**Box and Whisker Plots
for Factor Level Data**

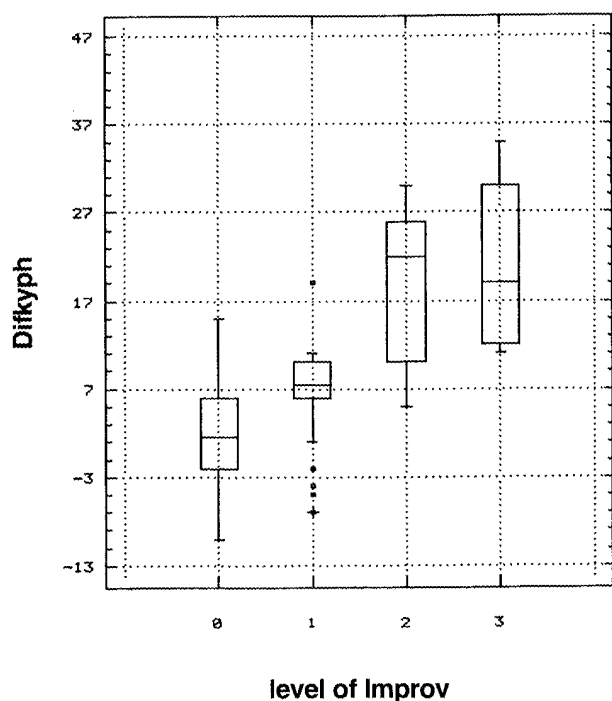


Fig. 3. — Difyph : Improvement of kyphosis (degrees). Level of improv. : improvement on the Frankel scale (levels). This figure demonstrates the influence of improvement of kyphosis among the various levels of improvement on the Frankel scale. Box and Whisker plots show the distribution of the individual observations with respect to the quartiles (The points presented with squares or crosses are extreme values).

Since we found that both initial kyphosis and correction of kyphosis influence neurological recovery, and in order to separate the benefit from reduction, we attempted a comparison of four groups. The first of them consisted of cases less than or equal to 4° , the second between 5 and 9° , the third between 10 and 15° , and the fourth

16° and above. The result of these comparisons is striking. In the first group 19 of 24 (79%) patients did not improve. In the rest of the groups the majority of the patients improved (72, 82 and 100% respectively) : $X^2(3) = 25.019$, $P = 0.0002$. These results clearly indicate that it is primarily the treatment (reduction) which influences the result favorably and not so much the initial alteration of the sagittal spine alignment (fracture pattern).

DISCUSSION

The 63 consecutive patients represent a statistically valid sample. Neither of the pattern variables, canal compromise, or level of maximum canal stenosis, influenced significantly the rate of neurological recovery, with the exception of the very severe canal compromise that did correlate significantly with complete paralysis. This is contrary to other reported data (27).

Neurological improvement appeared to be dependent on the initial fracture pattern but only with respect to the initial kyphosis. Contrary to previously reported data (4), we noted no correlation between location of the retropulsed vertebral body and disc fragments and improvement of paralysis. Dall and Stauffer (4) have also observed that burst fractures with severe kyphosis (greater than 15°) have a better prognosis in terms of neurological recovery. They assumed that these fractures with kyphotic deformity cause less force to be transmitted through the neural elements at the time of the injury ; thus they are predisposed to a more favorable neurological recovery. Contrary to our findings, they also reported that fractures with maximum canal compromise at the level of the ligamentum flavum had a better prognosis than fractures with maximum canal compromise at the level where bone encircles the canal. However these authors failed to recognize the effect of the correction of kyphosis (reduction) on neurological improvement. Consequently there is a question to be answered : it is only the initial kyphosis that influences the neurological outcome positively or the correction of this deformity ? Our study indicated that surgical treatment gave superior results than postural reduction or no reduction at all, concerning neurological recovery.

