The adjacent segment

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Recent interest and debate regarding the phenomenon of adjacent-segment disease following either lumbar or cervical arthrodesis has been driven primarily by the development of disc arthroplasty. The authors of reasonably well-designed clinical trials have established the safety, durability, and effectiveness of cervical disc prostheses both in the preservation of motion and relief of radiculopathy in most cases. There is, as yet, however, no compelling evidence of any added clinical benefit of artificial disc replacement over the current treatment of anterior cervical discectomy and fusion (ACDF). Nevertheless, there has been considerable interest by both industry and spine surgeons who postulate that this technology may represent an advance over present treatment. There are, for example, several cervical disc prostheses in development, in Food and Drug Administration (FDA) investigational device exemption trials, or awaiting FDA approval. This belief in the superiority of motion-sparing technology is due primarily, if not exclusively, to the assumption that preservation of motion at the surgically treated segment will reduce the incidence of symptomatic adjacent-segment disease. The central question in this debate relates not only to the incidence of adjacent-segment disease but also to its cause. Put simply, to what degree is the occurrence of adjacent-segment disease directly attributable to a previously performed fusion compared with the natural history of cervical disc degeneration? Unfortunately, in the case of ACDF, it is usually not possible to assess these factors independently.

One of the most widely referenced studies on adjacent-segment disease was performed by Hilibrand and associates. In their retrospective chart review of 374 patients treated with anterior cervical decompression and fusion, the investigators used the Kaplan–Meier survivorship analysis to estimate an annual rate of symptomatic adjacent-segment disease of 2.9% and found that there was a greater than 25% probability of adjacent-segment disease in the first 10 years following this procedure. These findings have been cited by numerous publications as evidence of the direct effect of fusion on adjacent-segment degeneration. Not identified by most of these publications, however, are Hilibrand and colleagues’ additional findings of a reduced rate of adjacent-segment disease in patients who underwent multilevel fusion compared with single-level arthrodesis (12 and 18%, respectively), and a strong inverse correlation between the time to the onset of adjacent-segment disease and the degree of preoperative imaging-documented degenerative changes. Indeed, the authors concluded, “we believe that symptomatic adjacent-segment disease is the result of progressive cervical spondylosis at adjacent levels and is not caused by the arthrodesis itself.” Also prominently referenced in these publications as clear evidence of the deleterious effects of cervical fusion on adjacent levels is the 2004 study by Goffin et al., in which the authors reported a 92% incidence of radiographic adjacent-segment disease following ACDF. In that study, however, a 1% loss of disc space height was considered to be evidence of progressive disc degeneration. Furthermore, the authors did not find a statistically significant correlation between radiographically documented changes and clinical deterioration, and only 6.1% of the patients remaining in the study for at least 5 years required surgery for adjacent-segment disease during the mean follow-up period of over 8 years.

Somewhat overlooked in this debate is the role of posterior cervical decompression for cervical disc herniation/spondylosis. Most surgeons would readily acknowledge the effectiveness of posterior cervical foraminotomy, particularly in the treatment of isolated foraminal nerve root compression. For many surgeons, myself included, posterior decompression is frequently the preferred procedure in cases of unilateral foraminal nerve entrapment. Not only are the results satisfactory in the appropriately selected patient, but this procedure also preserves motion at the surgically treated segment and, intuitively, probably has minimal or even negligible effects on the biomechanics of either the treated or adjacent segment.

The accompanying study by Clarke and colleagues is
interesting for several reasons. First, the authors attempt to assess specifically the incidence and annual rate of both adjacent- and same-segment disease following single-level posterior cervical foraminotomy in patients with radiculopathy. Thus, it may allow some inference, or at least hypothesis, into the natural history of cervical spondylosis in patients with a history of symptomatic foraminal disc herniation/spondylosis treated with decompressive surgery without fusion. Second, the authors virtually replicated the study design, methods, and definitions of the study by Hilibrand and associates8 and assessed outcomes in the same manner as well. Like the study conducted by Hilibrand et al., for example, the study by Clarke and coworkers is a retrospective chart review covering the same period (1972–1992). The operational definition of adjacent-segment disease used in the two studies (that is, new symptoms on two consecutive visits) is identical as is the statistical analysis utilizing the Kaplan–Meier survivorship method. Both studies also rely on post hoc assignment of outcome status measured using a modification of the nonvalidated four-point (excellent, good, fair, or poor) Robinson-Smith scale. These similarities will likely lead to comparison between the studies and their respective findings, whether such comparisons were intended or valid.

