Spinal Cord (1999) 37, 392–401

http://www.stockton-press.co.uk/sc

# *Clinical Case of the Month*

# Management of an unstable lumbar fracture with a laminar split

P Wing*,[1], M Aebi[2], F Denis[3], M Harris[4] and PR Meyer Jr[5]

*[1]Spine Program, Vancouver General Hospital, Vancouver, Canada; [2]McGill University Health Center, Montreal, Canada; [3]Twin Cities Spine Center, Minneapolis, Minnesota, USA; [4]Louisiana State University Medical Center, New Orleans, Louisiana, USA; [5]Midwest Regional Spinal Cord Injury Care System, Chicago, Illinois, USA*

This is a case report describing the injury sustained by a 36-year-old man injured in a motorcycle crash who sustained a fracture–dislocation of L2 upon L3, associated with a split in the lamina of L3. His neurologic lesion was T12 ASIA B: with a motor score of 52 but with preservation of sensory function (sensory score 96) in most parts of his lower extremities. He also suffered a lower extremity fracture. Imaging of the spine is presented showing a multiplanar fracture associated with translation and with a defect in the lamina that may be seen in certain AO type B or type C fractures, that may entrap the lumbar spinal nerve roots. Discussants of this case comment on the classification and clinical significance of this fracture pattern, and present their operative approaches, both for management of this particular fracture pattern and for any associated dural tear. The issues of steroid use and the place of rehabilitation are also discussed.

**Keywords:** paraplegia; spinal cord injury; vertebral fracture; laminar split; cauda equina lesion, dural tear

## Case presentation

A 36-year-old man was wearing a helmet and travelling at 30 kph when a truck passed closely in front of his motorcycle and he identified a collision as unavoidable. His passenger sustained a fractured femur; he sustained a compound comminuted fracture of his left distal tibia, and a mid-lumbar fracture with immediate loss of sensation of and ability to move his lower extremities. There was no loss of consciousness and his Glasgow Coma score at the scene was 15/15. There was no injury to chest or abdomen.

Generally in excellent health, our patient has worked in a number of occupations, most recently as a tour guide, which can be highly physical at times. He is a single parent of young children who usually live with him. His recreational interests are sports oriented. He lives in a community of 17 500 people, which has a 106-bed general hospital, and about an hour's drive away from a local centre that has a small rehabilitation unit. Travelling time by road to our regional spinal cord program is approximately 5 h.

Triage at the local hospital showed satisfactory vital signs in an alert and co-operative patient as well as the noted major injuries. Minor abrasions of the face and left upper extremity were seen. He had an evident neurologic deficit and the NASCIS-3 Solu-Medrol protocol was initiated 3 h after injury.

He was received at our Level 1 trauma hospital approximately 6 h after injury and admitted to an acute spinal cord injury unit. Assessment by the trauma surgeons revealed no other injury of note, and a FAST ultrasound scan was negative. The orthopaedic trauma surgeons identified the need for urgent management of the distal tibial fracture.

We found him to be an alert man, actively curious about his injury. Examination of his back revealed local swelling in the mid-lumbar region without skin damage, with quite marked tenderness of the mid-lumbar spinous processes. Using the international neurologic standards,[1] neurologic examination confirmed a bilateral T1 last normal motor level, with Grade 1 power of the left hip flexor and knee extensor, and no other lower extremity motor function. Last normal sensory level was L2 bilaterally, with impaired but present sensation to touch and pinprick distally to and including the sacral segments. His maximally impaired sensation was between L4 and S1 on the testable right leg (the left was splinted). Rectal tone was poor, no voluntary contraction was possible but rectal sensation was present. His neurological lesion was categorized as T12 last normal paraplegia, ASIA B, motor score 52.

*Correspondence: P Wing

X-ray and CT images are appended (see Figures 1–7). There were features of translational or rotational displacement, as well as features of bursting of the involved vertebra(e).

After assessment and decisions regarding management, this patient expressed a strong preference for acute management in this hospital with early return to his local community hospital(s).

## Questions and responses

A series of questions concerning classification and management was posed to the panel of experts. Edited responses are presented below, attributed by the names of the respondents.

1. *What type of fracture is this, and what are the critical features that lead you to choose this classification over closely related classifications? What are the implications of your classification?*

*PR Meyer*: According to the ASIA-sponsored New Universal Fracture Classification[2] this is a type C or 3 column fracture. The implications are that this fracture is grossly unstable and requires open reduction and internal fixation, as well as decompression.

*M Aebi*: I use the AO classification.[3] According to this classification this is a *C1.3* lesion of the segment L2–3.

*C indicates the injury type* In this specific-case, we deal with a rotational injury. This is expressed in the simple AP view where there is translation of the body and spinous processes of L2 to the left relative to L3. The horizontal cuts of the CT scan show the rotation of the body of L2 with respect to the comminuted body of L3. There is a wide open facet joint on the contralateral (right) side of the rotational direction at L2–3 underlining nicely the rotational component.

*1 of C1.3, indicates the major group within the C lesions* Group 1 is a burst fracture. In this specific case, this points to the major lesion in the anterior column (vertebral body and disc making up the entire column which is mostly loaded in compression).

