

Research Article

Evaluation the results of surgical management of traumatic paraplegia in traumatic thoracolumbar fractures

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ABSTRACT

Background: Thoracolumbar spine fractures are common injuries that can result in significant disability, deformity and neurological deficit. Aim of this study was to evaluate the results of surgical management of traumatic paraplegia, complete or incomplete as classified by Frankel scoring.

Methods: A prospective study was conducted in patients attending outdoor and emergency department of Orthopedics of a tertiary care teaching institute in Kolkata, West Bengal with traumatic paraplegia involving the dorsolumbar spine. The important objectives are the time for recovery of various functions like sensory, motor and bowel and bladder function, comparison between early and late decompression, results of posterolateral fusion and time taken for solid bony fusion after operation. Total 46 cases were selected within a minimum of 6-month post-operative follow-up of which 4 cases lost in follow-up. Data collected from patient records included age, sex, time from injury to hospitalization, initial neurological status as per Frankel Score, MRI findings, surgery performed, postoperative course and neurological status at the time of discharge and latest follow up. Patients lost to follow up were not studied for outcome analysis.

Results: When decompression done within 1st week in incomplete paraplegia, 80% of the patients showed return of grade 3 power. In complete paraplegia cases, 11% of the patients had return of power up to grade 3 when decompression done within 1 week, where no cases showed return of grade 3 power when decompression done after 2nd or 3rd week.

Conclusions: After recovery from spinal shock, the earlier the surgical compression done, the better the neurological and bowel/bladder function recovery both in complete and incomplete paraplegic cases. Reduction is better and easy and less time consuming in early decompression than in late. Motor recovery can continue for over 6 months after decompression.

Keywords: Spinal cord injury, Traumatic paraplegia, Dorsolumbar spine, Decompression, Posterior fixation, Neurological deficit

INTRODUCTION

Fractures of the thoracic and lumbar region constitute a spectrum of injuries ranging from the simple undisplaced

fractures to complex fracture dislocations. Anatomically and functionally, the thoracic and lumbar spine can be divided into three regions - thoracic spine (T1-T10), thoracolumbar junction (T10-L2) and the lumbar spine

(L3-L5). The thoracic spine is functionally rigid due to coronally oriented facet joints, thin intervertebral discs and the ribcage.

So, it requires huge amounts of energy to produce fractures and dislocations. The narrow spinal canal in this region predisposes to spinal cord damage resulting in a high incidence of neurological deficit.

The lumbar spine, on the other hand, is relatively flexible due to the thicker intervertebral discs, sagittal orientation of facet joints and the absence of the rib cage. The relatively lesser incidence of neurological injury in lumbar fractures can be attributed to the large size of the neural canal and the greater resilience of the cauda equina nerve roots.¹

Though fractures of the thoracolumbar spine are common injuries, 50% of these are unstable and can result in significant disability, deformity and neurological deficit.² Thoracolumbar fractures are more frequent in men, and the peak incidence is observed between 20 and 40 years.³ Neurological injury complicates 20-36% of fractures at the thoracolumbar junction in different studies. The chances and extent of neurological deficit depend on the type of fracture.⁴

Traumatic paraplegia is an unanticipated catastrophe in an individual's life, posing a huge economic as well as social burden. The healthcare does not end with fixation of spine and inculcates a programmed rehabilitation and preventive management plan involving multiple personnel and family members. Early surgery and comprehensive rehabilitation markedly reduces the overall morbidity of spinal cord injured patients by enabling the patient to lead an independent life.⁵

In a developing country like India, where tertiary health care is not universally accessible and acceptable, the consequences of traumatic paraplegia and loss of manpower are well imaginable.

Data of traumatic paraplegia at a tertiary care hospital, Kolkata was analysed with the analysis of the results of surgical management of traumatic paraplegia, complete or incomplete.

METHODS

The prospective study was conducted in the department of Orthopedics of a tertiary care teaching institute in Kolkata, West Bengal, India. Institutional Ethics Committee permission was sought before enrollment of study subjects in the above study.

