Radiological findings correlate with neurological deficits but not with pain after operatively treated sacral fractures
An 11-year follow-up study of 28 patients

Aron Adelved¹,², Anna Tötterman³, Johan C Hellund⁴, Thomas Glott⁵, Jan Erik Madsen¹,⁶, and Olav Roise¹,⁶

¹Department of Orthopaedics, Division of Surgery and Clinical Neuroscience, Oslo University Hospital, Ullevaal; ²Department of Orthopaedics, Akershus University Hospital, Lørenskog, Norway; ³Department of Orthopaedics, Karolinska University Hospital, Stockholm, Sweden; ⁴Department of Radiology, Oslo University Hospital, Ullevaal; ⁵Department of Spinal Cord Injury and Multitrauma Unit, Sunnaas Hospital, Nesodden; ⁶Institute of Clinical Medicine, Faculty of Medicine, University of Oslo, Norway.
Correspondence: aron.adelved@medisin.uio.no
Submitted 13-11-15. Accepted 14-01-07

Background and purpose — Neurological deficits and pain are common after displaced sacral fractures. However, little is known about the association between the long-term clinical outcomes and radiological findings. We examined the long-term radiological findings and their correlations with lumbosacral pain and neurological deficits in the lower extremities after surgery for sacral fractures.

Methods — 28 consecutive patients with operatively treated displaced sacral fractures were followed for mean 11 (8–13) years. Sensorimotor impairments of the lower extremities were classified according to the American Spinal Injury Association (ASIA). Pain was assessed using a visual analog scale (VAS). All patients underwent conventional radiographic examination and CT, and the images were scrutinized for nonunion, residual displacement, narrowing of the sacral foramina, and post-foraminal encroachment of the L5 and S1 nerves.

Results — There was residual displacement of ≥ 10 mm in 16 of the 28 patients. 26 patients had narrowing of 1 or more neural root foramina in L5-S4. 8 patients reported having no pain, 11 had pain only in the lumbosacral area, and 9 had pain in combination with radiating leg pain. Statistically significant correlations were found between narrowing of the sacral foramina and neurological deficits in the corresponding dermatomes. Significant correlations were also found between post-foraminal encroachment of L5 nerves and both sensory and motor deficits. No correlations were found between pain and radiological findings.

Interpretation — Pathological radiological findings are common 11 years after operatively treated displaced sacral fractures. Sacral foraminal and L5 post-foraminal bony encroachments were common findings and correlated with neurological deficits. However, lumbosacral pain did not correlate with radiological sequelae after fracture healing.

High-energy trauma with displaced sacral fracture is frequently associated with concomitant injuries to the intrapelvic soft tissue structures, including the lumbosacral plexus (Huitinen 1972, Denis et al. 1988, Majeed 1992). These injuries may cause considerable morbidity (Pohlemann et al. 1994, Tornetta and Matta 1996, Tötterman et al. 2006). However, little is known about which factors determine long-term clinical outcome in these patients, or what may explain the progression of neurological symptoms observed in a small proportion of patients (Adelved et al. 2012). Pelvic malunions and non-unions have been put forward as prognostic factors for impaired long-term outcome (Matta et al. 1996, Mears and Velyvis 2003, Oransky and Tortora 2007), but long-term structural changes of the sacrum after fracture healing have not been explored.

Our primary aim was to assess long-term radiological findings after surgically treated displaced sacral fractures. In addition, we wanted to assess whether pathological radiological findings, including bony structural changes of the sacrum, may contribute to neurological dysfunctions of the lower extremities or to the occurrence of pelvis-related pain.

Patients and methods
From July 1996 through October 2001, 39 consecutive patients with operatively treated displaced sacral fractures were prospectively registered at Oslo University Hospital, Ullevaal. Tötterman et al. (2006) conducted a 1-year follow-up study of these patients, where 32 patients were available for follow-up.

