Sagittal balance is more than just alignment: why PJK remains an unresolved problem

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Abstract

Background: The durability of adult spinal deformity surgery remains problematic. Revision rates above 20% have been reported, with a range of causes including wound infection, nonunion and adjacent level pathology. While some of these complications have been amenable to changes in patient selection or surgical technique, Proximal Junctional Kyphosis (PJK) remains an unresolved challenge. This study examines the contributions of non-mechanical factors to the incidence of postoperative sagittal imbalance and PJK after adult deformity surgery.

Methods: We reviewed a consecutive series of adult spinal deformity patients who required revision for PJK from 2013 to 2015 and examined in their medical records in detail.

Results: Neurologic disorders were identified in 22 (76%) of the 29 PJK cases reviewed in this series. Neurologic disorders included Parkinson’s disease (1), prior stroke (5), metabolic encephalopathy (2), seizure disorder (1), cervical myelopathy (7), thoracic myelopathy (1), diabetic neuropathy (5) and other neuropathy (4). Other potential comorbidities affecting standing balance included untreated cataracts (9), glaucoma (1) and polymyositis (1). Eight patients were documented to have frequent falls, with twelve cases having a fall right before symptoms related to the PJK were noted.

Conclusion: PJK is an important contributing factor to the substantial and unsustainable rate of revision surgery following adult deformity correction. Multiple efforts to avoid PJK via alterations in surgical technique have been largely unsuccessful. This study suggests that non-mechanical neuromuscular co-morbidities play an important role in post-operative sagittal imbalance and PJK. Recognizing the multi-factorial etiology of PJK may lead to more successful strategies to avoid PJK and improve surgical outcomes.

Keywords: Adult spinal deformity, Adult scoliosis, Proximal junctional kyphosis, PJK

Background

Surgical treatment of adult spinal deformity has progressed substantially over the past ten years. There have been significant advances in decision-making, medical management and surgical technique [1, 2]. These improvements in evaluation and treatment have broadened the applicability of adult deformity surgery and lead to more reproducible clinical benefit based upon health-related quality of life (HRQOL) scores [3, 4].

Despite these positive developments, the durability of adult spinal deformity surgery remains problematic.

Revision rates above 20% have been reported, with a range of causes including wound infection, nonunion and adjacent level pathology [5–7]. While some of these complications have been amenable to changes in patient selection or surgical technique, Proximal Junctional Kyphosis (PJK) remains an unresolved challenge.

The initial description of PJK in the pediatric literature was an increased sagittal angulation, without structural failure, at the upper aspect of a fusion construct [8]. At present, the term is applied much more widely to describe any failure or loss of alignment above an instrumented segment [9, 10]. This may result from adjacent level compression fracture, spondylolisthesis or fixation failure [6–10]. In general, this has been viewed as a
consequence of poor bone quality, over-aggressive deformity correction or inadequate fixation.

PJF has been the focus of intense scrutiny, with multiple studies proposing mechanical solutions including adaptations for osteoporotic bone and in particular specific sagittal alignment targets [11, 12]. Unfortunately, none of these mechanical solutions have effectively decreased the rate of PJF. The role of this study is to examine the contributions of non-mechanical factors to the incidence of postoperative sagittal imbalance and PJF after adult deformity surgery.

**Methods**

After receiving Institutional Review Board Approval, we reviewed a consecutive series of adult spinal deformity patients who required revision for PJF from 2013 to 2015 and examined in their medical records in detail. Standard demographic data including age, gender, smoking status, height and weight were collected. Indications for the index surgery, specifics of the index surgery including upper instrumented vertebra fixation, time to PJF diagnosis, time to PJF surgery, mode of failure. Medical records were extensively evaluated for preoperative comorbidities; specifically for preoperative neurologic disorders and other pathologies that may affect standing balance.

**Results**

From 2012 to 2014, 245 patients underwent surgical correction of their adult spinal deformity at our institution. A true incidence of PJF will be difficult to determine as (1) some patients presenting at our institution with PJF had their index surgery performed elsewhere and (2) some of the patients who had their index surgery at our institution could have developed PJF and had surgery elsewhere.