The study participants were followed up for 6 months in post-operative period. The cases included in this study were the patients attending outdoor and emergency with traumatic paraplegia involving the dorsolumbar spine.

Inclusion criteria

Presence of traumatic paraplegia (complete or incomplete) and fulfilled the following criteria

- Fracture and/or dislocation of the vertebra of dorso-lumbar spine involving D8 to L5 spine
- Fractures involving one or maximally two vertebrae
- Skin condition of the operative field normal patients and party agreed to have a surgical decompression

Exclusion criteria

- Patients below 18 years
- Patients unfit for undergoing operation in pre-anesthetic check-up
- Patients with head injury or other gross injuries that may preclude undergoing operation
- Patients with multiple vertebral injuries (> 2 vertebrae)
- Injury of the spinal cord and paraplegia with high dorsal spine (above D8)
- Patients presenting late (more than one month after injury)
- Patients with traumatic paraplegia but without signs of cord compression on MRI (where paraplegia is due to cord edema or myelomalacia)

Informed consent was taken after proper counseling and proper pre-anesthetic check-up. The patients were evaluated by X-ray of spine (AP and Lateral view) and sometimes CT scan. Due to financial constraint contrast myelography was done only few cases. In most cases pedicle screw with plate or rod was used and posterior stabilization and posterior fusion with cortico cancellous bone graft from posterior iliac crest was done. In all the cases water bed was used during pre-operative and postoperative period to prevent bed sore. Preoperative and post-operative neurological charts (according to Frankel's grade and ASIA score (motor and sensory) was maintained with regular assessment for proper post-operative neurological recovery assessment.⁶

Recovery from spinal shock was noted by using clinical methods like return of bulbocavernous reflex. Direct or indirect decompression was done. In most of the cases laminectomy was done for direct decompression.

Decompression was confirmed by using a narrow gauge rubber tube. Condition of the spinal cord was checked by direct vision. Any retro-pulsed fragment compressing upon the cord was taken out.

Pedicle screws were inserted into the proximal and distal stable vertebra under image intensification. Then the fracture was stabilized by rods or plates. The pedicle entry points were identified (by intersection method and confirmed by image intensifier guidance) and opened, probed all around and the pedicle screw was introduced. Peroperatively, features such as cord pulsation, cord atrophy and lacerations were looked for.

Post-operatively, wound healing, amount of drainage, neurological recovery, radiological assessment, time taken ambulation and ultimate recovery were recorded. We used Frankel grade and ASIA scoring system for pre and post-operative neurological assessment.⁶

In all cases, long dorso-lumber brace was given to the patients after removal of stitches at 14th postoperative day. Patients were discharged with advice for follow-up.

Follow-up

First follow up was done after 2 week, 2nd follow up after 6 weeks. Then monthly follow up until the radiological sign of solid fusion was seen on X-ray. Patients were assessed for neurological recovery and assessment of return of bowel and bladder function in every follow-up. Some case was referred to urosurgery department for management of bladder function problems.

RESULTS

46 patients in whom posterior stabilization of the spine was done in this institution and followed up for a period ranging from 6 months to 2 years, 4 of 46 patients lost follow-up. Remaining 42 patients were considered for the study (Table 1).

Table 1: Demographic characteristics of the traumatic paraplegia subjects.

Age (Years)	Number
16- 20	8
21-25	16
26-30	12
31-35	4
36-40	2
Total	42
Sex Incidence (n=42)	
Male	Female
32 (76.19%)	10 (23.81%)
Occupation (n=42)	
Manual labor	28 (66.67%)
Sedentary worker	10 (23.81%)
Unemployed/ housewife	4 (9.52%)

Table 2: Clinical characteristics of the traumatic paraplegia subjects.