In the present long-term study, 28 of these 32 patients were available for clinical and radiological follow-up. Of the 4 patients who were lost to follow-up, 1 died, 2 declined to participate, and 1 patient was excluded due to a complete spinal
cord injury with paraplegia. Of the patients included, 1 was excluded from the neurological examination in the study protocol due to hemiparesthesia after a head injury, but fulfilled the rest of the protocol. Mean follow-up time was 11 (8–13) years, female-to-male ratio was 7:21, and mean age was 43 (26–67) years. The mechanisms of injury included 20 motor vehicle accidents, 7 falls from heights, and 1 crush injury. Pelvic ring injuries were classified according to the AO/OTA fracture classification (Fracture and dislocation compendium. Orthopaedic Trauma Association Committee for Coding and Classification 1996). In 26 cases, the sacral fractures were part of a vertical shear pelvic ring disruption, classified as AO/OTA type-C injuries, and 2 patients had H-shaped sacral fractures. The sacral fractures were further classified according to Denis et al. (1988) as zone I (n = 1), zone II (n = 20), and zone III (n = 7). All fractures were treated operatively: 22 with open reduction and internal fixation, 12 with concomitant sacral laminectomy, and 6 with closed reduction and percutaneous SI-screw fixation (Adelved et al. 2012). Additional anterior plating was performed in 14 patients. The functional outcome of these patients after a mean of 11 years post injury has already been reported (Adelved et al. 2012).

**Radiological examination**

At follow-up, all patients underwent conventional radiographic examination of the pelvis and the lumbar spine. This included pelvic anteroposterior, inlet, and outlet views according to standard radiographic protocol (Bontrager and Lampignano 2005).

Residual displacement (RD) was defined as a cephalad or posterior displacement of the hemipelvis and sacrum, and was graded as < 10 mm or ≥ 10 mm. In the outlet views, cephalad residual displacement (CRD) was recorded by measuring the difference in height between the top lateral prominences of the 2 sacral transverse process elements (STPE). In 4 cases, PRD was determined using the difference in height between the ischial spines in the inlet views, since in these images the posterior pelvic borders were not sufficiently visualized in the films.

All patients were also examined with a 64-channel multidetector computer tomography (MDCT). The CT images were scrutinized for nonunion, ankylosis, osteoarthritis (OA) in the L5-S1 facet joints and the SI-joints, and heterotopic ossification. Fracture healing was confirmed by the presence of bridging trabecular bone across the fracture lines on CT.

To identify any bony entrapment of the nerves, all 3 sets of 2D CT images were used, following each nerve from the spinal canal to the point where the nerve was peripheral to the sacrum. Narrowing of the neural foramina were recorded and then divided into 4 categories: 1: no narrowing; 2: less than 50% narrowing; 3: more than 50% narrowing; and 4: total occlusion of the foramen (Figure 3).

L5 and S1 nerves were then followed in their post-foraminal course and any changes in their path were recorded—i.e. displacement of the nerves by pathological bony structures and
thus diversion from the assumed anatomical course or entrapment/overgrowth of the nerves by bony structures (Figure 4). The S2-S4 nerves were not as readily identifiable post-foraminally as L5 and S1, and they were therefore not included in the post-foraminal assessment. Finally, any narrowing of the spinal canal was recorded, using the midline sagittal images evaluating the inner tapering AP diameter.

A radiologist who was experienced in pelvic traumatology (JCH) reviewed all the radiographs and CT scans. He was blinded regarding the clinical information.

**Clinical examination**

Sensorimotor impairments were classified according to the American Spinal Injury Association (ASIA) score (Maynard et al. 1997, Adelved et al. 2012). The clinical assessment focused on neurological function in the lower extremities and the perineum.

Pain was assessed using a visual analog scale (VAS) ranging from 0 to 10, where zero represented no pain and 10 the most severe pain. The patients were asked to grade their average pain specifically in the lower back and posterior pelvic area. When present, radiating pain to the lower extremities was also recorded. Peripheral pain in the lower limbs was not considered when there were sequelae after lower extremity injuries.

**Statistics**

Due to small sample size and skewed distribution, non-parametric methods (namely, Spearman correlation coefficients) were used and p-values of ≤ 0.05 were considered to be statistically significant. We used PASW Statistics 18 software.