Twenty-nine cases of PJF requiring revision were identified (Table 1). Of these 9 (31 %) were males and 10 (34 %) were smokers. Mean age was 64.4 years. Mean BMI was 29. kg/m². Neurologic disorders were identified in 22 (76 %) of the PJF cases reviewed in this series. Neurologic disorders included Parkinson’s disease (1), prior stroke (5), metabolic encephalopathy (2), seizure disorder (1), cervical myelopathy (7), thoracic myelopathy (1), diabetic neuropathy (5) and other neuropathy (4). Other potential comorbidities affecting standing balance included untreated cataracts (9), glaucoma (1) and polymyositis (1) (Table 2). Eight patients were documented to have frequent falls, with twelve cases having a fall right before symptoms related to the PJF were noted. Seventeen cases used an assistive device such as a cane, crutches or a walker and required a wheelchair. One patient had 5 co-morbid conditions affecting standing balance, two had 4 co-morbid conditions, four had 3 co-morbid conditions, nine had 2 co-morbid conditions and ten had only one co-morbid condition (Table 3).

**Discussion**

Proximal Junctional Kyphosis was first identified in 1999 [8], and was initially described as a radiographic finding with limited clinical relevance [13, 14]. This sanguine assessment was short lived, as subsequent reports have documented the frequent need for revision surgery [5, 6] as well as the occurrence of catastrophic failures, termed Proximal Junctional Failure (PJF) [9, 10, 15, 16]. The reported increase in PJF was coincident with several major changes in treatment paradigm. Adult deformity surgery became more common in older patients, and more aggressive correction was undertaken using osteotomies and rigid instrumentation. Studies have highlighted these factors and examined their etiologic role in PJF and PJF [10, 17, 18].

Deformity surgeons clearly recognize PJF and PJF as important challenges, but often regard these complications as mechanical problems for which there should be a straightforward mechanical solutions. As osteoporosis is commonly identified as an etiology of PJF, surgeons have pursued options to offset poor bone quality. Strategies have included prophylactic medical treatment of low bone density, strengthening proximal instrumented and adjacent vertebral levels with cement injection. Other strategies have included decreasing rod rigidity, and softening the transition to unfused levels using hooks rather than screws [11, 19, 20]. Another major focus has been on selection of fusion levels and restoration of sagittal alignment [12, 18, 21, 22]. Studies have advocated both more aggressive and less aggressive deformity correction. Maruo et al. report that restoration of normal sagittal alignment protected against PJF, and that greater than 30-degree increase in lumbar lordosis was a significant risk factor for PJF. [18] As increase in lumbar lordosis is generally the mechanism by which normal sagittal alignment is restored, these observations appear contradictory.