Since surgical treatment comprises two procedures, namely decompression and correction of the sagittal spine alignment, it became necessary to separate the value of each one of them. Our statistical analysis did not reveal any significant association between direct decompression and neurological recovery. Thus the favorable effect of the operation can be attributed exclusively to the restoration of the sagittal spine alignment. Since we found that both variables, initial kyphosis and correction of kyphosis influence neurological recovery, and in order to separate the value of the latter, we compared four subgroups with different amounts of correction of the kyphosis with the rest. This comparison indicated that it is not so much the initial kyphosis that correlates with a favorable result but the correction of this deformity.

Thus having made the same observation as Dall and Stauffer (4) regarding the association of severe posttraumatic kyphosis and neurological recovery, we have a different hypothesis for the explanation of this observation. We assume that the neurological deficit associated with burst vertebral fractures is partially due to permanent cord or root damage and partially to tethering of the neural structures. Furthermore, the angulation of the spine causes vascular impairment of cord and cauda equina (10, 11). With the correction of the kyphosis, either postural or surgical, spinal cord and roots become lax, the element of neuroapraxia gradually disappears, the microcirculation of the neural structures is preserved, and the chances of neurological recovery increase significantly. Our results indicated that patients treated by conservative means had inferior results. The main reason for this observation is that conservative treatment was inadequate in terms of realignment...

We must underline that our study indicates only that burst thoracolumbar fractures with kyphosis and neurological deficit require reduction. However it does not provide sufficient data regarding the need for decompression. Since the natural history of incomplete spinal cord and cauda equina lesions is to some degree favorable, the claim that decompression does not provide a greater neurological recovery has been difficult to substantiate. An increasing number of authors, however, have stated that removal of bone from

the spinal canal does facilitate neurological improvement (14, 20, 21, 24, 29). There are at least two reasons that could explain the absence of correlation between decompression and neurological recovery. The first of them is our use of posterolateral approaches as opposed to anterior or anterolateral ones, which give better results (3). The second is the relatively small number of our patients who underwent this procedure, who do not make a statistically valid group. Perhaps the combination of anterior decompression with realignment is the one which might give patients the maximum chance for neurological recovery, but this conclusion can not be supported by our data.

In conclusion, in terms of neurological recovery, burst fractures with severe kyphosis have a better prognosis than fractures that do not alter the sagittal plane alignment of the spine for the same initial neurological deficit. The most favorable outcomes can be expected following restoration of the sagittal spine anatomy.

REFERENCES

1. Bedbrook J. Treatment of thoracolumbar dislocations and fractures with paraplegia. *Clin. Orthop.*, 1975, 112, 27-43.
2. Been H. D. Anterior decompression and stabilization of thoracolumbar burst fractures by use of the slot-Zielke device. *Spine*, 1991, 16, 70-77.
3. Bradford D. S. Deformities of the thoracic and lumbar spine secondary to spinal injury. In Saunders W. B. *Moe's textbook of scoliosis and other spinal deformities*, Philadelphia, 1987, p. 443.
4. Dall B. E., Stauffer E. S. Neurologic injury and recovery patterns in burst fractures at the T12 or L1 motion segment. *Clin. Orthop.*, 1988, 223, 171-176.
5. Davis W. E., Morris J. H., Hill V. An analysis of conservative management of thoracolumbar fractures and fracture dislocations with neural damage. *J. Bone Joint Surg.*, 1980, 62-A, 1324-1328.
6. Dendrinos G., Asimakopoulos A., Exarhou E., Papagianopoulos G. Kyphosis as a prognostic factor in burst thoracolumbar fractures with neurological deficit. *J. Bone Joint Surg.*, 1993, Suppl. II, 75, 180.
7. Denis F. The three column spine and its significance in the classification of acute thoracolumbar spine injuries. *Spine*, 1983, 8, 817-823.
8. Denis F. Spinal instability as defined by the three column spine concept in acute trauma. *Clin. Orthop.*, 1984, 189, 65-77.

