Neurosurgical Forum
LETTERS TO THE EDITOR**Giant presacral schwannomas**

TO THE EDITOR: We read with great interest the review article by Pennington et al.¹ on the surgical management of giant presacral schwannomas (Pennington Z, Westbrook EM, Ahmed AK, et al. Surgical management of giant presacral schwannoma: systematic review of published cases and meta-analysis. *J Neurosurg Spine*. 2019;31[5]:711–722). We ourselves have treated 4 cases in the last 5 years that largely confirm the recommendations mentioned in the article by Pennington et al. (2 Klimo type III and 2 Klimo type II cases²). Based on this small experience, we have a few comments on the management recommendations mentioned by the authors in the discussion.

As for their first recommendation that staged anterior-posterior approaches are preferred for Klimo type II lesions with large presacral components, we agree with the authors that the combined procedure provides the best overview of the proximal and distal parts of the tumor and the nerve roots, and therefore is the best option to achieve gross-total resection, but we would like to add that the age of the patient should also be considered in the process of surgical decision-making. As illustrated in our case in Fig. 1, a substantial resection can also be achieved via the posterior approach. Although the chance for progression-free survival (PFS) using this surgical strategy is lower than that of the combined anterior-posterior approach, the risks for complications are also lower. In our opinion, therefore, the risks for an additional anterior approach should

be weighed against the chance of developing symptoms should a small part of the tumor remain anteriorly.

As for their second recommendation that lesions involving the L5–S3 nerve roots should generally be treated with GTR, we agree on this strategy, but in addition would like to comment that the same principle should be applied that is used for the resection of schwannomas in the peripheral nervous system,³ namely to identify and transect the fascicle or filament from which the schwannoma has originated proximally and distally. In our small series, this strategy did not lead to any motor deficits. What is important to realize in this regard is that these giant presacral schwannomas have developed slowly, and the function of the affected nerve root in time may have been overtaken by another nerve root. In our case illustrated in Fig. 1, for example, both the ventral and dorsal filaments of the affected L5 nerve root were stimulated intraoperatively (nerve action potentials) after opening of the dura, but this did not elicit a motor response in the leg. As also stated by Pennington et al. of these benign lesions, there is no need to perform an en bloc resection. We would like to add that there also is no need to transect the entire nerve root but to perform an intracapsular resection as in the peripheral nervous system.

Finally, the authors recommend that surgeons carefully consider stabilization in cases of extensive sacral dissection. We agree, but we would like to add that stabilization is also indicated in cases of extensive involvement of L5, as illustrated in Fig. 1.

We congratulate the authors on the great work they

FIG. 1. Case of a 71-year-old man with a presacral schwannoma (Klimo type II, diameter 8 cm) originating from the L5 root on the right side (A) with substantial destruction of the L5 vertebral body (B). Subtotal intracapsular resection was performed through a posterior approach only. Reconstruction was performed using allograft bone shaped to fit the defect that was connected to the construct with a pedicle screw (D). There were no postoperative complications. There was a small potential residue on the MRI obtained half a year after the surgery (C).

have done in reviewing the current literature on giant presacral schwannomas.

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Disclosures

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Response

We were eager to read the comments published in this issue by de Ruiter et al. on our proposed treatment algorithm for patients with giant presacral schwannomas. Although our recommendations were based on dozens of cases treated across multiple institutions, they were made by members of a single institution, which carries with it the risk of institutional bias. Therefore, we were pleased to find that our proposed algorithm was similar to that endorsed by our peers at other academic institutions.

With regard to the authors' proposed amendments to our algorithm, we find them to all be reasonable modifications. In our article we emphasize gross-total resection because of its improved control rates relative to subtotal resection. However, we acknowledge that in some patients, oncological control may not be the ideal outcome for surgery. Specifically, in older patients and those with extensive medical comorbidities who have limited life expectancy, the patient's ultimate cause of death is unlikely to be the schwannoma. Consequently, prioritization of symptomatic relief and emphasis on minimizing complications is a more than reasonable therapeutic approach. As evidenced by the authors, such symptomatic debulking can be reasonably achieved via a posterior-only approach for Klimo type II lesions.¹

Similarly, we also agree with de Ruiter et al. that efforts should be taken to dissect out and isolate the parent fascicle so as to preserve the function of the affected root. Similar to de Ruiter, we prefer to use a stimulator with a threshold of 1 mA, which has been described elsewhere as a safe and highly effective technique for the excision of peripheral nerve sheath tumors.^{2,3}

As for the authors' second major point, namely the need for stabilization for lesions crossing the lumbosacral junction, we wholly agree. The lumbosacral junction represents the intersection of the mobile spine and the rigid sacrum. Consequently, it experiences both high compressive and shear forces.⁴ Destruction of the bony elements of the lumbosacral junction, as may occur in the setting of a Klimo type I or type II lesion,¹ is associated with significant destabilization⁵ that can generate severe long-term disability.⁶ Therefore, we agree with the argument that there should be a low threshold for instrumented fusion in any lesion crossing the lumbosacral boundary. Our preferred method is the placement of S2-alar-iliac screws, when possible, and iliac bolts when the sacrum has been eroded by tumor. These pelvic anchors can help to reduce the S1 screw stain known to arise in lumbosacral constructs ending at S1.⁷ However, the constructs should be individualized based on the size of the patient's lesion; some patients with only partial involvement of the lumbosacral junction may not require pelvic instrumentation.

