The Biomechanics of Spondylolysis, Part 2 by Dr. Arthur C. Croft

In part I of this two-part series (Sept. 23 issue), I described a rear-impact motor-vehicle crash in which a man was rear-ended by a police cruiser and pushed into an SUV. His vehicle sustained moderate-plus damage to its front and rear portions, while the police car and SUV sustained only minor damage. The car driver, a 30-year-old man, complained immediately of low back pain and was transported to the hospital. In time he underwent bilevel lumbar fusion at L3-4 and L4-5 with PEEK (poly-ether-ether-ketone) cage placement. The procedure was via lateral approach and no additional instrumentation (rods or screws) was used in the procedure. Subsequently, the man developed a rather profound lumbosacral plexopathy with very severe atrophy in both lower extremities as a complication of surgery.

When a CT was performed later to assess the fusion integrity and, no doubt, to look for a cause of the postsurgical lumbosacral plexopathy, they discovered a bilateral pars defect at L5-S1. This had not been seen earlier. I came into the case after the surgery had been performed; my primary task was the crash reconstruction and biomechanical assessment. However, I couldn't help but wonder whether the bilateral pars defects were pre-existing or whether the defects represented an acute fracture due to the trauma. If they were an acute fracture, might they have been a major contributor to the man's pain? Might the surgical fusion procedure have been unnecessary? Might the man's lumbosacral plexopathy have been avoided altogether? Or, could the pars defects have developed as a consequence of the fusion?

Pars Defects After Spinal Fusion

Interestingly, spondylolysis has been seen in individuals following spine fusion.1 In theory, the fused segment(s) produce a stress riser at the segments adjacent to them and could increase bending moments at the pars. Although these are older reports from the 1950s and 1960s, there was one recent report of a postsurgical pars defect developing in a patient who had been given a prosthetic disc, the intention of which was to preserve motion, and was thought by the authors to be the first report of this complication.2 To date, no long-term studies on disc arthroplasty have been published.3 Acceleration of disc disease is known to occur at levels adjacent to fused spinal sections, and secondary herniations are more likely there than in other segments.

This is particularly true in the cervical spine. While spine surgeons attempt to retain a lordotic curvature at the fused level, the final fusion mass may be kyphotic in nature. It has long been known that adjacent-level disc disease or spondylosis, or a combination thereof, will develop at adjacent levels within a decade,4,5 some requiring secondary surgeries. Goffin, et al.,4 theorized that such fusions induce accelerated changes and offered, in support of this thesis, that virtually 100 percent of the discs adjacent to congenital cervical spine fusions in Klippel-Feil syndrome cases are degenerative, and that in the lumbar spine, when pseudarthrosis (i.e., failure of attempted fusion) results, patients are less likely to develop adjacent degenerative changes later.

Katsuura, et al.,5 found that adjacent level disease was present in about 50 percent of cases after 10 years, and that it was twice as likely when the fused segment was in kyphosis as opposed to lordosis. They theorized that the kyphotic curve might place shear stresses on the adjacent levels that might precipitate or accelerate their secondary breakdown.

Problems With the PEEK Cage?

The PEEK (poly-ether-ether-ketone) is a synthetic cage designed to maintain the intervertebral disc height while providing a stable matrix for bony fusion. It is used in both cervical and lumbar procedures. In my literature search, I discovered several papers describing surgical outcomes using PEEK cages, in some cases comparing them to titanium cages. The procedure is to remove part or all of the disc and then to place the cage in the disc space. Into this region, they inject recombinant human bone morphogenic protein (rhBMP-2), which aids in new bone formation.6 It is claimed that BMPs induce differentiation of undifferentiated mesenchymal cells into osteogenic cells and enhance the function of osteoblasts.

The use of PEEK cages as a stand-alone devices or supplemented with posterior instrumentations remains controversial6 and there is a significant lack of biomechanical data with regard to the properties of the PEEK system.7 Surgical procedures employed in the placement of PEEK cages include anterior lumbar interbody fusion (ALIF), posterior lumbar interbody fusion (PLIF), and transforaminal lumbar interbody fusion (TLIF). Other than the symptom domain, the complications watched for are end-plate resorption, cage migration, and subsidence of the space. In contrast to ALIF, poor results have been achieved with TLIF and PLIF patients.6 Cage migration was most pronounced in the TLIF group and was not observed until six months after surgery.