Vertebra involved	
Vertebra	Number of patients (n=420)
D9	1 (2.38%)
D10	2 (4.76%)
D11	5 (11.90%)
D12	6 (14.29%)
L1	7 (16.66%)
L2	7 (16.66%)
L3	8 (19.05%)
L4	4 (9.52%)
L5	2 (4.76%)
Days elapsed after injury at presentation	
Days	Number of patients
0-3	28
4-7	10
8-11	3
12-15	0
16-19	1
Time of decompression (post-injury)	
Days	Number of patients Complete Incomplete
0-7	9 5
8-14	8 11
15-21	4 5
Mechanism of injury	
Road accidents	18 (42.85%)
Fall from height	24 (57.14%)
Paraplegia	
Complete	22 (52.38%)
Incomplete	20 (47.62%)
Initial observation (ASIA impairment scale)	
Grade	Number
A	22 (52.38%)
B	12 (28.57%)
C	8 (19.05%)
D	Nil
E	Nil
Classification of fractures	
Types	Number of patients
True wedge compression	28 (66.67%)
Burst	10 (23.81%)
Fracture dislocation	4 (9.52%)

Table 3: Post-operative sensory recovery (mainly fine touch) in traumatic paraplegia patients.

Onset of sensory recovery	Number of cases
1 st week	22
2 nd week	12
3 rd week	6
4 th week	2

Return of power in incomplete paraplegia

In all cases some return of power was there, mostly from grade 3 or grade 4 or grade 5.

Return of Power in Complete Paraplegia

Hip flexors: within grade 3 and 4, Hip abductors/ Quadriceps/ Hamstrings: within grade 2 and 3, Tibialis Anterior/ EHL/ FHL and Gastrosoleus: power did not return at all

Table 4: Onset of motor recovery incomplete paraplegia in study subjects.

Onset of motor recovery	Number of cases
1 st week	10
2 nd week	7
3 rd week	2
4 th week	1

Table 5: Motor recovery in complete paraplegia in study subjects

Muscles	Power at presentation	Post operative	Number of cases
Hip flexors	0	3	7
	0	2	5
Hip abductors	0	2	5
	0	3	7
Quadriceps	0	2	6
	0	3	6
Hamstrings	0	2	8
	0	3	4
Tibialis anterior	0	0	All
EHL	0	0	All
FHL	0	0	All
Gastro-Soleus	0	0	All

Table 6: Onset of motor recovery in complete paraplegia.

Onset of motor recovery	Number of cases
1 st week	1
2 nd week	4
3 rd week	8
4 th week	7
5 th week	3
6 th week	0

Table 7: Bowel and bladder functions recovery.

Complete	Incomplete
Autonomic in all cases	Normal in 9 cases Hesitancy, incomplete in 11 cases

Stability of implants

We got 3 patients where there was pull-out of the screws completely out of pedicle. In one patient there was loosening of the outee/innie followed by loosening of the rod. In all three cases these happened within 4 weeks postoperatively. In these patients we had to continue on

conservative management and solid bony fusion developed between 12 to 20 weeks.

Table 8: Time taken for recovery of bladder function in incomplete paraplegia (weeks).

Time	week
2 nd	3
3 rd	4
4 th	4
5 th	4
6 th	2
8 th	1
10 th	1
12 th	0
16 th	1

Table 9: Comparison among return of muscle power after decompression at different time in incomplete paraplegia.

	Grade 3	Grade 2	Grade 1	NIL
1st Week	80%	20%		
2nd Week	36%	36%	18%	9%
3rd Week	25%	25%	25%	25%

Table 10: Comparison among return of muscle power after decompression at different time in complete paraplegia.

	Grade 3	Grade 2	Grade 1	NIL
1st Week	11%	22%	22%	45%
2nd Week		16%	16%	68%
3rd Week			20%	80%

Table 11: Comparison between onsets of sensory recovery after decompression done at different time in incomplete paraplegia

	1 st W	2 nd W	3 rd W	4 th W
1st Week	80%	20%		
2 nd Week	27%	54%	9%	9%
3rd Week		25%	50%	25%

Table 12: Comparison between onset of sensory recovery after decompression done at different time in complete paraplegia.

	1 st W	2 nd W	3 rd W	4 th W	NIL
1st Week		11%	11%		78%
2 nd Week		12%			88%
3rd Week				20%	80%

Table 13: Complications in clinical study participants.

