Neurosurgical Forum

LETTERS TO THE EDITOR

Brachial plexus stretch injury needs anatomical context

TO THE EDITOR: We read the article by Zapałowicz and Radek¹⁰ with interest (Zapałowicz K, Radek M: The distribution of brachial plexus lesions after experimental traction: a cadaveric study. *J Neurosurg Spine* 29:704–710, December 2018). They are to be commended on their study; however, we wish to raise several concerns about their methodology and conclusions. They described 30 brachial plexuses (BPs) that were stretched to mechanical failure, with 140 injuries noted, but they did not describe the patterns of injuries in terms of clinical injuries (e.g., C5–7 or Narakas II).⁶ For example, the finding of 89 preganglionic injuries may mean that every specimen had an avulsion, in contrast to most clinical injuries.

It appears that the authors' observations do not match previously reported clinical experiences. In a series of surgical BP stretch injuries,⁴ avulsions were present in only about a third of C5–T1 injuries. Birch² noted a 46% incidence of root avulsion. Most authors have noted that avulsions are less common in the upper roots of the BP.^{1,6} And although Zapałowicz and Radek¹⁰ concluded that injuries were more common in the lower nerves in groups A and C, this conclusion was not tested statistically. Chi-square comparison of groups A and B found no statistical difference ($p = 0.13$). Group C was statistically different from group B ($p = 0.014$); however, this testing paradigm is anatomically impossible—group C demonstrates traction 180° caudal to the foramen and not what is demonstrated in their Fig. 2C. Furthermore, the authors found only 3 injuries to the trunks among all 140 injuries, whereas trunk-level injuries are common in BP injuries. The results do not match clinical experience.

We suspect that the abnormal results arise from complete dissection of the nerves from the anatomy adjacent to the spine. We have demonstrated that the nerve branches arising from large nerves create tethers to the adjacent tissues,⁵ and traction on the large nerve leads to rupture at these branch points. When the BP is viewed in this perspective, the proximal branches arising from the C5–7 roots shield the stress applied to the spinal rootlets and preferentially lead to extraforaminal rupture, whereas the C8 and T1 roots do not have branches above the clavicle and thus are more liable to avulsion. By only testing the relationship of the BP to the spinal column, the authors

recapitulated other studies of the same experimental paradigm,⁷ not the anatomical conditions of clinical injuries.

Having experience in formal biomechanical testing, we would like to address some other concerns. 1) The clamping method for the distal end of the nerve is not described, yet their Fig. 1 appears to depict differences among their three testing setups, which may influence failure mode. 2) The authors attribute the mechanical protection against traction to collagen in the epineurium and perineurium, which we⁹ and other authors^{3,8} have found to be thin layers that are minimally involved in tensile strength.

We believe that the conclusions of this paper are unique to the experimental setup and warrant careful consideration before application to clinical scenarios. There is more to traction injuries of the BP than foraminal ligaments.

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Disclosures

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Published online June 14, 2019; DOI: 10.3171/2019.2.SPINE19148.

Response

We would like to thank Drs. Yeoh and Mahan for showing interest in our work. The aim of our study was to explore the course of injury to an isolated human BP without the impact of the surrounding tissues. We drew inspiration from the experimental works of Stevens¹² and Frykholm⁶ as well as the work of Destandau et al.⁴ Our study does not recapitulate the excellent work of Sunderland,¹³ as it is not of an experimental nature.

Tensioning of 30 BPs led to 63.6% preganglionic injuries and 36.4% postganglionic injuries. Preganglionic injuries occurred in 25 (83%) of the plexuses and in 89 (59%) of 150 spinal nerves. The injuries were not classified in accordance with the clinical division, as they were related to specimens. The level of destruction of each of the BPs would make its neurological function in a living organism impossible.

The method of attaching the distal elements of the BP has been described: the BP hung loosely for several minutes, then the nerves coming out of the cords, placed in parallel, were wrapped with gauze, wound on a longitudinally grooved cylinder, and pressed to the cylinder using a metal plate such that the nerves surrounded three-quarters of the cylinder circumference. Our article documents the method of fixing the spine in a testing machine. In a motorcycle accident, the impact on the shoulder at high speed can be in the cephalocaudal direction,^{3,5,8,10,11} which explains the use of such a tension direction in the group C specimens.

Our article also mentions that postganglionic elements of the BP contain collagen; however, its contribution to the BP mechanical strength is not touched upon, as it was not the aim of the study.

The experimental conditions did not correspond to injury conditions that a living organism is exposed to; therefore, the discussion took into account previous experimental studies on animals and cadavers. Because of the anatomical differences, the tension results for the BPs of small animals may differ from the results of corresponding experiments on human BPs. On the other hand, the previous studies on human BPs were different in terms of methodology, which makes results comparison more difficult.