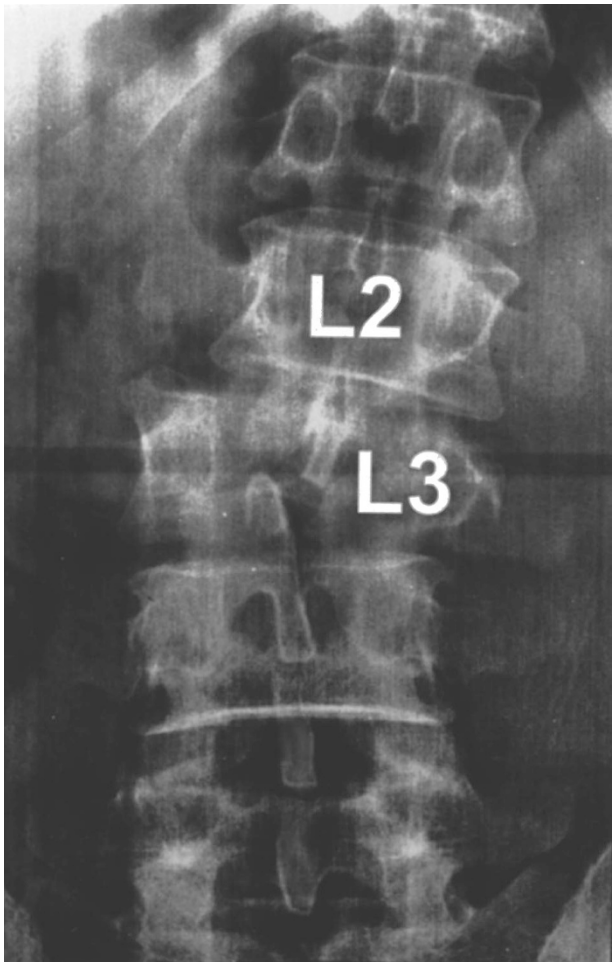
*3 of C1.3, indicates the subgroup, adding a qualifier to the anterior column lesion* This is a complete burst fracture since the cranial and caudal endplate are fractured. There are only three horizontal cuts of the burst body of L2 available, leaving me with some uncertainty. However, I believe we deal here with a burst fracture which is characterized by a dislocated reversed posterior wall fragment which is dislocated into the spinal canal. This would indicate that 'ligamentotaxis' will not work to reduce the dislocated posterior wall fragment and this reversed fragment needs to be mechanically removed, if the canal is to be cleared. The lesion of this patient is not a simple burst fracture of L3 because there is a major translational–rotational dislocation of L2 relative to L3 and 4. The dislocation happens within the L2–3 disc space and the burst body of L3 so that L2 is dislocated with the adherent fragments of L3.

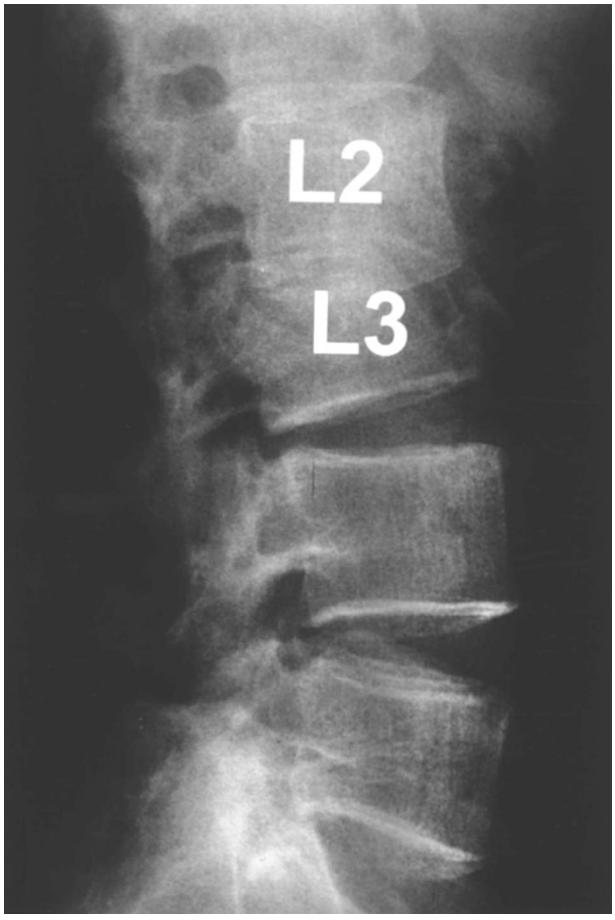
The AO classification is a pathomorphological classification based on mechanistic principles which are governed by the principal criteria of stability. A spine segment is stable when it can resist compression in the anterior column, distraction (elongation) in the posterior elements (tension banding system) and torsion (rotational forces). This man's fracture demonstrates failure of all these three components, namely failure to resist compression anteriorly, to resist distraction posteriorly (disruption) and, finally, to resist rotation. It is therefore a highly unstable injury. The extent of the failure of stability is very highly correlated with neurologic deficit: in this patient with an incomplete paraplegia.

*P Wing*: In their comprehensive classification of thoracic and lumbar injuries, Drs Magerl, Aebi and colleagues (who devised the AO classification) identify their type C injuries as fractures combining anterior compression (Type A characteristics) or ligamentous disruption and elongation (Type B) with rotation or shear. This fracture shows features of bursting and of shear. Like the burst fracture, it shows a wide laminar fracture extending into the spinous process with the potential for cauda equina entrapment. Unlike the simple burst fracture, examination shows significant swelling in the back from disrupted fascia and muscle. The type C fracture is identified as the most unstable fracture with the greatest likelihood of neurologic deficit and the potential for displacement in any direction.

*M Harris*: I agree with the two previous respondents: the fracture pattern illustrated represents a fracture-dislocation of L2–3. Utilizing the AO Spinal Injury Classification, it would be classified as a Type C injury. Regardless of the classification utilized, the important feature of this injury is the presence of a (post-traumatic) multi-planar deformity. This is readily appreciated on the AP and lateral X-rays provided. Of the two films reviewed, the AP film shows an angular deformity, which can only occur in the presence of a significant ligamentous injury. The lateral X-ray confirms the other plane of deformity, kyphosis, as well as the suggestion of a translational deformity. Together, these radiographic signs are strongly supportive of a very unstable fracture pattern.