**Ethics**

The study was reviewed and approved by the Regional Committee for Medical and Health Research Ethics, Region South-East Norway. All patients signed an informed consent document at follow-up.

### Results

**Radiological findings**

Residual displacement of more than 10 mm was observed in 16 patients: 6 with CRD, 3 with PRD, and 7 with a combination of CRD and PRD. In the remaining 12 patients with residual displacement of less than 10 mm, 10 had a combination of CRD and PRD, 1 had a pure CRD, and the other a pure PRD (Table 1). The average CRD of ≥ 10 mm was 15 (10–28) mm and the average PRD of ≥ 10 mm was 19 (11–35) mm. The average CRD of < 10 mm was 4 mm and the average PRD of < 10 mm was 6 mm (Table 2).

Nonunions were seen in only 2 patients, both of them occurring in the anterior pelvic ring. These patients were operated with both anterior and posterior fixation: in 1 of them all the implants were intact, while both anterior and posterior implant failure was noted in the other patient. All sacral fractures were healed.

---

**Figure 3.** Considerable distortion and narrowing of the left S1 neural foramen, marked with an arrow. Note the unaffected contralateral foramen for comparison.

**Figure 4.** a. Bony encroachment of the left L5 nerve post-foraminally (E). The contralateral L5 is unaffected in its post-foraminal path (N). b. A few slices more distally. The left L5 is dislocated laterally from its anatomical path (D), running through the area with fracture sequelae, with topographical changes of the adjacent bony surface. (A) shows the unaffected contralateral L5 nerve running in its anatomic path.
26 of 28 patients had narrowing of 1 or more neural foramina from L5 to S4. Post-foraminal bony encroachment of the L5 nerve was observed in 22 patients (Table 3).

In 9 patients, the implants had been removed due to posterior pelvic pain. In the remaining 19, breakage or loosening of implants was seen in 8 cases (involving anterior implant failure in 3 cases, posterior implant failure in 3, and both anterior and posterior implant failure in 2 cases).

### Clinical Findings

Neurological assessment in 1 patient was not possible due to sensory impairments in the right side of the body after a head injury. In the remaining 27 patients, 26 had neurological deficits in the lower extremities. 12 had minor to moderate sensory deficits affecting the L5-S4/S5 dermatomes. The L5-S1 dermatomes were mainly affected. Combined sensory deficits and muscle weakness were present in 14 patients. The deficits were unilateral in 9 and bilateral in 14. Uni- or bilaterality of neurological deficits could not be assessed in 3 patients who had undergone unilateral amputation below the knee: 2 initially due to crush injuries and 1 due to sequelae after severe foot and ankle fractures 5 years post injury.

8 patients reported having no pain at the long-term follow-up. Of the remaining 20 patients, 11 reported pain limited to the lumbosacral area while 9 reported both lumbosacral pain and radiating pain involving the L5-S2 dermatomes. However, 15 patients had scores of \( \leq 2 \) on the VAS scale, indicating slight or no pain. Of the remaining 13 patients, 5 had VAS scores between 4 and 7, and 5 had scores of \( \geq 8 \).

### Correlation of Clinical and Radiological Findings

Narrowing of the sacral foramina in S1-S3 had a statistically significant correlation with neurologic deficits in the corresponding dermatomes. Similarly, there was a correlation between narrowing of the sacral canal and neurological deficits at the S2 level. Post-foraminal involvement of the L5 nerves was significantly correlated with both sensory and motor deficits in the corresponding dermatomes (Table 4). No significant correlations were found between any of the radiological findings and posterior pelvic pain (Table 5).
Malunion is imprecisely defined after pelvic ring injuries, but most authors have considered displacement in any dimension of less than 10 mm, with no gross rotational malalignment, to be a satisfactory result (Tornetta and Matta 1996, Lindahl et al. 1999). The consequences and treatment of sacral malunions after surgery are poorly documented. Most reports have been case reports or small case series, and late surgical decompression of the neural roots in these patients has seldom been described in the literature (Alexander et al. 2013).