It is highly probable that force acting on the BP of a living organism is modified by the mechanical properties of the surrounding tissues; it is evidenced by frequent associated musculoskeletal injuries.^{3,5,8,10} For this reason, the injuries of the isolated BP achieved in laboratory conditions should be very carefully compared with accident victim injury statistics.

The percent values of preganglionic injuries given by

us are higher or similar to values given by authors discussing large batches of clinical cases. For example, Narakas found preganglionic injuries in 68.3% of spinal nerves;¹¹ Kaiser and colleagues, in 72% of patients;⁷ and Birch, in 46%–63% of patients.² In 208 patients with C5–T1 spinal nerve injuries, Kim et al. found 45% preganglionic injuries among 1040 spinal nerves.⁹ Bertelli and colleagues found supraclavicular injuries of the brachial plexus in 91% of 565 patients; in 45% of cases, they found a preganglionic injury of at least one spinal nerve.¹

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INCLUDE WHEN CITING

Published online June 14, 2019; DOI: 10.3171/2019.4.SPINE19172.

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PEEK interbody device and pseudarthrosis

TO THE EDITOR: We read, with keen interest, the article published by Krause et al.² (Krause KL, Obayashi

JT, Bridges KJ, et al: Fivefold higher rate of pseudarthrosis with polyetheretherketone interbody device than with structural allograft used for 1-level anterior cervical discectomy and fusion. *J Neurosurg Spine* 30:46–51, January 2019). We commend the authors for analyzing this pertinent clinical problem. The authors concluded that the use of polyetheretherketone (PEEK) devices in 1-level anterior cervical discectomy and fusion (ACDF) is associated with a significantly higher rate of radiographically demonstrated pseudarthrosis and the need for revision surgery compared with the use of allografts. However, there are a few important points that we would like to bring to the attention of the readers of this journal.

Diabetes and osteoporosis are two important patient factors leading to pseudarthrosis, apart from smoking and obesity.^{3,4} We believe that the PEEK group and the allograft group must be matched for these probable confounding factors.

The authors mentioned that 408 patients underwent 1-level ACDF during the collection period, which ended in July 2016. Twelve patients received iliac crest autograft, and the remaining 396 received either PEEK cage or structural allografts. Only 127 met the study's inclusion criteria. We would like to know if there were any other exclusion criteria besides inability to procure radiological follow-up of the remaining 269 patients. Kindly comment on the limited inclusion of 127 patients.

We would also like to know the material used (PEEK, structural allograft, or autograft) during the repeat surgery of patients with symptomatic pseudarthrosis following ACDF using PEEK cages. It would be pertinent to know if their symptoms resolved after the repeat surgery. The authors also mentioned that repeat ACDF with PEEK cage was done in the single case of symptomatic pseudarthrosis following primary ACDF with structural allograft. Although the use of PEEK versus allograft versus autograft in primary cervical arthrodesis is contentious, most studies support the use of autograft in revision operations to optimize fusion potential.^{3,5} It is relevant to know the thought process behind choosing PEEK cage in that scenario.

CT evaluation may actually omit the nonunion and overstate the fusion rates because of the imaging features of cortical allografts.¹ Hence, the use of a single standardized method for evaluating pseudarthrosis in all cases will eliminate the intrinsic bias associated with using either one of these modalities.

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Disclosures

The authors report no conflict of interest.

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Published online June 14, 2019; DOI: 10.3171/2019.3.SPINE19244.

Response

We thank Drs. Sardana, Sahu, and Kedia for their interest and comments regarding our article. We would like to respond to the questions they posed.

First, we agree that diabetes and osteoporosis are both important factors to consider. In terms of diabetes, 6 (10.7%) of 56 patients in the PEEK group were diabetic compared to 10 (14.1%) of 71 in the structural allograft group. Despite the higher prevalence of diabetes in the structural allograft group, fusion rates were much higher. However, this does not take into account whether patients' diabetes was well or poorly controlled. Osteoporosis prevalence was not studied in this patient population, but we agree that future prospective studies should control for osteoporosis.

Second, as mentioned in the manuscript, follow-up practices varied from surgeon to surgeon. Some surgeons did not see patients beyond a few months postoperatively, and others did not obtain imaging unless clinically indicated. The 269 excluded patients were so solely due to the lack of radiography at or after 12 months following surgery.

Third, all patients who suffered from pseudarthrosis with PEEK cages received posterior cervical fusion. Only one patient underwent a redo ACDF but had a posterior fusion in addition. All patients were clinically stable or improved following the reoperation.

Lastly, for one patient who suffered from pseudarthrosis in the setting of structural allograft, the surgeon who provided care is no longer at our institution, and the rationale behind the use of a PEEK cage for the revision surgery is not clearly documented. It should be noted, however, that the patient did ultimately require a posterior foraminotomy at the surgical level for persistent radiculopathy.

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Published online June 14, 2019; DOI: 10.3171/2019.4.SPINE19276.

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