Letter to the Editor

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In response to Mr Pringle

The authors appreciate Mr Pringle's questions and comments and the opportunity to respond.

Mr Pringle acknowledges the acute disruption of the C3/4 disc, which was evident as bleeding into the disc, and questions whether the disc protrusion (retropulsion) was temporally dissociated from the disruption; that is, it predated the injury. The facts that (i) the hemorrhage within the cord was also recent and (ii) the hemorrhage was juxtaposed to the retropulsed disc, and applying the principle of 'Occam's razor', led us to consider that disc retropulsion, at the time of injury, causing cord hemorrhage, was the most parsimonious explanation.

Mr Pringle correctly identifies that the figure legend erroneously refers to herniation of the C4/5 disc instead of C3/4.

Mr Pringle challenges the concept of 'transiently retropulsed discs' at other levels and questions 'if the discs are retropulsed by the injury would they not remain so?' As noted in the manuscript, we made no attempt to discern between the putative mechanisms of transient disc retropulsion and a pincer-like process involving transiently retroluxed vertebrae. Both mechanisms have been previously considered instrumental

in causing cord damage in the absence of vertebral fracture or residual deformity.¹⁻⁴ The supposition here is that the intrinsic stiffnesses of the posterior longitudinal ligament and dura are sufficient to spontaneously reduce transient subluxation of vertebrae, disc retropulsion or disc bulging.

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References

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