The findings of the present study suggest that our failure to control the rate of PJF may be related in part to the narrow focus on mechanical factors. This study demonstrates that 76 % of patients with PJF after spinal deformity correction have co-morbidities that adversely affect standing balance, regardless of alignment. These include neuromuscular disease, history of cerebrovascular accident, cervical myelopathy and neuropathy. All of these conditions may contribute to an inability to re-balance through unfused segments after deformity correction. This phenomenon is clearly recognized with substantial neurologic impairment such as patients with Parkinson’s disease [23], but has not been clearly defined in those patients with less severe neurologic impairment.
| Case. No. | Age/ Sex | Smoker | BMI | Indication for Index Surgery | Index Surgery | UIV Fixation | Time to PJK diagnosis | Mode of Failure | PJK surgery | Fall prior | Assistive device | CCMI | Other co-morbidities |
|-----------|----------|--------|-----|-------------------------------|---------------|--------------|----------------------|----------------|-------------|-----------|------------------|------|---------------------|
| 1         | 68/F     | Yes    | 40.9| Kyphoscoliosis                | PSF T10 to Pelvis, TLIF L3-L4 | bilateral pedicle screws | 8 months | Fracture of T9-T10 with cord compression | T9-T10 laminectomy, extension of fusion T4-T11 | No | No | 11 | None |
| 2         | 64/M     | Yes    | 19.7| Stenosis                      | PSF L3 to L5 | bilateral pedicle screws | 18 months | Fracture of L3 | PSO L3, PSF T11 to pelvis | Yes | Cane | 11 | CVA, Loss of reflexes below knee |
| 3         | 58/M     | No     | 33.9| Multilevel stenosis           | PSF L3 to Pelvis | bilateral pedicle screws | 17 months | Fracture of L3 | AIF L5-S1, Ponte osteotomies, PSF T10 to pelvis | Yes | No | 4 | CSM post ACDF |
| 4         | 63/F     | No     | 25.9| Multilevel stenosis           | PSF L2 to L5 | bilateral pedicle screws | 21 months | Compression of L2 with complete loss of L1-L2 interspace | Extension to T10 | No | Wheelchair | 10 | CVA, Cauda equina requiring emergent decompression, Diabetic neuropathy |
| 5         | 65/F     | Yes    | 34.9| ASF L4-S1, PSF T10 to Pelvis  | PSF T10 to Pelvis | bilateral pedicle screws | 11 months | Compression Fracture T11 | Extension of fusion to T3 | Yes | Walker | 9 | Diabetic neuropathy, Frequent falls, post bilateral TKA, ORIF L ankle |
| 6         | 70/F     | No     | 25.7| Kyphoscoliosis                | ASF, PSF T10 to Pelvis | bilateral pedicle screws | 12 months | Compression Fracture T9 | Extension of fusion to T3 | No | Cane | 7 | Cataracts |
| 7         | 52/F     | Yes    | 25.6| Degenerative scoliosis, stenosis | PSF, L2 to sacrum | bilateral pedicle screws | 64 months | Kyphosis at L1-L2 impingement of screws into disc space | TLIF L1-L2, PSF L1-L2 | Yes | Crutches | 8 | Diabetic neuropathy |
| 8         | 64/M     | No     | 31.0| Flatback S/P L3-L5 PSF        | ASF L5-S1, PSF T9 to Sacrum | bilateral pedicle screws | 18 months | T8-T9 Lis thesis | Extension of fusion to T2 | Yes | Walker | 8 | CVA, Neuropathy, Cataracts (removed), CSM post laminectomy, CSM post laminectomy |
| 9         | 57/F     | No     | 30.5| Flatback S/P L3-L5 laminectomies | PSF T11 to Pelvis | bilateral pedicle screws | 25 months | Compression Fracture T9 - T10 | Extension of fusion to T3 | No | No | 9 | TIAs, Diabetic neuropathy, Cataracts, Frequent falls, post bilateral TKA, multiple foot surgeries |
| 10        | 60/M     | Yes    | 19.3| Kyphoscoliosis                | ASF L4-S1, PSF T10 to Pelvis | bilateral pedicle screws | 82 months | Fracture of T9, T8-T9 spondylolisthesis | PSF T4 to T12 | Yes | Walker | 9 | CVA, Sensory neuropathy, Glaucosa, Frequent falls, post multiple revisions of bilateral TKA |
| 11        | 58/M     | No     | 34.7| Degenerative scoliosis, stenosis | PSF T10 to Pelvis, TLIF L5-S1 | bilateral pedicle screws | 1 month after 1st PJK | Pull out of claw construct fracturing T4 to T8 laminae | Extension of Fusion T2 to T12 | | | | |