Complications	Number of patients
Bed sore	05
Infection	05
Pull out of screws	04
Dural tears	04
Persistent fistula with leakage of urine	03
Morbidity of bone graft donor site	04
Late back or leg pain	03
Prominence of screw	01
Post-op increase in neuro deficit	01

DISCUSSION

Thoracolumbar junction is the commonest area involved in spinal injury. This area represents the transition from thoracic kyphosis to lumbar lordosis and the axis of the body passes in front of this junction when the patient is erect. So, there is anterior bending moment working at this junction resulting in maximum stress concentration in this area which may be responsible for implant failure in this junction.⁷ Decompression in the spinal injury is one of the most controversial concepts.

Both experimental and clinical findings of Benzel EC et al, Dolan EJ et al, and Maiman DJ et al clearly documented the role of neural decompression in improving the neurological outcome.⁸⁻¹⁰ The essential key to reduction of intracanal fragments in burst fractures is distraction. So any device used posterior must have large distractive force. The pedicular screw system can provide large amount of distraction. Open up the collapsed

anterior segment at specific level by appropriately contouring the rod according to the saggital curvature of the spine. Vaile et al. also provided evidence that transpedicular decompression in experienced hand is usually able to restore an almost normal cross-sectional area at the affected level of spinal canal.¹¹ In present study, decompression was done in patients with >50% collapse of the vertebral body and with canal narrowing, posterior-laterally by scooping out the retro pulsed bony fragments from the canal as well as pushing some of the fragments anteriorly.

Cord injury consists of the primary contusion, secondary injury due to cellular changes at the injury site and the effects of ongoing neural compression. The first mechanism is amenable only to preventive treatment. Intensive investigation for effective agents is underway that may modify the secondary injury response. The use of methylprednisolone in the immediate post-injury phase has been shown to marginally improve outcome in

national acute spinal cord injury study investigations, but this improvement has not been substantiated in other studies, and its role remains controversial. In our study we have routinely used injection methylprednisolone with a dosage and indication as recommended by NASCIS, but we have not found any neurological improvement with use of methylprednisolone in all of the 22 patients in whom it was used.¹²⁻¹⁸

Regarding the procedure of decompression, we had two options in our hand, either direct or indirect decompression. In most of the early cases we have found, indirect decompression was satisfactory as seen on image intensifier. Laminectomy, screw fixation followed by slight distraction was all to be done in cases of early decompression. But in late cases (beyond 11 days) it has been found that the decompression was not possible only by postural reduction. After pedicle screw fixation good amount of power was necessary to distract the fracture fragments and for the decompression. In late cases, extensive fibrosis and bleeding of the operative field made the procedures difficult and time consuming. The distorted anatomy of the old fracture made the situation more problematic.

Regarding neurological recovery, some amount, be it complete or incomplete, be it early or be it sensory or motor or bowel and bladder function, was noticed in all the cases (Table 3-7). As per report published by Denis F, there is improved neurological outcome in effective cord compression after injury, stands for our findings regarding post-operative neurological recovery in spinal injury patients.^{19,20}

In all the cases of spinal injury where it was treated by surgical management, the onset of sensory recovery was earlier than motor recovery in all the cases (Table 11-12). Almost 75% of the cases showed some amount of sensory recovery within first 5 days of the operation. The onset of sensory recovery continued for maximally upto 4th week post-operatively in the cases studied by us. According to Kostuik JP, persistent neural compression can inhibit neurologic recovery and anterior decompression can provide dramatic improvement in many patients.²¹

In present studies patients were divided in three categories:

- Decompression done within 1 week
- Decompression done in 2nd week
- Decompression done in 3rd week

When decompression done within 1st week in incomplete paraplegia, 80% of the patients show grade 3 power return, whereas 25% of the patients show return of grade 3 power when decompression done in 3rd week in incomplete paraplegia cases (Table 9). In complete paraplegia cases, 11% of the patients had return of power up to grade 3 when decompression done within 1 week,

where no cases showed return of grade 3 power when decompression done after 2nd or 3rd week (Table 10).