Finally, Clarke and coworkers’ study vividly illustrates the challenges and obstacles that confront investigators when attempting to reach reliable, valid, and generalizable conclusions from a retrospective database.

In the present study, the authors conducted a retrospective chart review of 303 patients treated with posterior cervical foraminotomy over a 20-year period. They performed the Kaplan–Meier survivorship analysis to estimate the annual rate of adjacent-segment disease, recurrent same-segment disease, and axial neck pain. Excellent or good outcomes were reported in 268 (96.4%) of 278 patients, although we are not told when this final assessment occurred (mean or range). At least some of the excellent or good outcomes, however, appear to have been obtained in the 12% of patients with virtually no follow-up data (that is, the data were collected fewer than 10 days after the procedure). The authors calculated a “relatively stable annual 0.7% rate of developing adjacent-segment disease” over the first 10 years (although in Years 2, 4, 5, 8, and 10, the annual rate was 0%) and a 10-year rate of 6.7%. There was a 5-year rate of developing same-segment disease of 3.2% and a 10-year rate of 5.0%.

The results of this study should be interpreted in light of the numerous limitations in study design and methods. First, it is a retrospective chart review. The last patient was treated nearly 15 years ago, and it appears that none of the authors was involved in the care of any patient in this series. Only data that were recorded in the chart were available for review, and it is improbable that there was uniform data collection and recording in the hospital/clinic charts. It is further unlikely that all outcomes were prospectively assessed in a standardized fashion, so the reviewer probably had to assign the outcome on the basis of his/her interpretation of the patient’s record.

Second, many patients were lost to follow up in this study. Nearly one third of the patients did not even finish follow-up duration of 1 year and thus their data contributed nothing to the Kaplan–Meier survivorship analysis. A minimum of 5 years of follow-up information was available only for 55% of the patients. Such a high loss to follow up is not surprising given the nature of the condition under study (that is, single-level cervical radiculopathy) because surgeons and/or the referring neurologist will rarely actively monitor these patients in the long term, particularly when 96.4% of patients have a good or excellent result. Unfortunately, however, one of the most serious risks to the study’s validity is patient attrition. Patients who do not return for follow-up examination may differ from those who do in ways that may be related to the outcome under study. The authors believe that any patient with recurrent symptoms would likely return. From a purely methodological perspective, however, such a postulation is not valid. Ultimately, the critical reader must decide whether the authors’ assumptions regarding loss of patients to follow up are tenable. The observation that in 12% of the cases there were virtually no follow-up data, only 3 months of follow-up data in 22%, and a loss to follow up of nearly one third of the patient population before the end of the 1st year may belie this assumption.

Finally, an additional threat to the accuracy and completeness of the long-term assessment is the passive nature of the long-term follow-up review. Apparently, patients were not contacted by the authors for up-to-date follow-up information nor did they likely undergo periodic evaluation performed postoperatively by a staff surgeon and staff neurologist for a mean of over 8 years (range 0–33 years). In fact, the reader does not know the extent of active follow up by the treating surgeon and/or neurologist. It appears that as long as patients had any medical interaction with the Mayo Clinic or affiliates (for example, emergency department, internist, or outpatient clinic) they would be considered as continuing in the study. In other words, for the purpose of event calculation, no news was good news. This is problematic because event identification in this study relied on voluntary patient reporting to identify each event (that is, adjacent-segment disease, same-segment degeneration, and axial neck pain) based on an assumption that the patient would return to the surgeon/neurologist for each event. As noted in standard statistical texts, the use of such passive databases for the construction of Kaplan–Meier estimates is “notorious for underreporting events and overestimating performance as estimated in the survival curve.”1 This is at least partially acknowledged by the authors in explaining the extremely low incidence of axial neck pain in this population of patients with a history of surgery. It is simply not enough to state that the Kaplan–Meier survivorship analysis does not assume that all patients lost to follow up are event free. The critical reader must be confident that those lost to follow up are not different than those who remain in the study in ways that are associated with outcome (that is, informative censoring). Although the Kaplan–Meier method accounts for patient attrition, it does not explain, correct, or control for the reasons for the attrition.

The results from several other published studies on posterior cervical foraminotomy differ from those of the present study. In the study by Wirth et al.10 for example, 27% of patients treated with posterior cervical foraminotomy required a reoperation during a mean follow-up period of 45 months. The reoperations were evenly distributed between treated and adjacent segments. In their long-term follow-up study of 62 patients who underwent posterior