*F Denis*: This spinal injury is an L2–3 fracture dislocation[4] of the flexion rotation type. It could be mistaken for a burst rotation for three reasons: there is some loss of the posterior height of the body, an apparently retropulsed fragment of bone back into the canal and a vertical laminar fracture. However, those three morphologic signs can be seen also in fracture dislocations. Although there is some loss of posterior height, one can see in the AP X-ray that the annulus fibrosus joining the right lower corner of the body of




**Figures 1 and 2** Initial AP and lateral X-rays of the lumbar spine. They show relative displacement anteriorly and to the left of the L2 body upon L3, and also show comminution of L3. Note also the spinous process malalignment seen on the AP

L2 to the right upper corner of L3 must be disrupted. There is significant evidence of disc disruption at that level. In a burst rotation in the same direction, the left upper corner of L3 would have followed the left lower corner of L2. In addition, the loss of alignment of the spinous processes is more frequently present in fracture dislocations than it is in burst fractures where the rotary displacement is primarily anterior. In fracture dislocations, it is both anterior and posterior.

The so-called retropulsed fragment is not really such. It is more of an avulsed piece of the posterior wall during the dislocation. That piece has relocated into the canal in a way that is not similar to the way it would be in a burst fracture. In the axial loading injury, the postero-superior corner of the vertebral body tends to break off the posterior wall and then pushes back into the canal still remaining attached to the mid-line annulus fibrosis. It is most frequently still covered by an intact posterior longitudinal ligament. In this case, the piece has flipped or rotated around and has a very different appearance than would be seen in a burst fracture as the posterior cortex of the body now faces anterior. The vertical laminar fracture has resulted from the splaying of the L3 pedicles occurring during dislocation. It becomes, in a way, a fracture of necessity whenever half of the body goes one way and the other half remains where it originally was. Vertical laminar fracture and cauda equina entrapment is to be expected in this case,[5] although the dural laceration is not likely to be as clean as it is in a typical burst. I would anticipate a higher likelihood of laceration or avulsion of some of the rootlets, although the neurological examination with good preservation of some sensation in the lower extremities suggests that most of the rootlets are still intact.

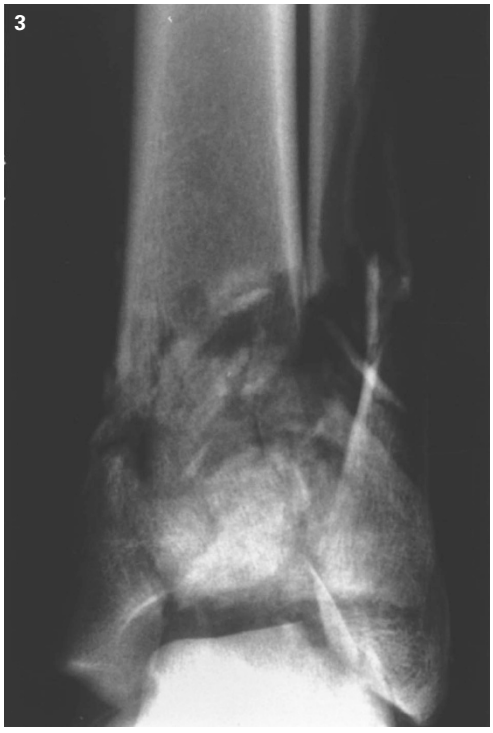
*2. Where is his neurologic lesion?*

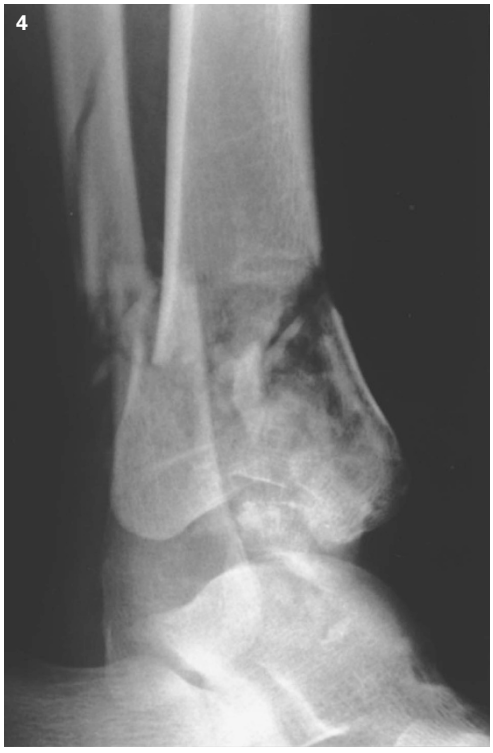
*M Harris*: Either a low conus lesion or a cauda equina lesion.