In incomplete paraplegia, 80% of the patients had onset of sensory recovery within 1 week, when the decompression done within 1st week (Table 9). In complete paraplegia, 11% of the patients had sensory recovery within 2 weeks when decompression done within 1 week (Table 10). So our conclusion is early decompression definitely has some role regarding motor and sensory function return, both in complete and incomplete paraplegia.

But the timing of surgery for spinal cord injuries is controversial. Most authors agree that in the presence of a progressive neurological deficit, emergency decompression is indicated. In patients with complete spinal cord injuries or static incomplete spinal cord injuries, some authors advocate delaying surgery for several days to allow resolution of cord edema, whereas others favor early surgical stabilization. There is no conclusive evidence in the literature that early surgical decompression and stabilization improve neurological recovery or that neurological recovery is compromised by a delay of several days.

Studies by Bohlman et al, Transfeldt et al, Bradford et al, and others have documented return of neurological function after anterior decompression done more than a year after the initial injury.²²⁻²⁵ For neurological normal patients with unstable spinal injuries and those with non-progressive neurological injuries, we believe that open reduction and internal fixation should be carried out as soon as possible.

Mirza et al. in a recent study concluded that patients who sustain acute traumatic injuries to the cervical spine with associated neurologic deficit may benefit from cervical decompression and stabilization within 72 hours of injury. Surgery within 72 hours of injury is not associated with a higher complication rate. Early surgery may improve neurological recovery and decrease hospitalization time in patients with cervical spinal cord injuries.²⁶ In some patients in our study it has been found some return of sensory or motor function was possible even when the MRI findings showed there was complete transection of the cord.

In all the cases it was not possible to restore the height of the vertebral body, as seen on the post-operative X-ray. But even then the neurologic recovery continued. According to literature, canal compromise becomes a concern only when a high degree of compromise is recognized. Residual compromise greater than 50% of the cross-sectional area is worrisome at the T12/L1 level, where the conus medullaris and cauda equine fill the spinal canal. Further small increments or axial or sagittal collapse can compromise neurologic elements and the decompression and stabilization should be considered for both mechanical and neurological reason.

On the other hand, 80 to 85% of the canal compromise can well be tolerated in the lower lumbar spine, where only a few roots remain in the otherwise capacious canal. Retropulled bony fragments resolved remodel over time and do not need to be removed. Regarding the onset of motor recovery in complete paraplegia, it was maximum between 3rd and 4th week but it continued even up to 6 months in 2 of the 22 patients of complete paraplegia. In incomplete paraplegia the peak of onset of motor recovery was between the end of the first week and the second week. And it continued up to 3 months in 4 of the incomplete paraplegic patients.

According to Ann S et al and Leandro U, Taniguchi LU et al, in their series of the spinal injury, the motor recovery continued for 6 months which in the line of our observation.^{27,28} In follow-up, in only the incomplete paraplegic patients, it was possible to change paraplegia since complete sensory recovery was seen in no case of complete paraplegia. This observation supports the findings of the study of Yilmaz O et al.²⁹

Water reported on 148 patients and found that 73% who were to be motor complete paraplegics at 1 month were unchanged in neurological level at 1 year. 70% of the muscles initially graded 1 to 2 on a scale of 5 improved to 3 or greater at 1 year, and 3 to 7% of those initially grade 0 improved to grade 3 at 1 year. The majority of neural recovery occurred in first 6 to 9 months, with no improvement beyond 12 to 18 months.

4% of these patients initially assessed as complete converted late to incomplete status. Of these 6 cases, 4 regained continence and 2 became ambulatory with a reciprocal gait. In contrast, those with incomplete paraplegia had the following results: 85% of the muscles graded 1 to 2 on a scale of 5 at 1 month improved to grade 3 or greater at 1 year and of the muscles graded 0/5 at month fully 55% regained some volitional control and 26% regained useful motor function. In most of the patients the total amount of blood transfused preoperatively and post-operatively to keep the hemoglobin level above 10gm% is between 3 to 5 units. In one patient we had to transfuse 7 units and in 3 patients 3 units and all these cases late decompression was done.