We again thank de Ruiter and colleagues for their input on our article and would like to encourage further dialogue of this nature when trying to identify management algorithms for all rare spinal pathologies.

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Indirect decompression via lateral interbody fusion for patients with severe degenerative lumbar spinal stenosis

TO THE EDITOR: We read with great interest the article by Shimizu and colleagues¹ (Shimizu T, Fujibayashi S, Otsuki B, et al. Indirect decompression with lateral interbody fusion for severe degenerative lumbar spinal stenosis: minimum 1-year MRI follow-up. *J Neurosurg Spine*. 2020;33(1):27–34). In this article, the authors provide 1-year clinical and radiographic outcome data for patients with severe degenerative lumbar spinal stenosis who underwent indirect decompression via lateral interbody fusion. The severity of central stenosis was graded according to the Schizas classification for the severity of lumbar stenosis.² All patients underwent a lateral retroperitoneal pre-psoas approach lumbar interbody fusion augmented with posterior percutaneous pedicle screw fixation without direct decompression. The degree of decompression was then assessed with MRI at 3 weeks and 1 year postsurgery. Clinical improvement was assessed using the Japanese Orthopaedic Association (JOA) score for degenerative lumbar diseases.³

The authors report excellent clinical improvement, with JOA scores improving by 74.9% at the 1-year follow-up. Radiographic improvement was likewise noted, with the cross-sectional area (CSA) improving from 54.5 ± 19.2 mm² preoperatively to 132.6 ± 37.5 mm² at the last follow-up ($p < 0.001$). Among 45 surgically treated levels, they noted good CSA expansion at 29 levels (defined as expansion $> 200\%$), whereas 16 levels showed poor CSA expansion ($< 200\%$). Univariate analysis revealed a smaller preoperative CSA and pseudarthrosis on the 1-year postoperative CT as risk factors for poor CSA expansion. Somewhat counterintuitively, there was no correlation between CSA expansion and improvement in JOA scores. Risk factors and incidence of suboptimal JOA score improvement are not discussed.

The appropriate candidate for indirect decompression in degenerative spinal stenosis remains a hotly contested topic, with a growing body of evidence suggesting that stenosis can be treated via arthrodesis alone.⁴ While the relationship between clinical improvement and degree of decompression is unclear, as Shimizu and colleagues report, there does appear to be a subset of patients that benefit from direct decompression along with arthrodesis. The authors discuss radiographic contraindications to the indirect treatment paradigm; patients with extra-ligamentous disc herniation, locked facets, and stenosis due to bony compression were not deemed candidates in this study. An additional consideration not discussed in this article is the response of patients' clinical symptomatology to positional changes. In our experience, patients who experience positional relief of their symptoms (with either forward flexion, rest, supine positioning, or recumbency) will achieve acceptable postoperative results after indirect decompression. Patients with symptomatology that does

not respond to positional changes, who continue to be symptomatic in recumbency, may not attain clinically acceptable results without direct neural decompression. The question of how much is enough is, as of yet, unanswered. Supplementing the discussed radiographic criteria with a detailed history of present illness may help clinicians to appropriately select patients who will obtain adequate decompression indirectly.

This article represents a significant contribution to the ever-growing evidence basis for using minimally invasive surgical techniques in the treatment of degenerative spondylosis. We congratulate the authors on their excellent paper.

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Disclosures

Dr. Mundis is a consultant for NuVasive, K2M, Viseon, Seaspine, and Allosource; has direct stock ownership in NuVasive and Viseon; receives royalties from K2M; and is a board member of Global Spine Outreach and San Diego Spine Foundation. Dr. Uribe is a consultant for NuVasive, Si Bone, and Misonix; and has direct stock ownership in and receives royalties from NuVasive.

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Response

We would like to thank Dr. Vivas and colleagues for their interest and valuable review of our article. We would like to take this opportunity to address their comments. They raised an extremely important topic regarding the suggestion that stenosis can be treated via arthrodesis alone. According to Goel et al., the patients in their series were successfully treated with transarticular fixation alone.¹ Although we are somewhat skeptical with regard to

the stability of transarticular screws, their concept seemed reasonable. In our study, all patients experienced clinical symptomatic recovery after lateral interbody fusion with supplemental pedicle screw fixation. Apparently, this technique provides rigid stability with both intervertebral and posterior fixation, resulting in the greatest degree of resolution of spinal instability–related symptoms. Nonetheless, the most critical aspect is to maintain this stabilization in the long term and ultimately achieve solid segmental arthrodesis. Therefore, in our paper, we emphasized that the patients without solid fusion at the 1-year follow-up were at risk for failure of maintaining the indirect decompressive effect.

Dr. Vivas and colleagues also raised another interesting point: the utility of symptom relief by positional change. We agree with this comment, as positional pain relief is a characteristic clinical sign of lumbar stenosis. Given the retrospective nature of our study, we were unable to assess the patients' detailed histories, including positional symptom relief. However, some of the patients with persistent intractable radicular pain while in the recumbent position demonstrated symptomatic relief postoperatively. We believe that a thorough evaluation for determining the specific mechanism of pain relief by positional change in lumbar stenosis would be very challenging. To address this issue, for instance, the extent of the decompressive effect

actually achieved in the recumbent and/or flexion position should be radiologically investigated in every study participant and then would need to be compared with the postoperative decompressive status. In addition, meticulous workup would be required to identify the pain generator, particularly in the setting of coexisting foraminal stenosis (radiculopathy). Prospective research including these assessments may help to develop new criteria for determining an appropriate candidate for indirect decompression.

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