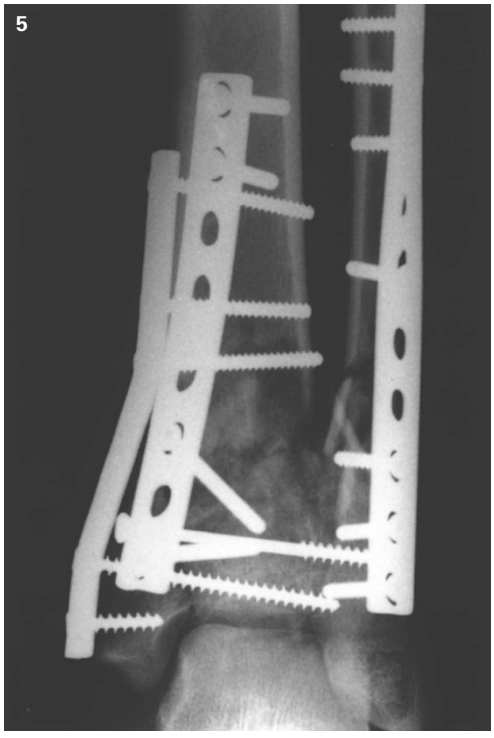
*P Wing*: In spinal injury, we always ask the question: are the identified bony and neurologic lesions compatible? While his patchy deficit appears to be

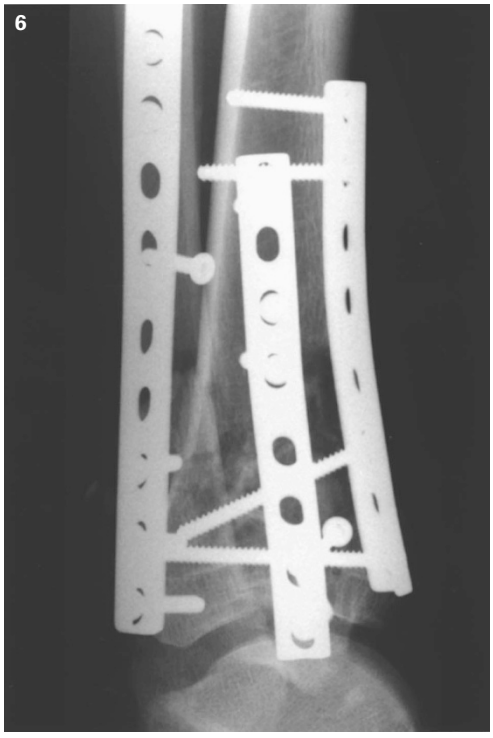
that of a cauda equina lesion, his sensory level appears to be higher than the bony lesion, possibly because of traction on the conus of the spinal cord.

*M Aebi*: This is a paraplegia, incomplete, at the level of T12/L1 with a lesion to the conus.




**Figures 3–6** AP and lateral views of the distal tibia and fibula, showing the comminuted compound fracture. A small non-contaminated wound was present, while distal vascularity was good

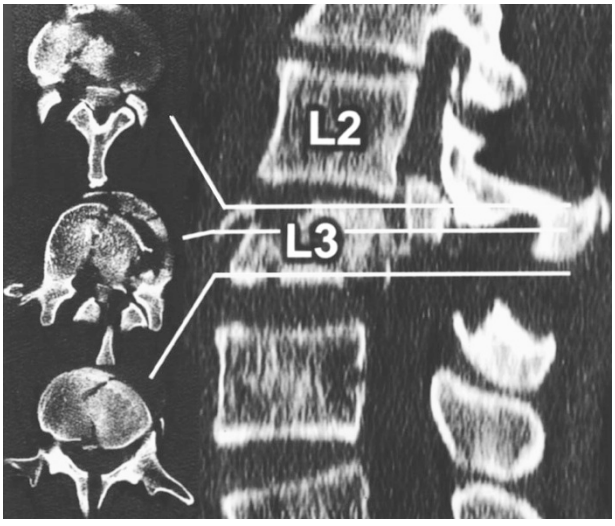



**Figure 7** Axial and reformatted CT of the lumbar spine showing the injury of L3. Features include: Anterior (vertebral body) comminution; Retropulsion of a bony fragment into the canal; Lateral translation of L2 on L3 and disruption of the posterior elements with a split spinous process

*F Denis*: His neurologic injury is a pure cauda equina injury. The fact that his maximally impaired sensation was between L4 and S1, strongly dispels the notion of a conus injury combined with cauda equina. At the L2–3 level, although the injured vertebrae are completely in front of the cauda equina, fracture dislocations often tug on the conus leading to conus plus cauda equina injuries.

*PR Meyer*: The picture as presented is a confusing one. I would interpret this as a 'mixed', Brown-Sequard-like peripheral nerve cauda equina lesion. With the perineal sensation, and the presence of L2/3 function in the hip flexors and quads on the left there is a concern that there could be a second lesion (L1 and 2 come off the cord at T10/T11 bony level, with L3 arising at the upper border of T12). It is a little early for edema to produce an ascending conus lesion, though that is a possibility. With the last normal sensory level at L2, this would indicate 'root escape'. Regardless, the confusing nature of the neurological findings often suggests an opportunity for some neurological recovery.

*3. What are your (operative and/or nonoperative) options for management of this fracture?*

*F Denis*: Several considerations arise in managing this fracture-dislocation: (i) a very unstable situation exists and, just for this reason, it should be approached from posteriorly first. (An anterior approach would be highly likely to lead to some oblique distraction due to the rotation, in which case the structural struts may end up situated obliquely to the axis of the spine); (ii) the cauda equina entrapment is, by itself, a strong contraindication to doing an anterior fusion or any manipulation of the spine until full restitution of dural continuity; (iii) whereas in burst fractures, the retropulsed fragment is somewhat difficult to remove from posteriorly, it is much easier to do so in fracture dislocations. The bony fragment is more easy to move around and less fixed by the annulus fibrosis; and (iv) be aware that in a fracture dislocation pedicle screw fixation from L1–3 in the absence of a strong cross-linking system is likely to fail in lateral shear.