Regarding the return of bowel and bladder function in all the cases, this was automatic in complete paraplegia. In two cases where suprapubic cystostomy was done, fistulae developed from bladder to anterior abdominal wall. In one patient scrotal fistula developed. In 9 out of 20 patients of incomplete paraplegia bowel and bladder function got almost normal in 6 months follow-up, whereas rest of the patients developed hesitancy or incontinence. But the bladder sensation returned back in 15 patients (75%) of incomplete paraplegia. According to Burns AB et al, most patients with paraplegia can regain social continence with appropriate rehabilitative training, urologic care and surveillance.³⁰

In present study, 5 patients developed bed sore over the sacral region and posterior iliac crest and all of them developed bed sore preoperatively. This was due to not using water bed and improper postural care. All of these patients presented late after the injury and it was very difficult to deal with the bed sore. All of the patients were complete paraplegics. In 6 months follow-up 3 of these patients had non healing bed sore and this got complicated by infection. Bed sore healed in remaining 2 patients with proper postural care and after using water bed and by local dressing.

Infection of the surgical wound with in situ implants complicated 5 cases in our series (Table 13). This acute infection was associated with increasing pain, fever and drainage within first month postoperatively. The patients were taken to the operating room. Soft tissue debridement, obliteration of the cavity if present and closure of the wound over drain were done. The drain was left for extended period of time and intravenous antibiotics administered after obtaining C/S reports. In 2 patients we had to remove implant at 5th month after observing solid posterior bony fusion on X-ray to deal with intractable infection and spinal brace had to be continued for extended period of time.

Pull-out of the screw or posterior migration was a complication in 4 patients in our series (Table 13). In all the patients this complication occurred within 3 months post-operatively. We had to remove the screws and rods and the patients were treated conservatively with bed rest, postural care and spinal hyperextension brace. 2 of the 4 patients developed kyphotic deformity of 40 degree but pseudoarthrosis did not developed in these patients. About 4 of the patients had iatrogenic dural tear during laminectomy or screw fixation. Repair of the dural tear done immediately with the thoracodorsal fascia using simple stitches.

The literature supports the immediate repair of dural tear noted at the time of surgery before proceeding with the remainder of the surgical procedure. Post-operatively 2 patients developed spinal headache which was treated by postural care and analgesics.

The recovery was uneventful in all the 2 cases. Persistent fistula with urethral leakage of urine was the complication in 2 patients where suprapubic cystostomy was done. In 1 patient with scrotal hematoma, fistula developed after incision and drainage of the hematoma (Table 13).

Late back and/ leg pain after spinal fracture can originate the damaged structural element of the spine, in soft tissue through mal-alignment or spinal imbalance; from the neural element through compression, syrinx or tethering. According to McLain et al. 5 patients (12%) of our case series had severe late back of leg pain post-operatively and it was difficult to treat. At mean follow-up of 52 months, they found that 58% of his surgical patients were

pain free. 17% had mild pain and 25% had moderate to severe pain.³¹

Prominence of screw was a problem in only patient in our series which was symptom free. The patients were treated with reassurance and that did not pose any problem to the patient in 1 year follow-up. Postoperative neurologic deficit can be classified according to the severity of the deficit. Minor deficit takes the form of radiculopathy, sensory impairment without motor loss, temporary dysesthesias in the feet or lesser degrees of neurologic deficit.

Major deficits are considered those in which the patient suffers from postoperative paraparesis, paraplegia or a spinal cord syndrome. Among these 70% deficits are transient, according to the literature. The Stagnara wake up test is still the gold standard test to detect gross motor deficit. In our series, we had 1 patient with incomplete paraplegia who had deterioration of 1 grade of power postoperatively.³² The patient was taken back to operation theatre immediately for exploration to find out the pathology for deterioration of power. A block of bone, given as a bone graft, found to be compressing on the cord and it was taken out. The recovery was uneventful.

Regarding the return of bowel and bladder function in all the cases this was autonomic in complete paraplegia. In 9 out of 20 patients developed hesitancy or incontinence (Table 7). But the bladder sensation returned back in 15 patients (75%) patients of incomplete paraplegia.

