

when considered from the perspective of the operation that worked better (transaxillary rib resection).

In some patients who have had insufficient symptom relief following the transaxillary operation, I have resorted to performing a supraclavicular neuroplasty. This follow-up surgery seems to help many, but not all, patients. Perhaps a first-rib resection via the supraclavicular approach is the best option because the neuroplasty can be easily performed as part of the procedure. I have found that first-rib resection is awkward via the supraclavicular approach. Randomized studies can address these issues in the future.

Unfortunately, too many patients do not benefit from whatever we do surgically. Perhaps resection by itself (be it bone, bands, and/or muscle) makes the plexus vulnerable to traction from the arm hanging at the patient's side. Indeed, many patients—in particular, those who have not benefited from surgery—appear to achieve symptom relief by wearing a sling that holds the arm in a position such that the shoulder is elevated. A technique that preserves suspension of the shoulder may be required in some cases. Can we identify these patients a priori?

PubMed lists 1080 studies if one searches with the entry “thoracic outlet surgery.” Our study is the only randomized prospective study of 2 different surgical techniques. The lack of other randomized trials is unfortunate. Despite innumerable clinical reports, we have much still to learn about surgery for TOS. Failure rates, regardless of management, are too high. Careful study, rigorous trial design, and avoidance of rigidly held points of view based simply on experience may be ways for us to start making some inroads into the treatment of this disease.

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## Prognosis in Spine Surgery

TO THE EDITOR: We believe there may be error in Table 6 of the excellent article by Mastronardi and colleagues (Mastronardi L, Elsawaf A, Roperto R, Bozzao A, Caroli M, Ferrante M, et al: Prognostic relevance of the postoperative evolution of intramedullary spinal cord changes in signal intensity on magnetic resonance imaging after anterior decompression for cervical spondylotic myelopathy. *J Neurosurg: Spine* 7:615–622, December, 2007).

### Abstract

**Object.** Areas of intramedullary signal intensity changes (hypointensity on T1-weighted magnetic resonance [MR] images and hyperintensity on T2-weighted MR images) in patients with cervical spondylotic myelopathy (CSM) have been described by several investigators. The role of postoperative evolution of these alterations is still not well known.

**Methods.** A total of 47 patients underwent MR imaging before and at the end of the surgical procedure (intraoperative MR imaging [iMRI]) for cervical spine decompression and fusion using an anterior approach. Imaging was performed with a 1.5-tesla scanner integrated with the operative room (BrainSuite). Patients were followed clinically and evaluated using the Japanese Orthopaedic Association (JOA) and Nurick scales and also underwent MR imaging 3 and 6 months after surgery.

**Results.** Preoperative MR imaging showed an alteration (from the normal) of the intramedullary signal in 37 (78.7%) of 47 cases. In 23 cases, signal changes were altered on both T1- and T2-weighted images, and in 14 cases only on T2-weighted images. In 12 (52.2%) of the 23 cases, regression of hyperintensity on T2-weighted imaging was observed postoperatively. In 4 (17.4%) of these 23 cases, regression of hyperintensity was observed during the iMRI at the end of surgery. Residual compression on postoperative iMRI was not detected in any patients.

A nonsignificant correlation was observed between postoperative expansion of the transverse diameter of the spinal cord at the level of maximal compression and the postoperative JOA score and Nurick grade. A statistically significant correlation was observed between the surgical result and the length of a patient's clinical history. A significant correlation was also observed according to the preoperative presence of intramedullary signal alteration. The best results were found in patients without spinal cord changes of signal, acceptable results were observed in the presence of changes on T2-weighted imaging only, and the worst results were observed in patients with spinal cord signal changes on both T1- and T2-weighted imaging. Finally, a statistically significant correlation was observed between patients with postoperative spinal cord signal change regression and better outcomes.

**Conclusions.** Intramedullary spinal cord changes in signal intensity in patients with CSM can be reversible (hyperintensity on T2-weighted imaging) or nonreversible (hypointensity on T1-weighted imaging). The regression of areas of hyperintensity on T2-weighted imaging is associated with a better prognosis, whereas the T1-weighted hypointensity is an expression of irreversible damage and, therefore, the worst prognosis. The preliminary experience with this patient series appears to exclude a relationship between the time of signal intensity recovery and outcome of CSM.

Table 6 summarizes changes in measurements of the spinal cord at the level of maximal compression before and after surgery. Preoperative measurements for anterior subarachnoid space in millimeters and transverse diameter of the spinal cord in millimeters and postoperative measurements are given in ranges of 0–46, 41–53, 16–49, and 48–74. We suspect the authors meant 0–4.6 mm, 4.1–5.3 mm, 1.6–4.9 mm, and 4.8–7.4 mm.

We want to compliment the authors on an excellent article. We have seen similar signal changes in the spinal cord when we have used even mid-field and low-field MR imaging units. The discussion and research are particularly enlightening, and we agree with the conclusions. We have seen better prognoses for patients whose T2-weighted images have no altered intramedullary signal or at least have only a hyperintense signal, such as a “snake eye” appearance. Such signals sometimes indicate that gliosis, myelomalacia, and edema would be reversible. The more ominous low signal change on T1-weighted MR images is indicative of irreversible spinal cord damage and poor prognosis. We agree with the references and discussion of the direct effects of chronic compression and secondary circulatory disturbances in the central gray matter and the ventral posterior columns supplied by the terminal branches of the anterior spinal artery.

Cervical myelopathy continues to be a difficult challenge. Despite excellent decompression by neurosurgeons, some patients may develop progressive problems caused by what we term intraneural fibrosis or even delayed postoperative signal changes related to the initial protrusion, stenosis or compression. Such signal changes result from

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the initial myelopathic diagnosis rather than any surgical event.

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RESPONSE: First of all I would like to thank Dr. Gilbert and colleagues for their letter and for the nice comments to our article. It is obvious that there is an error in the preoperative and postoperative data in Table 6. As Dr. Gilbert and colleagues have suggested, the correct ranges are 0–4.6 mm, 4.1–5.3 mm, 1.6–4.9 mm, and 4.8–7.4 mm.

I am very grateful to Dr. Gilbert and his colleagues for the confirmation of our preliminary data. Regression of the

hyperintensity on T2-weighted imaging is associated with a better prognosis, whereas the T1-weighted hypointensity is not reversible and, therefore, in these cases the prognosis is worse. In our paper we tried to stress that there is not enough information regarding the possible different outcomes in relation to the early or late regression of hyperintensity on T2-weighted images. Our preliminary experience seems to exclude a relationship between time of signal recovery and outcome of myelopathy. Also the amount of reexpansion of the spinal cord at the level of maximal compression does not seem to be correlated with long-term outcome.

Again, thank you for the comments and for prompting me to clarify the errors relative to measurements.

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