There have been a few publications describing clinical manifestations and surgical treatment of pelvic malunions and nonunions (Pennal and Massiah 1980, Matta et al. 1996, Mears and Velyvis 2003, Oransky and Tortora 2007). In these series, the initial treatment of the fractures consisted of either nonoperative treatment or external fixation in the majority of cases, resulting in inadequate posterior pelvic stability (Lindahl et al. 1999, Kanakaris et al. 2009). A substantial number of the patients in these reports had major pelvic ring deformities with considerable morbidity related to the deformity.

Our series is therefore not directly comparable to these studies. All our patients were initially treated with internal fixation, and at the long-term follow-up, none of them had major deformities or nonunions in the posterior pelvic ring. As reported in our previous publication, 26 of our 28 patients were able to walk independently (Adelved et al. 2012).

Numerous authors have considered a residual cephalad displacement of more than 10 mm to be a poor prognostic factor (McLaren et al. 1990, Matta and Tornetta 1996, Matta et al. 1996, Tornetta and Matta 1996, Lindahl and Hirvensalo 2005) and have recommended accurate reduction of a displaced vertically unstable sacral fracture.

Other studies have shown conflicting results. Nepola et al. (1999) presented the results of 33 patients with type-C vertical shear injuries, treated with external fixation or nonoperatively, with residual displacement ranging from 2 mm to 52 mm. They found no correlation between residual displacement and functional outcome, including pain. Pohlemann et al. (1996) reported the results of 30 patients with type-C, vertically unstable pelvic ring fractures, treated with internal and/or external fixation. 18 had slight pain or no pain, 28 had residual displacement of < 10 mm, and only 8 had good or excellent outcome.

Displaced unstable sacral fractures are frequently associated with neurological lesions (Huittinen 1972, Gibbons et al. 1990, Majeed 1992). The configuration and size of the anterior sacral foramina may be altered, either due to insufficient reduction, or later due to callus formation during the bone-healing process. In the present study, narrowing of the sacral foramina was observed in several patients and correlated with neurological deficits. As opposed to sacral foramina, structural changes in the L5 neural foramina did not appear to have any effect on neurological deficits. However, post-foraminal encroachment of the L5 nerves correlated well with neurological deficits in the corresponding dermatomes.

### Table 4. Correlation between radiologically verified narrowing of the neural foramina and neurological deficits in corresponding dermatomes; n = 27

| Neural foramen level | Spearman's correlation coefficient | p-value |
|----------------------|------------------------------------|---------|
| Narrowing of the sacral central canal | 0.42 | 0.03 * |
| L5 | | |
| Sensory | 0.22 | 0.1 |
| Motor | 0.08 | 0.6 |
| S1 | | |
| Sensory | 0.30 | 0.03 |
| Motor | 0.15 | 0.3 |
| S2 | | |
| Sensory | 0.50 | < 0.001 |
| Motor | 0.45 | 0.001 |
| S4 | | |
| Sensory | 0.22 | 0.1 |
| Motor | | |
| L5-post-foraminal boney encroachment | | |
| Sensory | 0.31 | 0.03 |
| Motor | 0.35 | 0.01 |

*significance only at S2-level.

### Table 5. Correlation between radiological findings and lumbosacral pain; n = 28

| L5-S1 | Spearman's correlation coefficient | p-value |
|-------|------------------------------------|---------|
| Disc space narrowing | 0.09 | 0.7 |
| Facet joint osteoarthritis | 0.28 | 0.2 |
| Facet joint ankylosis | 0.27 | 0.2 |
| SI-joint | | |
| Ankylosis | 0.01 | 1.0 |
| Osteoarthritis | 0.05 | 0.8 |
| Presence of implants | 0.29 | 0.1 |
| Residual displacement ≥ 10 mm | | |
| Cephalad | 0.14 | 0.5 |
| Posterior | 0.005 | 1.0 |