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| Case  | Gender/Age | Previous Procedure | Diagnosis | Fusion Level | Treatment Details | Effects | Complications |
|-------|------------|---------------------|-----------|--------------|-------------------|--------|---------------|
| 12    | 75/F       | No                  | Degenerative Scoliosis | S/P L2-L3 PDSF | PSF T10 to Pelvis, TLIF L5-S1 | bilateral pedicle screws | 27 months | Screw pull out | Extension to T4 | Yes | No | 5 | None |
| 13    | 62/F       | Yes                 | Flatback deformity | S/P L2-L5 S/P | PSF L2 to S1 | bilateral pedicle screws | 12 months | Fracture L1 | Extension to T10 | No | Cane | 6 | Tremors, Multiple foot surgeries |
| 14    | 69/F       | Yes                 | Degenerative Scoliosis, Stenosis | S/P L3 to S1 | Extension of fusion L1 to S1 | bilateral pedicle screws | 1 month | L1-L2 listhesis | Extension from T10 to S1 | No | No | 7 | None |
| 15    | 57/F       | No                  | Degenerative Scoliosis, Stenosis | S/P L1 to S1 | PSF L2 to S1 | bilateral pedicle screws | 92 months | L1-L2 listhesis | Extension from T10 to S1 | No | Cane | 5 | Parkinson's disease |
| 16    | 63/F       | No                  | Adjacent segment degeneration S/P L3 to S1 | PSF | PSF T9 to L3 | bilateral pedicle screws | 7 months | Posterior lysis of T9 and T10 | Removal of instrumentation, PSF T4 to L3 | Yes | Cane | 8 | Cataract, CSM post C3 to T1 ACDF |
| 17    | 73/F       | No                  | Scoliosis | PSF T6 to Sacrum | PSF T6 to Sacrum | bilateral hooks | 80 months | Fracture T6 | Removal of instrumentation, PSF T4 to L3 | No | No | 9 | Cataract |
| 18    | 61/M       | Yes                 | Scoliosis | PSF T8 to Sacrum | PSF T8 to Sacrum | bilateral hooks | 11 months | Fracture T8 | Removal of instrumentation, PSF T4 to Pelvis | No | Walker | 7 | Polymyositis |
| 19    | 72/M       | No                  | Scoliosis | PSF T11 to L3 | PSF T11 to L3 | bilateral pedicle screws | 23 months | T10-T11 listhesis | Removal of instrumentation, PSF T8 to T11 | No | Walker | 6 | CSM post laminoplasty, Frequent falls |
| 20    | 78/F       | No                  | Scoliosis | PSF L1 to S1 | PSF L1 to S1 | bilateral pedicle screws | 14 months | Fracture T12 | Extension of Fusion to T8 | No | No | 7 | Metabolic encephalopathy, Cataract |
| 21    | 71/F       | Yes                 | Degenerative Scoliosis | S/P L2-L3 | PSF L2-L3 | bilateral pedicle screws | 45 months | L1-L2 collapse and localized scoliosis | Extension to T10 | No | Cane | 10 | Cataract (removed), Cervical osteomyelitis with cord compromise |
| 22    | 75/F       | No                  | Scoliosis | PSF T4 to Pelvis | PSF T4 to Pelvis | bilateral hooks | 2 months | Hook pull-out with T4-T6 laminar fractures | Extension to T2 | No | No | 10 | Cataract |
| 23    | 69/M       | No                  | Post-laminectomy instability | ASF L3 to S1, PSF L2 to S1 | bilateral pedicle screws | 1 month | Compression Fracture of L2 with screw pullout | Removal of instrumentation, PSF T10 to L1 | No | No | 6 | Diabetic neuropathy |
| 24    | 55/F       | No                  | 38.0 | PSF L2 to S1 | Compression Fracture T10 | 9 months | Compression Fracture T10 | Extension to T3 | Yes | No | 8 | CSM post ACDX |
| No | Age | Gender | Duration of Symptoms | Diagnosis | Treatment | Outcome | Comorbidities |
|----|-----|--------|----------------------|-----------|-----------|---------|--------------|
| 25 | 70/F | No     | 35.5                 | Stenosis  | PSF L2-L5 | Compression of L2 | Metabolic encephalopathy, Cataract, Frequent falls |
| 26 | 62/F | No     | 21.4                 | Adjacent segment stenosis | ASF L2-L5 | Fracture T10 | CSM post ACDF, Neuropathy, Frequent falls |
| 27 | 73/F | No     | 20.6                 | Scoliosis | PSF T10 to Pelvis | T10 compression fracture | Mild cognitive impairment, Benign thoracic tumor S/P excision |
| 28 | 65/F | No     | 21.1                 | Scoliosis | PSF T11 to S1 | T10-T11 listhesis, nonunion L5-S1 | Seizures, Eye surgery |
| 29 | 33/M | Yes    | 26.7                 | Scoliosis | PSF L1 to L4 | Compression of T12 | Chronic dropfoot |

*PSDF posterior spinal decompression and fusion, PSF posterior spinal fusion, ASF anterior spinal fusion, TLIF transforaminal lumbar interbody fusion, CVA cerebrovascular accident, CSM cervical spondylotic myelopathy, ACDF anterior cervical discectomy and fusion, TKA total knee arthroplasty, ORIF open reduction internal fixation, THA total hip arthroplasty, DT delirium tremens*
Beyond potential neurogenic causes of standing imbalance, other factors such as visual impairment, vestibular dysfunction and severe muscular deconditioning also impact balance and gait [24, 25]. Visual impairment was noted in 40% of PJK cases and more than a single potentially relevant co-morbidity was noted in more than 66% of cases. While these findings do not implicate neuromuscular disease as the direct cause of PJK, they certainly suggest a multi-factorial etiology.

The mechanisms by which these non-mechanical risk factors contribute to PJK are not well defined, and probably do not represent a unique common pathway. In some instances, such as patients with neuropathy or central neurologic deterioration, an impaired feedback loop may limit the ability to compensate appropriately after mechanical realignment. In essence, the patient’s brain does not properly register the “improved alignment” as determined by radiographic assessment. In other cases, lack of appropriate sensory feedback may result in accelerated proximal segment degeneration, akin to the appearance of a Charcot joint. In patients with severe deconditioning, muscular support may be inadequate regardless of mechanical alignment.

It is not completely clear how best to apply these observations in clinical practice. Our case series methodology cannot provide a relative risk assessment for any of the individual co-morbid conditions, and to-date no diagnostic test has been developed to quantify a global risk for post-operative standing imbalance or PJK. It is also unknown as to whether these risks can be modified by pre-operative interventions such as balance training, in the same way that treatment of osteoporosis is thought to reduce the risk of post-operative vertebral fracture or screw pull-out.