Insofar as the spinal operation itself is concerned, I would first proceed with a meticulous exposure of L2–4 with particular care not to apply pressure on the spinous process of L3. This would be likely to push directly over the nerve roots against the so-called avulsed posterior wall fragment. I would then go on to perform a progressive exposure of the dural tear, taking very meticulous care not to damage the tiny rootlets that will be encountered on the way. If necessary for purposes of approach of the canal, I might consider doing a left vertical laminotomy on L3 so as to use the open door technique as I described allowing full visualization of the dural tear and entrapped rootlets. A fracture dislocation is quite different, however, from the burst fracture and I anticipate that the laminar fracture is completely disrupted and fairly obvious whereas it is not so in a burst fracture. Instrumentation and fusion would occur only after full decompression and dural repair. My choice would be for pedicle screw fixation from L2–4 with cross-linking at the top and the bottom of the rods so as to create a rectangle.

*M Harris*: An important point to consider once the diagnosis has been made, is the need to limit the number of levels fused in the lumbar spine. This is essential in the treatment of fractures of L2 and caudal. The priority of limiting the number of levels of the fusion may also influence the surgeon's decision-making with respect to performing a combined anterior and posterior procedure.

Once the diagnosis of a fracture-dislocation (or Type C injury) has been made, my surgical approach to this injury pattern generally includes posterior stabilization. This is independent of whether or not an anterior procedure is required. The main thrust for using posterior instrumentation in this injury is to neutralize the multi-planar deformity. Although anterior instrumentation continues to evolve and improve, it has not yet reached the level of versatility nor does it independently have the corrective capacity that posterior instrumentation provides.

My approach to this fracture would be a posterior approach spanning L1–4. It would include a careful exposure and laminectomy of the L2–3 level, as the triad of a lumbar burst fracture with a neurological deficit and a laminar fracture suggests the strong likelihood of a dural tear and exposed neural

elements.[6,7] Once the exposure was completed and the dura repaired with the roots returned to their pre-morbid location, pedicle screws would be placed bilaterally in L1, 2 and 4. Ideally at least one pedicle screw would be placed into the fractured L3 vertebra (from the CT it looked possible to screw the right pedicle). The pedicle screw within the fractured vertebra seems to assist in any attempts at reduction as well as providing some additional mechanical strength to the construct.

With direct exposure of the repaired dura, the retropulsed fragments would either be tapped back into the body of L3 or removed posteriorly. This approach should provide a thorough decompression.[8]

Upon completion of the procedure we should achieve satisfactory sagittal and coronal alignment as well as an effective direct decompression. Once the patient recovered from his surgical insult, upright unsupported lateral X-rays would be obtained to assess whether anterior column reconstruction was necessary for stability and/or to protect the pedicle screws from breakage via repetitive loading. Additionally, if the intra-operative assessment of the decompression was sub-optimal, a post-operative CT scan would allow a secondary evaluation of the residual canal/root compromise and help facilitate the decision for the secondary anterior decompression and reconstruction. If a corpectomy is indicated, I prefer to utilize autogenous iliac crest; however, one can also utilize bone bank bone or the *avant garde* approach of a cage with the corpectomized bone.

*PR Meyer*: I recommend posterior stabilization, using one of the following: (i) Hook-rod fixation (two above, one above, one below/or pedicle screw, with distraction), plus fusion; (ii) Pedicle screws one above, one below the fracture, (with distraction) and fusion; or (iii) Harrington rods with sublaminar wires and Edward sleeves, plus fusion (distraction . . . with care). This is probably the least desirable posterior option because of the bone in the canal.

A postoperative CT-myelogram should be performed looking for evidence of distraction of the fragments. The pre-operative CT reveals that ligamentotaxis is likely to fail because the posterior smaller fragments in the canal are rotated, indicating that the posterior longitudinal ligament is disrupted. Therefore there is less of a chance for improvement in fragment realignment, so an anterior procedure is required, including the following: (i) Decompression of L-3; (ii) Humeral/femoral allograft with cancellous bone in the marrow (from the fractured-decompressed vertebra or 'tricortical' iliac crest graft); (iii) Anterior plate/screws with fixation one level above, one level below and to the graft in the center; and (iv) Followed in each case with a TLSO (thoracolumbosacral orthosis).

*M Aebi*: Treatment of such a lesion is a stable internal fixation with a proper decompression which needs to be performed through an anterior approach, when we accept that the major fragment in the spinal canal is a reversed posterior wall fragment. The indication for surgery in this patient is the highly unstable osteoligamentous injury and the incomplete paraplegia, which may partially be related to the persistent bony mechanical compression. The fact that the motor deficit occurred instantly after the trauma, however, speaks for a direct lesion to the neural structures.

The surgical options form a two-step procedure: (1) Posterior approach, reduction and stabilization with a short pedicular device which allows correction in proper lordosis, such as the USS system. With this, it is certainly possible to fix this injury from L1 – 3. Because of the rotational component of the injury, we would prefer to cross-link the vertical stabilization system to avoid collapse in the frontal plane; (2) Since this injury is in a critical area it is not recommended to open the canal to try to manipulate this fragment through a posterior approach, although it is possible to decompress it through a postero-lateral approach. To obtain the best decompression and also provide anterior column support, a second, anterior, step would be necessary. The defect can be filled either with an iliac tricortical graft, fibular strut or with a cage filled with the cancellous fragments of the vertebral body. Additional anterior stabilization may not be necessary due to the strength of the posterior stabilization.

*P Wing*: In our centre, surgical decisions for treatment of a spinal fracture with deficit are based on neurologic, mechanical and patient factors. Can we make a difference to the patient's short and long term outcome with a degree of certainty that makes the risks of surgery worthwhile? In this case, the strongest surgical indication is for mechanical restoration of a fracture that is very unstable, and if treated conservatively, will likely drift into a more severe deformity. The neurological concern is that his laminar split may be associated with root entrapment and an attempt to correct the deformity without first releasing the roots could result in increased deficit. The patient factors to be considered here are not unusual, in that we wish him to be painfree and into his rehabilitation as soon as possible. Correction of the displacements can be most readily achieved through use of a posterior instrumenting approach, with initial laminectomy to release any entrapped roots in the laminar split. The strength of a short fusion and instrumentation can be enhanced by use of the heavier screws and an offset laminar hook at the lowest level to protect the pedicle screw.

