Editorial

Adjacent-level disease

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Kulkarni, et al., have provided insight and confirmed most surgeons’ biases regarding degenerative changes at adjacent-level segments following fusion for cervical spondylotic myelopathy. Their findings suggest, but do not prove, that kyphotic deformity predisposes to accelerated degenerative changes and that fusion itself predisposes to such lesions. The fact that they observed disc bulging at adjacent levels, however, does not prove that these changes were related to the prior fusion because no control population was evaluated in this retrospective study. Their reporting of sagittal alignment, both pre- and postoperatively, is subjective or at least not totally objective. Perhaps a more objective measurement of pre- and postoperative angulation in this patient population would have yielded different results. Nevertheless, this reader’s bias is that fusion and sagittal malalignment accelerate (kyphosis) degenerative changes at levels adjacent to a fusion site. The results demonstrated by Kulkarni, et al., support this bias. If we take the aforementioned comments into consideration and do not overreact to the authors’ observations, their data can be appropriately integrated into our knowledge base. As is usually the case, care must be taken when interpreting these and other retrospective data. For this, the authors are to be congratulated regarding their observations and presentations.

RESPONSE. Dr. Benzel has raised two important issues regarding our study. First is the issue of an appropriate control study by which to evaluate the rate of a normally occurring process (spondylotic changes in the cervical spine in our case) that might have been accelerated because of our surgical intervention. As we explained in our article, it is almost impossible to obtain an ideal control population for such a study, whether prospective or retrospective in nature. Spondylotic processes occur at varying rates in different individuals and, as such, assessment of an age- and sex-matched group would not have unequivocally addressed the issue. Furthermore, control individuals would have to volunteer to undergo cervical magnetic resonance imaging on two occasions (comparable with the pre- and postoperative studies acquired in our patients). Our strategy for a control involved the use of a motion segment remote from the fusion site in the same patient (internal control). We concede that this is not ideal because it frequently involved the assessment of a segment, such as C2–3 or C7–T1, that is normally not prone to spondylotic change. We believe that an ideal control group for a study such as ours remains elusive. The second issue that Dr. Benzel raises is that of using a more objective measurement of cervical sagittal alignment. Our method was simple and objective, but it did not allow for quantitative assessment of the sagittal alignment of the cervical spine. It is possible that a statistically significant correlation might have been obtained between kyphotic change and adjacent-segment degeneration if a quantitative assessment had been made of the sagittal alignment. As it stands, our findings do point to a trend toward such a correlation.

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