The complications were bed sore, infections, pull-out of screws, dural tear, and persistent fistula with leakage of urine, morbidity of the bone graft donor site, late back or leg pain, prominence of screws, post-operative increase in neurodeficit (Table 13).

CONCLUSION

In our series of 46 cases of traumatic paraplegia, 22 cases were complete paraplegia and 20 cases were incomplete paraplegia, 4 cases being lost in follow-up after analysis of the results we could draw the following conclusions:

- After recovery from spinal shock, the earlier the surgical compression done, the better the neurological and bowel/bladder function recovery both in complete and incomplete paraplegic cases.
- Reduction is better and easy and less time consuming in early decompression than in late.
- Higher dosage of methylprednisolone is not effective for betterment in the neurological outcome.
- In early cases indirect decompression is possible, but in late cases direct decompression has to be done.
- Even with MRI findings of complete transection of cord, some inexplicable sensory recovery took place, which mandates surgical decompression in all cases, be it early or late.

- Motor recovery can continue for over 6 months after decompression.
- At follow-up, only incomplete injury cases could be converted to higher ASIA scale.
- In spite of lack of restoration of vertebral height, neurological recovery can continue.
- Sensory recovery occurs earlier than motor recovery in all the cases.
- Use of water bed and proper postural care definitely decrease the possibility of bed sore in all paraplegic patients.
- Bowel and bladder function may return to normal in incomplete paraplegic cases but never in complete paraplegic cases.

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REFERENCES

1. Rajasekaran S, Kanna RM, Shetty AP. Management of thoracolumbar spine trauma: An overview. *Indian J Orthop.* 2015;49(1):72-82.
2. Gertzbein SD. Scoliosis Research Society. Multicenter spine fracture study. *Spine (Phila Pa 1976).* 1992;17:528-40.
3. Gertzbein SD, Khoury D, Bullington A, St John TA, Larson AI. Thoracic and lumbar fractures associated with skiing and snowboarding injuries according to the AO Comprehensive Classification. *Am J Sports Med.* 2012;40:1750-4.
4. Magerl F, Aebi M, Gertzbein SD, Harms J, Nazarian S. A comprehensive classification of thoracic and lumbar injuries. *Eur Spine J.* 1994;3:184-201.
5. Scivoletto G, Morganti B, Molinari M. Early versus delayed inpatient spinal cord injury rehabilitation: An Italian study. *Arch Phys Med Rehabil.* 2005;86:512-6.
6. Maynard FM Jr, Bracken MB, Creasey G, Ditunno JF Jr, Donovan WH, Ducker TB, et al. International standards for neurological and functional classification of spinal cord injury. American Spinal Injury Association. *Spinal Cord.* 1997;35:266-74.
7. Aebi M, Etter C, Kehl T, Thalgott J. Stabilization of the lower thoracic and lumbar spine with internal spinal fixation system: indications, techniques, and the first results of treatment. *Spine (Phila Pa 1976).* 1987;12(6):544-51.
8. Benzel EC. Short segment compression instrumentation for selected thoracic and lumbar spine fractures: the short-rod/two claw technique. *J Neurosurg.* 1993;79:335-40.
9. Dolan EJ, Tator CH, Endrenyi L. The value of decompression for acute experimental spinal cord compression injury. *J Neurosurg.* 1980;53:749-55.
10. Maiman DJ, Larson SJ, Benzel EC. Neurological improvement associated with late decompression of