*4. Would you at this stage continue steroids and if so, for how long?*

*PR Meyer*: Yes, until the 23 h mark, or until the surgeon wants to discontinue them. If continuing, we would use dexamethasone.

*M Harris:* NASCIS-3[9] showed that if the steroids were initiated within 3 h, only 24 h of solumedrol was necessary (for cord lesions). I would probably utilize the solumedrol for 48 h.

*M Aebi*: I would use steroids until a formal decompression has been done.

*P Wing*: Not normally. There is as yet no evidence that the NASCIS-3 protocol has any effect on cauda equina injuries. I might not stop the steroids in the absence of a relative contraindication, because we have seen little evidence of harm from the short-term dose regimen used.[9,10]

*F Denis*: I would continue steroids at this stage in spite of the fact that we are not dealing with spinal cord but rather with cauda equina. It has been my experience in those cases that the rootlets swell up significantly and that in spite of the best dural repair, two problems are likely to occur: (i) the cauda equina tends to swell up and it is not unusual for it to feel somewhat tight after closure of the dura. The steroids decrease the swelling of the rootlets; (ii) in spite of best technique the likelihood of arachnoiditis is fairly significant due to the combination of radicular inflammation, swelling, intradural bleeding and radicular manipulation. It is my opinion that steroids play a role in minimizing the phenomenon. I would personally continue regular dosage IV steroids, or a Medrol 'Dose Pak', on patient resumption of feeding for 2 weeks to 10 days.

5. *Please include the timing of your treatment modalities*

*F Denis*: Although I am no longer a general orthopaedic surgeon, I still respect the need to deal emergently with a compound fracture of the ankle. However, because of the minimal contamination observed, I would try to persuade the trauma team to proceed with both repair of the ankle and spinal decompression at the same time. The relationship between neurologic recovery and timing of that decompression is well demonstrated in the dog. I believe that at 6 h after injury there is still a chance to save the rootlets.

*M Harris*: Urgent but not emergent. Since this patient was going to the OR for his open tibia fracture, I would most likely do the spinal procedure at the same setting. If the anterior procedure was determined to be necessary for either the stability issue or the residual canal compromise, I would probably plan to do it 5–10 days after the index procedure.

*M Aebi*: To optimize the chance of recovery of this incomplete lesion with an obvious mechanical compression (although the persistent mechanical compression may not fully be responsible for the neurologic deficit) we would operate on him as soon as possible. This means as soon as his vital signs are stable, most probably even before the distal tibia fracture is operated on or during the same anesthesia. While there is no proof that this early surgery will give a better outcome, there are experimental data suggesting this, as does my clinical experience (which is uncontrolled and unrandomized).

*PR Meyer*: In my opinion, there are only two emergent/urgent surgery indications after spinal injury. These are the incomplete neurological injury with an unreduced dislocation, or the incomplete lesion with evidence of deteriorating neurology. This, however, does not or should not preclude the surgeon from using his discretion. Included in this is 'staging' the posterior and anterior procedures, or doing the posterior first, and then turning the patient into the lateral decubitus position, and immediately following with the anterior decompression and stablilization.

*P Wing*: The open ankle fracture needs treatment early to minimise the risk of infection. The spine should be operated on within 24–48 h unless the patient begins to lose more function, in which case a more urgent approach should be considered.

6. *After posterior decompression there is a 3 cm longitudinal rent with edematous cauda equina nerve roots herniating through it. The spine is now stabilized and reasonable hemostasis achieved. What is your local practise regarding management of these herniated roots? (repair, use of sealants or patches, drains)*

*M Harris*: The laminectomy would be extended sufficiently cephalad and caudad to identify the full extent of the tear. A primary repair of the dura would be attempted and if found to be impossible, a portion of the lumbo-dorsal fascia would be utilized to patch the defect. All roots would be replaced within the reconstructed dural tube. Accurate documentation of avulsed or lacerated nerve roots is helpful both for clinical prognosis and medico–legal issues. I have had very little experience with sealants. I rarely drain spinal incisions anymore, especially if I have a residual CSF leak.

*P Meyer*: Primary closure of the dura if possible, after 'reducing' the herniated filaments of the cauda equina which is difficult, but can be done. Use a fascial graft over the dural tear if unable to repair primarily, followed by a fat graft, before the application of the hardware and a 'lateral' fusion.

*F Denis*: Without having a specific description of the dural tear, it is difficult to say exactly what would be

my favored approach. In most burst fractures, the laminar entrapment of the cauda equina is seen in conjunction with a very clean vertical tear of the dura which is easy to repair. In fracture dislocations, this tear is more often irregular and may sometimes require use of dural patches.

*M Aebi*: If the dura has clean cut edges, then we repair it. Otherwise, we reposition the roots, covering the dural defect with surgicel which we try to seal to the dura. No drain. Flat position or even Trendelenburg position in the first 24–48 h.

*P Wing*: Once the roots were exposed, I would tip the head of the table down somewhat as they then often reduce, after which I may repair a clean tear, or for an untidy rent may patch with fascia, or seal with fat and Tisseal (reg) tissue adhesive.