In all of these reports, the surgeons used additional hardware to support a single-level fusion. In the case I investigated, the fusion was bilevel and no supporting rods of screws were placed. Biomechanical studies have now been conducted, but these also looked only at single-level constructs with support hardware,7 which are mechanically less challenging than bilevel fusions, particularly those in which no supportive hardware is used.

Interpreting the Research: Sources of Bias

While the results reported in the literature seemed fairly favorable, it is important to read these papers with care. Historically, surgical outcome studies suffer from several common flaws. One major limitation is a lack of randomization; another is a lack of controls. These hurdles can be - and have sometimes been - overcome, but most studies still do not have strong designs and lack randomization and control. Selection bias is another potential confounder. Even in a randomized trial, one could select patients who seem like excellent candidates for surgery. And it would be unethical to offer surgery to those who are unlikely to benefit from it.

In real-life situations, however, as in the case I was investigating, the need for surgery may be less clear. In that case, while there were some small lateral protrusions at L3-4 and L4-5, they were on the side opposite the patient's complaints. After an unsuccessful trial of two epidural steroid injections, a discogram was performed and was confirmatory for concordant pain, but only at the L4-5 level.

Examiner bias is seen when the surgeon or their staff conduct the follow-up interviews. Some patients typically offer their surgeon a more favorable picture than is warranted, inflating the level of surgical success and/or minimizing their current discomfort or impairment. Ideally, follow-up should be conducted by researchers not connected with the surgeon's office.

Information bias is yet another problem. How should one measure outcome? Is a visual analog scale (VAS) adequate, or should one use an outcome questionnaire such as the Roland-Morris or the Oswestry Disability Index? In one study that was favorable to PEEK cage instrumentation, an improvement in VAS or Oswestry of only 10 percent was considered a positive outcome.8 Going from a VAS of 9 to 8 would constitute an 11 percent (positive) outcome.

What about functional ability? Perhaps the best method would incorporate a combination of functional capacity evaluation (FCE) and these other outcome assessment tools. The timing of outcome assessment is also important. In some cases, patients are followed only for 3-6 months or for 12 months. A stronger study design would include a long-term follow-up. In several large, controlled studies in which some patients had back surgery and some elected not to have back surgery, after five years there were no important differences in outcome. The surgical patients may be predisposed to an accelerated degenerative process and other longer-term complications because of the surgery, but this has not been investigated carefully.

One last source of bias is journal bias. Papers that fail to reveal some new discovery or scientific breakthrough, overturn prior theory, or simply fail to reject their own null hypotheses lack cache in the world of medical publication and are less likely to be published in mainstream journals. Studies paid for by manufacturers of medical equipment or devices may not see the light of day if the outcomes are not favorable. As is the case in some drug studies, the researchers who crunch the final data and derive the final statistics are often not the primary investigators.

The Final Denouement

I finally did have the opportunity to look at the two MRIs taken before the car-crash patient's surgery, and the CD scan taken after surgery, as well as plain films taken immediately after surgery. The first MRI did show the bilateral L5 pars fractures, and it was my opinion that they were caused by the high compressive loading and lumbar extension induced by the rear-impact collision, which was likely only compounded by the secondary frontal collision (see part I of this series).

The fractures on this study were admittedly difficult to see on the first MRI, but for the acuity of the infamous "retrospectoscope." The MRI taken a few months prior to surgery, however, showed a more clear-cut pars fracture on one side, and another orthopaedic surgeon also commented on this after the fact. The plain films taken on the day of surgery also showed good evidence of the fractures. It is possible that these fractures pre-existed, although the changes seen across the two MRIs and CT scan suggest an acute process. The jagged and non-sclerotic fracture margins also argue against fatigue fracture. In any event, this weak point now serves as a highly unstable bending point and is made worse by the fusion mass above it (L3-5).

In my view, this is a fusion that will ultimately need to be extended to S1, although the prior complication of lumbosacral plexopathy certainly urges caution. In any event, based on the fact that pars defects are not mentioned in any presurgical documents or reports, one wonders whether the surgeon was aware of them at all when planning this procedure.

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