9. Dickson J. H., Harrington P. R., Erwin D. Results of reduction and stabilisation of severely fractured thoracic and lumbar spine. *J. Bone Joint Surg.*, 1978, 60-A, 799-805.
10. Dohrmann G. I., Wagner F. C. Jr., Bucy P. C. The microvasculature in transitory traumatic paraplegia. An electron microscopic study in monkey. *J. Neurosurg.*, 1971, 35, 263-271.
11. Ducker T. B., Kindt G. W., Kempe L. G. Pathological findings in acute experimental spinal cord trauma. *J. Neurosurg.*, 1971, 35, 700-708.
12. Erickson D. L., Leider L. L. Jr., Brown W. E. One-stage decompression stabilisation for thoracolumbar fractures. *Spine*, 1977, 2, 53-56.
13. Flesh J. R., Leider L. L., Ericson D. L. Harrington instrumentation and spine fusion for unstable fractures and dislocations of the thoracic and lumbar spine. *J. Bone Joint Surg.*, 1977, 59-A, 143-153.
14. Fountain S. S. A single — stage combined surgical approach for vertebral resections. *J. Bone Joint Surg.*, 1979, 61-A, 1011-1017.
15. Frankel H. L., Hancock D. O., Hyslop G., Melzak J., Michaelis L. S., Ungar G. H., Vernon J. D. S., Walls J. J. The value of postural reduction in closed injuries of the spine in paraplegics and tetraplegics. *Paraplegia*, 1969, 7, 179-192.
16. Grantham S. A., Malberg M. I., Smith D. M. Thoracolumbar spine flexion distraction injuries. *Spine*, 1976, 1, 172-178.
17. Hashimoto T., Kaneda K., Abumi K. Relationship between traumatic spinal canal stenosis and neurological deficit in thoracolumbar burst fractures. *Spine*, 1988, 13, 1268-1272.
18. Holdsworth F. W., Hardy A. Early treatment of paraplegia from fractures of the thoracolumbar spine. *J. Bone Joint Surg.*, 1953, 35-B, 540-550.
19. Jacobs R. R., Asher M. A., Snider R. K. Thoracolumbar spine injuries. A comparative study of recumbent and operative treatment in 100 patients. *Spine*, 1980, 5, 463-477.
20. Jelsma R. K., Rice J. F., Jelsma L. F., Kirsch P. T. The demonstration of significance of neural compression after spinal injury. *Surg. Neurol.*, 1982, 18, 79-92.
21. Kostuick J. P. Anterior spinal cord decompression for lesions of the thoracic and lumbar spine, techniques, new methods of internal fixation, results. *Spine*, 1983, 8, 512-531.
22. McAfee P. C., Bollman H. H., Yuan H. A. Anterior decompression of traumatic thoracolumbar fractures with incomplete neurologic deficit using a retroperitoneal approach. *J. Bone Joint Surg.*, 1985, 67-A, 89-104.
23. McAfee P. C., Yuan H. A., Lasda N. A. The unstable burst fracture. *Spine*, 1982, 7, 365-373.
24. Paul P. C., Michael R. H., Dunn J. E., Williams J. P. Anterior transthoracic surgical decompression of acute spinal cord injuries. *J. Neurosurg.*, 1975, 43, 299-307.
25. Riska E. B., Myllynen P., Bostman O. Anterolateral decompression for neural involvement in thoracolumbar fractures. *J. Bone Joint Surg.*, 1987, 69-B, 704-709.
26. Stauffer E. S. Cervical spine trauma. Section 24. Orthopaedic knowledge update 1. Home study syllabus. Chicago, AAOS, 1984, 199-208.
27. Trafton P. G. Computed tomography of thoracic and lumbar spine injuries. *J. Trauma*, 1984, 24, 506-510.
28. Yosipovitch Z., Robin G. C., Makin N. Open reduction of unstable thoracolumbar spinal injuries and fixation with Harrington rods. *J. Bone Joint Surg.*, 1977, 59-A, 1003-1015.
29. Young B., Brooks W. H., Tibbs P. A. Anterior decompression and fusion for thoracolumbar fractures with neurologic deficit. *Acta Neurochir.*, 1981, 57, 287-298.

SAMENVATTING

G. K. DENDRINOS, J. G. HALIKIAS, P. N. KRALLIS, A. ASIMAKOPOULOS. Factoren die de neurologische recuperatie begunstigen bij thoracolumbale „burst” fracturen.