7. *The patient is keen to proceed and is a compliant rehabilitation participant. However, he wants to get home to his family as soon as possible. Would you insist that he proceed through the rehabilitation course and look for an early discharge, or would you recommend that he have some form of abbreviated program and continue through the resources of his local hospital(s)?*

*PR Meyer*: The average acute hospital stay (without complications or major additional injury concerns), is 9–13 days for the paraplegic. Because of the concern for the fractured tibia (which by the way looks excellent on the available radiograph), I would probably extend the patient's initial hospital stay, during which time we can instruct in bowel, bladder and skin care, and basic transfer techniques. Then he should go home for a period of time (wheelchair, non-weight-bearing on the left) after which he can enter a rehabilitation program through the resources of his local hospital. But, he must be informed that if re-employment is an option, he will likely require vocational and social services.

*F Denis*: I believe that rehabilitation of this patient is going to be very important. I would recommend an abbreviated local program and then arrange for continued physical therapy and rehabilitation closer to his family.

*M Aebi*: For his rehabilitation program, I would insist that the patient has a proper rehabilitation course to learn about the possible complications of paraplegia which occur when rehabilitation principles are neglected. I would not give this patient an abbreviated program and rely on resources in local hospitals. Paraplegic patients require specialized expertise both in primary acute and in rehabilitation management. It usually does not pay off to abbreviate the rehabilitation process, and the more structured and more rigorous is this rehabilitation process, the higher is the chance that the patient will not end up with the classical complications of neglect.

*M Harris*: I am a firm advocate that the rehabilitation after a SCI is every bit as important as the surgical procedure. I would therefore strongly encourage the patient and his family to participate in the rehabilitation program which has the most experience and expertise in SCI rehabilitation rather than the one which may be closer to home.

*P Wing*: We do not have access to a half-way house that would best enable this man to return to the community early. The principles of rehabilitation in our community would be to initiate much of the education in the acute unit, to prioritise the SCI goals in our spinal cord injury rehabilitation program, and continue physical conditioning near home.

8. *Given his neurologic pattern, what prognosis (and upon what evidence) would you give for (a) motor recovery and (b) recovery of bladder and sexual function?*

*PR Meyer*: Fair to good. Motor, unpredictable, but I would hedge and say fair, believing that the injury is below the conus medullaris, and only within the cauda equina. Sensory, moderate prognosis for improvement. None of the exam findings stated the presence or absence of perianal pin/pain sensation. If pain is present, there is a better chance that both bowel and bladder function may return to some extent. Sexual function is more complicated. With perineal sensation, and sexual centres in both the brain and the T12 areas preserved, he may have return of function, but I have found it less rewarding, and not predictable (unless one has more neurological data on which to base the answer than what we have here).

*F Denis*: Provided that no further injury be observed in the course of the surgery, I would expect this patient to become a community ambulator with crutches and bilateral AFO's early on and possibly having enough return of his ankle dorsiflexion to get rid of that AFO during his rehabilitation. I would expect his bladder function to return after a period of intermittent catheterization. I would have somewhat more reservations about the return of his sexual function.

*M Aebi*: (a) This patient has a chance to have recovery of L1, 2 and 3 motor function to a certain extent. Most probably the worst roots like L4 and S1 will not recover. These are probably the roots described as lying free in the decompression area. There may be a slight hope that this patient will be able, under rigorous rehabilitation, to stabilize his hips and knees and become able to stand, at least, with braces. This would probably also allow him to do some walking with crutches and braces; (b) This is a

conus lesion with poor sphincter tone and impaired bladder function. The chance that he will recover this function is very small.

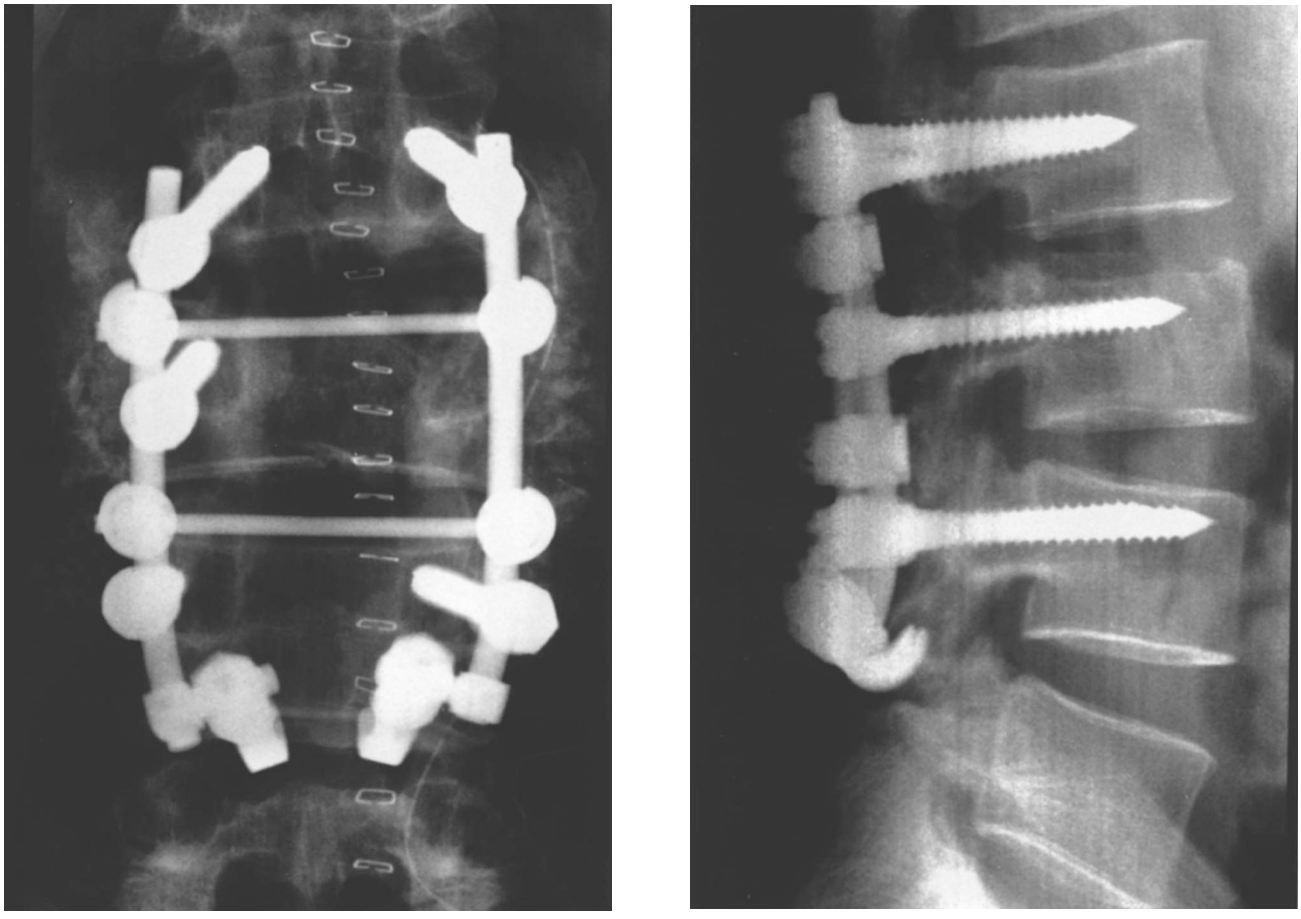
*P Wing*: I expect some motor function but cannot say how much; I anticipate bladder function to return.