Weaknesses of this study include firstly the case series methodology. As some of the patients had their index procedure elsewhere, we do not have an accurate denominator to assess the incidence of PJK in the primary cohort. This series is also relatively small, so that the relative risk of the various co-morbidities cannot be effectively compared. Despite these weaknesses, this study clearly supports the role of concomitant neuromuscular disease in the development of post-op standing imbalance and PJK. The data does not provide a specific threshold at which surgery should be withheld, but certainly emphasizes the importance of including an assessment of associated neuromuscular disease in pre-operative planning and shared decision-making.

Spine surgeons have devoted a great deal of time and effort to defining optimal sagittal alignment, but sagittal balance is more than just alignment. Dubousset outlined the many interactive systems that contribute to ambulation and stated, “good alignment is preferable in order to obtain a good balance, but it is not sufficient” [26]. Understanding and avoiding PJK requires that we move beyond the one-dimensional view that finding an ideal sagittal alignment, softening the transition at the proximal aspect of the instrumented segment, or improving the adjacent bone strength will solve the problem of PJK. Thinking about PJK more broadly is a step in the right direction.

**Conclusions**

PJK is an important contributing factor to the substantial and unsustainable rate of revision surgery following adult deformity correction. Multiple efforts to avoid PJK via alterations in surgical technique have been largely unsuccessful. This study suggests that non-mechanical neuromuscular co-morbidities play an important role in post-operative sagittal imbalance and PJK. Recognizing the multi-factorial etiology of PJK may lead to more successful strategies to avoid PJK and improve surgical outcomes.

**Competing interests**

SDG - is an employee of Norton Healthcare; received research grants from Norton Healthcare; holds patents from and receives royalties from Medtronic. Medtronic provided funds directly to database company. No funds were paid directly to Individual or Individual’s Institution 01/2002 to 09/2009. Nuvasive provided funds directly to database company. No funds are paid directly to Individual or Individual’s Institution 06/2012-04/2015. MPK has nothing to disclose.

| Table 2 | Frequency of co-morbid conditions that can affect balance |
| --- | --- |
| Co-morbid condition | Frequency |
| Prior stroke | 5 |
| Metabolic encephalopathy | 2 |
| Parkinson’s disease | 1 |
| Seizures | 1 |
| Polymyositis | 1 |
| Diabetic Neuropathy | 5 |
| Neuropathy | 4 |
| Cataract | 9 |
| Glaucoma | 1 |
| Myelopathy | 8 |
| Frequent falls | 8 |

| Table 3 | Number of co-morbid conditions that can affect balance |
| --- | --- |
| Frequency |  |
| None | 3 |
| One | 10 |
| Two | 9 |
| Three | 4 |
| Four | 2 |
| Five | 1 |
LYC - is an employee of Norton Healthcare; Member, Editorial Advisory Board, Spine and Spine Journal; Institutional Review Board Member, University of Louisville Institutional Review Board; Global Evidence Advisory Board Member, Medtronic 2012, 2013; Research Committee Member, Scoliosis Research Society; received research grants from Norton Healthcare and AO Spine paid directly to Scoliosis Research Society 2010–2011; receives research funds from the Orthopedic Research and Educational Fund, 2013-present; received research grant from Norton Healthcare, 2013; receives Scoliosis Research Society Research Funding, 2013-present; receives research funding from the Norton Healthcare James R. Petersdorf Fund 2015; received funds for travel for Study Planning Meetings from Orthopedic Educational Research Fund, Department of Defense, Association for Collaborative Spine Research and the Center for Spine Surgery and Research of the University of Southern Denmark; received funds for travel for Annual required Continuing Education for Institutional Review Board Members, University of Louisville Institutional Review Board; Honoraria for participation in Review Panels National Institutes of Health, Children’s Tumor Foundation, and Global Evidence Advisory Board, Medtronic. Medtronic provided funds directly to database company. No funds were paid directly to Individual or Individual’s Institution 01/2002 to 09/2009. Nuvasive provided funds directly to database company. No funds are paid directly to Individual or Individual’s Institution 06/2012-04/2015.

Authors’ contributions

SDG - conception and design, analysis and interpretation of data, drafting of the manuscript, acquisition of data, MPC - acquisition of data, analysis and interpretation of data, critical revision of the manuscript, LVC - acquisition of data, analysis and interpretation of data, critical revision of the manuscript. All authors read and approved the final manuscript.

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