Het verband tussen thoracolumbale wervelzuilfracturen, hun behandeling en het herstel van het neurologisch deficiet werden onderzocht. Drieënzestig patiënten met wervelzuilfracturen van Th11 tot en met L2 gepaard gaande met een neurologisch letsel werden geëvalueerd met standaard radiographieën, CT-scan en een quantitatief neurologisch onderzoek. Het percentage van kanaalvernauwing, de lokalisatie van de achterwaarts verschoven botfragmenten, de kyfose, de behandelingswijze en het neurologisch herstel werden bestudeerd. De „follow-up” schommelt tussen 24 en 84 maanden (gemiddeld 44 maanden). Vijftien patiënten werden conservatief behandeld, achtenveertig patiënten heelkundig. Een laterale en posterieure decompressie werd bij zessentwintig patiënten verricht.

De relatie tussen de initiële verlamming en het type fractuur was niet duidelijk behalve misschien voor de Frankel A breuken. De initiële kyfose bepaalt gedeeltelijk de prognose van het neurologisch herstel maar er bestaat geen verband tussen neurologische recuperatie en de vermindering in kanaaldoorsnede, de lokalisatie van de achterwaarts verschoven botfragmenten of uitgevoerde decompressie. De verbetering van de verlamming hangt samen met de reductie in het sagitaal vlak. De meerderheid van de patiënten die een correctie gehad hebben van de kyfose van meer dan 5°, vertoonden tevens een verbetering van hun verlamming. De neurologische recuperatie was slecht in die gevallen

waar een correctie van de kyfose van minder dan 5° verricht werd.

In geval van „burst fractures” zou de verlamming die gepaard gaat met een belangrijke kyfose enerzijds te wijten zijn aan een letsel van de wortels of het ruggemerg maar anderzijds ook aan de neuropraxie veroorzaakt door de angulatie van de neurale structuren en hun bloedvaten. Na reductie van de kyfose zullen het ruggemerg, de zenuwwortels en hun bloedvaten niet meer onder spanning staan en is de kans op recuperatie van het neurologisch letsel significant verhoogd.

RÉSUMÉ

G. K. DENDRINOS, J. G. HALIKIAS, P. N. KRALLIS, A. ASIMAKOPOULOS. Facteurs qui influencent la récupération neurologique dans les fractures-éclatements du rachis thoraco-lombaire.

Les auteurs ont étudié les relations entre le type de fractures du rachis thoraco-lombaire, le traitement et la récupération neurologique. Soixante-trois patients présentant une fracture des vertèbres D11 à L2 avec un déficit neurologique associé ont été évalués sur la base des radiographies standard, du CT scan et d'un examen neurologique quantitatif. Le pourcentage de réduction du diamètre du canal, la localisation du

fragment osseux rétro pulsé, la cyphose, le traitement et la récupération neurologique ont été les paramètres utilisés. Le suivi a varié de 24 à 84 mois (moyenne 44 mois). Le traitement a été conservateur pour 15 patients et chirurgical pour 48 patients. Une décompression postérolatérale a été pratiquée chez 26 patients.

La gravité de la paralysie initiale ne dépend pas du type de fracture sauf peut être pour les cas Frankel A. La récupération neurologique est influencée par le degré de cyphose initiale mais pas par l'importance de la sténose du canal, ni par la localisation du fragment osseux rétro pulsé, ni par la décompression. L'amélioration de la paralysie est associée à la restauration de l'alignement dans le plan sagittal : la majorité des patients qui ont eu une correction de la cyphose de plus de 5 degrés, ont présenté une amélioration neurologique. La récupération neurologique fut médiocre pour les cas où la correction de la cyphose était de moins de 5 degrés.

Nous estimons que la paralysie initiale dans les fractures-éclatements accompagnées de cyphose importante est due d'une part à des lésions des racines ou de la moëlle épinière et, d'autre part, à une neuropraxie qui a pour cause l'angulation des structures nerveuses et de leurs vaisseaux. Après la réduction de la déformation en cyphose, la moëlle épinière, les racines et leurs vaisseaux deviennent détendus et la possibilité d'observer une récupération neurologique augmente significativement.