## What was done

Within 24 h of injury, and immediately following surgical treatment of the tibial fracture, spine surgery was undertaken. The laminar split was to the left of midline; there were entrapped rootlets. There was a dural split with inadequate dura to repair primarily. 7 mm pedicle screws were placed in L2, 3 and 4 on the right, but only L2 and 4 on the left. The lamina of L3 was resected, as was the left L2 inferior facet and subsequently the left L2 pedicle. The left rod was placed, and the offset laminar hook at L4; distraction on the left to correct the scoliosis was followed by use of an impactor to tamp the fragment back into the body. The right rod and offset hook was then placed. Two crosslinks were used (Figures 8 and 9). Gelfoam, fat graft and Tisseal were used over the dural defect, and a suction drain placed in the paravertebral gutter. The patient mobilized quickly without wound problems and by 4 weeks after injury was an active participant in his rehabilitation program at our centre, having accepted the need for the full program.

## Discussion

This case provides an example of the need to detail every component of fracture morphology, while not losing sight of the overall care plan for the person with SCI. We know that laminectomy has no place in the management of spinal cord injury. However, little has been written about laminectomy after major cauda equina root entrapment in laminar fracture fragments, yet decompression may allow improved recovery,[8] and less pain. Dural tears are associated with 8–25% of burst fractures,[6,11] requiring attention if operating on the patient with an incomplete lesion, even if only for mechanical indications. We do not have extensive follow-up on this man and cannot describe his neurologic recovery, but have outlined



**Figures 8 and 9** Postoperative views of the lumbar spine, showing the USS Instrumentation with pedicle screws in both sides of L2 and L4, unilaterally in L3. Offset hooks under the lamina of L4 are thought to 'protect' the L4 pedicle screws while conserving motion with a shorter fusion. Posterolateral bone graft can be seen on the AP view

the immediate management steps and the reasoning behind them.

## References

1 Maynard FM *et al*. International Standards for Neurological and Functional Classification of Spinal Cord Injury. American Spinal Injury Association, Chicago, Illinois, 1996.
2 Meyer P *et al*. Proposed new spine fracture classification, 22nd Annual Meeting of the American Spinal Injury Association, Seatlle, WA, April 22–24, 1996.
3 Magerl F *et al*. A comprehensive classification of thoracic and lumbar injuries. *European Spine Journal* 1994; **3:** 184–201.
4 Denis F. The three column spine concept and its significance in the classification of acute thoracolumbar spinal injuries. *Spine* 1983; **8:** 817–831.
5 Denis F, Burkus JK. Diagnosis and treatment of cauda equina entrapment in the vertical lamina fracture of lumbar burst fractures. *Spine* 1991; **16** (8 Suppl): S433–S439.
6 Cammisa Jr FP, Eismont FJ, Green BA. Dural laceration occurring with burst fractures and associated laminar fractures. *J Bone Joint Surg [Am]* 1989; **71:** 1044–1052.
7 Miller CA, Dewey RC, Hund WE. Impaction fracture of the lumbar vertebrae with dural tear. *J Neurosurg* 1980; **53:** 765–771.
8 Garfin SR, Mowery CA, Guerra Jr J, Marshall LF. Confirmation of the posterolateral technique to decompress and fuse thoracolumbar spine burst fractures. *Spine* 1985; **10:** 218–223.
9 Bracken MB *et al*. Administration of methylprednisolone for 24 or 48 hours of tirilazad mesylate for 48 hours in the treatment of acute spinal cord injury. Results of the Third National Acute Spinal Cord Injury Randomized Controlled Trial. National Acute Spinal Cord Injury Study. *JAMA* 1997; **277:** 1597–604.
10 Wing PC, Nance P, Connell D, Gagnon F. Risk of avascular necrosis following NASCIS-2 Steroid protocol. *Spinal Cord* 1998; **36:** 633–636.
11 Keenen TL, Antony J, Benson DR. Dural tears associated with lumbar burst fractures. *J Orthop Trauma* 1990; **4:** 243–245.