### Discussion

There are no established validated protocols for radiological quantification and measurement of pelvic deformities and other sequelae after pelvic ring disruptions (Lefaivre et al. 2012). For measurement of residual displacement, we used conventional radiographs, since they are readily available and are most commonly used by most orthopedic surgeons in outpatient clinical settings. We used the methods described by Mears and Velyvis (2003), but modified the landmarks for the CRD measurements on the outlet films; we were mainly interested in the sacral fracture and any displacements of the sacral bone. In addition, a few patients sustained sequelae after iliac wing fractures, resulting in inaccurate measurements when the tops of the iliac wings were used as reference landmarks. We therefore used the sacral transverse process elements of the lateral masses as CRD landmarks.
tomes. Post-foraminally, impingement of the L5 nerve may be caused by several structures, formed by the lumbosacral ligament (Nathan et al. 1982). Also, it runs distally along the anterior aspect of the lateral mass between the SI-joint and the sacral foramina. This path is usually the main fracture area in a vertically unstable sacral fracture, and the L5 nerve may thus be avulsed or stretched by bony fragments in cases of severe fracture displacement (Huittinen 1972, Denis et al. 1988). Later, during fracture healing, bony encroachment caused by callus or ectopic bone formation may contribute to further neurological dysfunction. However, the exact cause of L5 pathology observed in our patients cannot be determined from this study.

Several studies have shown limited long-term recovery of the neurological injuries after pelvic fractures (Matta and Saucedo 1989, Tornetta and Matta 1996, Rommens and Hessmann 2002). To our knowledge, changes in the sacral topography—including changes in the shape and diameter of the anterior sacral foramina after fracture healing—have not been studied in detail. In the present study, with no CT scans from previous follow-ups, comparison with earlier radiographs and recording of changes over time was not possible. Thus, we cannot determine whether the neurological deficits were caused or deteriorated by gradual changes in the topography of the bony structures, although we found a close relationship between boney impingement/encroachment of neural structures and neurological deficits.

We observed a substantial proportion of patients with osteoarthritis of the facet joints, the lumbosacral junction, and the SI-joints. Degenerative processes in the lumbosacral spine, including facet joint OA and L5-S1 disc space reduction, are often reported in epidemiological and cadaveric studies (Eubanks et al. 2007, Kalichman et al. 2010). These studies also suggest that the association between these findings and low back pain is variable. There are no epidemiological data on the prevalence of OA in SI-joints. Radiological changes in the SI-joints have been suggested to contribute to low back pain (Hodge and Bessette 1999). Yet, other authors have shown normal variations in the appearance of the SI-joints, depending on factors such as sex and age (Vogler et al. 1984, Faffia et al. 1998). In these studies, degenerative changes were frequent with increasing age in individuals without back pain. Our results back up these studies. 15 out of 28 of our patients had no pain or only slight pain in the posterior pelvic area (VAS ≤ 2). For the remaining 13 with moderate to severe pain, no correlations were found between pain and any of the radiologic findings, including residual displacement.

There were some methodological differences between our study and the studies mentioned: all our patients were treated with internal fixation and we used a VAS scale to quantify pain. Despite these differences, our results support the results of the studies indicating a lack of association between residual displacement and pain. Our results indicate that foraminal architecture and bony changes around the fracture area may play a greater role in the long-term outcome than the overall pelvic alignment.

The present study had some limitations. The number of patients was low, so the results of the statistical analyses should be interpreted with caution. In addition, due to limited access to postoperative and 1-year follow-up radiographs, comparison of the 11-year radiological results with the earlier images was not possible. The strength of the study lies in the long-term follow-up and high response rate, and also the thorough neurological and radiological assessments.

In summary, pathological radiological findings are common after operatively treated displaced sacral fractures. Sacral foraminal and L5 post-foraminal encroachments correlated with neurologic deficits. Lumbosacral pain did not correlate with radiological sequelae after fracture healing, including residual displacement.

This work was supported by research funds from the Sophies Minde Ortopedi AS Foundation, Oslo, Norway.

No competing interests declared.
Kalichman L, Kim D H, Li L, Guermazi A, Hunter D J. Computed tomography-evaluated features of spinal degeneration: prevalence, intercorrelation, and association with self-reported low back pain. Spine J 2010; 10 (3): 200-8.

Kanakaris N K, Angoules A G, Nikolau V S, Kontakis G, Giannoudis P V. Treatment and outcomes of pelvic malunions and nonunions: a systematic review. Clin Orthop 2009; (467) (8): 2112-24.