- the thoracolumbar spinal cord. *Neurosurgery.* 1984;14:302-7.
11. 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;87:239-46.
 12. Hugenholtz H, Cass DE, Dvorak MF, Fewer DH, Fox RJ, Izukawa DM, et al. High-dose methylprednisolone for acute closed spinal cord injury--only a treatment option. *Can J Neurol Sci.* 2002;29(3):227-35.
 13. Bracken MB, Shepard MJ, Collins WF, Holford TR, Young W, Baskin DS, et al. A randomized, controlled trial of methylprednisolone or naloxone in the treatment of acute spinal-cord injury. Results of the Second National Acute Spinal Cord Injury Study. *N Engl J Med.* 1990;322(20):1405-11.
 14. Steroids for acute spinal cord injury (Review). *The Cochrane Collaboration;* 2009.
 15. Gerhart KA, Johnson RL, Menconi J. Utilization and effectiveness of methylprednisolone in a population-based sample of spinal cord injured persons. *Paraplegia.* 1995;33:316-21.
 16. George ER, Scholten DJ, Buechler CM. Failure of methylprednisolone to improve the outcome of spinalcord injuries. *Am Surg.* 1995;61:659-63; discussion 663-4.
 17. Gerndt SJ, Rodriguez JL, Pawlik JW, et al. Consequences of high-dose steroid therapy for acute spinal cord injury. *J Trauma* 1997; 42:279-284.
 18. Bracken MB, Shepard MJ, Collins WF. A randomized, controlled trial of methylprednisolone or naloxone in the treatment of acute spinal-cord injury. Results of the Second National Acute Spinal Cord Injury Study. *N Engl J Med.* 1990;322(20):1405-11
 19. Denis F. Spinal instability as defined by the three-column spine concept in acute spinal trauma. *Clin Orthop Relat Res.* 1984;189:65-76.
 20. Denis F, Armstrong GW, Searls K, Matta L. Acute thoracolumbar burst fractures in the absence of neurological deficit (a comparison between operative and nonoperative treatment). *Clin Orthop Relat Res.* 1984;(189):142-9.
 21. Kostuik JP. Anterior spinal cord decompression for lesions of the thoracic and lumbar spine, techniques, new methods of internal fixation results. *Spine.* 1983;8:512-31.
 22. Bradford DS, McBride GG. Surgical management of thoracolumbar spine fractures with incomplete neurologic deficits. *Clin Orthop Relat Res.* 1987;218:201-16.
 23. Bradford DS, Akbarnia BA, Winter RB, Seljeskog EL. Surgical stabilization of fracture and fracture dislocations of the thoracic spine. *Spine.* 1977;2:185-96.
 24. Bohlman H.H. Bahniuk E. Raskulinecz G. Field G. Mechanical factors affecting recovery from incomplete cervical spinal cord injury: a preliminary report. *Johns Hopkins Med J.* 1979;145:115-25.
 25. Transfeldt EE, White D, Bradford DS, Roche B. Delayed anterior decompression in patients with spinal cord and cauda equina injuries of the thoracolumbar spine. *Spine.* 1990;15:953-57
 26. Mirza SK, Krengel 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; 359:104-14.
 27. Choe AS, Belegu V, Yoshida S, Joel S, Sadowsky CL, Smith SA, et al. Extensive neurological recovery from a complete spinal cord injury: a case report and hypothesis on the role of cortical plasticity. *Front Hum Neurosci.* 2013;7:290.
 28. Taniguchi LU, Pahl FH, Lúcio J ED, Brock RS, Gomes M QT, Adoni T, et al. Complete motor recovery after acute paraparesis caused by spontaneous spinal epidural hematoma: case report. *BMC Emergency Medicine BMC series ç open, inclusive and trusted* 201111:10. Available at: <http://bmccemergmed.biomedcentral.com/articles/10.1186/1471-227X-11-10>.
 29. Gorio A, Gokmen N, Erbayraktar S, Yilmaz O, Madaschi L, Cichetti C, et al. Recombinant human erythropoietin counteracts secondary injury and markedly enhances neurological recovery from experimental spinal cord trauma. *Proc Natl Acad Sci USA.* 2002;99:9450-5.
 30. Anthony S. Burns, Colleen O'Connell. The challenge of spinal cord injury care in the developing world. *J Spinal Cord Med.* 2012;35(1):3-8.
 31. McLain RF, Benson DR. Urgent surgical stabilization of spinal fractures in polytrauma patients. *Spine;* 2004:1646-1654.
 32. Vauzelle C, Stagnara P, Jouvinroux P. Functional monitoring of spinal cord activity during spinal surgery. *Clin Orthop Relat Res.* 1973;93:173-8.

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