Lefaivre K A, Slobogean G, Starr A J, Guy P, O’Brien P J, Macadam S A. Methodology and interpretation of radiographic outcomes in surgically treated pelvic fractures: a systematic review. J Orthop Trauma 2012; 26 (8): 474-81.

Lindahl J, Hirvensalo E. Outcome of operatively treated type-C injuries of the pelvic ring. Acta Orthop 2005; 76 (5): 667-78.

Lindahl J, Hirvensalo E, Böstman O, Santavirta S. Failure of reduction with an external fixator in the management of injuries of the pelvic ring. Long-term evaluation of 110 patients. J Bone Joint Surg (Br) 1999; 81 (6): 955-62.

Majeed S A. Neurologic deficits in major pelvic injuries. Clin Orthop 1992; (282): 222-8.

Matta J M, Saucedo T. Internal fixation of pelvic ring fractures. Clin Orthop 1989; (242): 83-97.

Matta J M, Tornetta P I. Internal fixation of unstable pelvic ring injuries. Clin Orthop 1996; (329): 129-40.

Matta J M, Dickson K F, Markovich G D. Surgical treatment of pelvic nonunions and malunions. Clin Orthop 1996; (329): 199-206.

Maynard F M Jr, Bracken M B, Creasey G, Ditunno J F Jr., Donovan W H, Ducker T B, Garber S L, Marino R J, Stover S L, Tator C H, Waters R L, Wilberger J E, Young W. International standards for neurological and functional classification of spinal cord injury. American Spinal Injury Association. Spinal Cord 1997; 35 (5): 266-74.

McLaren A C, Rorabeck C H, Halpenny J. Long-term pain and disability in relation to residual deformity after displaced pelvic ring fractures. Can J Surg 1990; 33 (6): 492-4.

Mears D C, Velyvis J. Surgical reconstruction of late pelvic post-traumatic nonunion and malalignment. J Bone Joint Surg (Br) 2003; 85 (1): 21-30.

Nathan H, Weizenbluth M, Halperin N. The lumbosacral ligament (LSL), with special emphasis on the "lumbosacral tunnel" and the entrapment of the 5th lumbar nerve. Int Orthop 1982; 6 (3): 197-202.

Nepola J V, Trenhaile S W, Miranda M A, Butterfield S L, Fredericks D C, Riemer B L. Vertical shear injuries: is there a relationship between residual displacement and functional outcome? J Trauma 1999; 46 (6): 1024-9.

Oransky M, Tortora M. Nonunions and malunions after pelvic fractures: why they occur and what can be done? Injury 2007; 38 (4): 489-96.

Pennal G F, Massiah K A. Nonunion and delayed union of fractures of the pelvis. Clin Orthop 1980; (151): 124-9.

Pohlemann T, Bosch U, Gänsslen A, Tscherne H. The Hannover experience in management of pelvic fractures. Clin Orthop 1994; (305): 69-80.

Pohlemann T, Gänsslen A, Schellwald O, Culemann U, Tscherne H. Outcome after pelvic ring injuries. Injury (Suppl 2) 1996; 27: B31-B38.

Rommens P M, Hessmann M H. Staged reconstruction of pelvic ring disruption: differences in morbidity, mortality, radiologic results, and functional outcomes between B1, B2/B3, and C-type lesions. J Orthop Trauma 2002; 16 (2): 92-8.

Standring S, Gray H, Borley N R. Gray’s anatomy: the anatomical basis of clinical practice. In: Churchill Livingstone Elsevier, Edinburgh, 2008; (40th ed.): 724-8.

Tornetta P I, Matta J M. Outcome of operatively treated unstable posterior pelvic ring disruptions. Clin Orthop 1996; (329): 186-93.

Tötterman A, Glott T, Madsen J E, Røise O. Unstable sacral fractures: associated injuries and morbidity at 1 year. Spine 2006; 31 (18): E628-35.

Vogler J B, III, Brown W H, Helms C A, Genant H K. The normal sacroiliac joint: a CT study of asymptomatic patients. Radiology 1984; 151 (2): 433-7.