Delayed Vision Loss and Therapeutic Intervention After Blast Injury

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Explosive blasts associated with even mild traumatic brain injury can lead to substantial but delayed visual deficits, even in the absence of overt clinical presentation.¹ There are two major challenges for these patients: lack of sensitivity in diagnostic testing to reveal the visual deficit and a dearth of viable treatment options to restore lost vision. In this issue of *IOVS*, Dutca et al.² induce mild traumatic brain injury by exposing mice to a blast wave in a closed chamber. They provide compelling evidence that pattern electroretinogram (pERG) testing is effective for detecting early and delayed vision loss in their mouse model. They report an initial decrease in the pERG at 1 week after blast followed by a transient recovery and then further decline 16 weeks after blast. In contrast, cell death was limited to a subset of retinal ganglion cells (RGC) in the first week after blast. They do not report death of other cell types. The lack of outer retina damage, which is a hallmark of closed globe blunt injury, suggests that their model mimics blast, not blunt, trauma. Their detection of delayed vision loss in the absence of cell death is in agreement with our studies using an eye-directed air blast trauma model that also showed delayed and primarily inner retina driven vision loss (by flash ERG)³ and may suggest inner retinal cell dysfunction. The detection of delayed vision loss in our models supports their clinical relevance, which is very promising for the development and testing of potential therapeutics. In fact, Dutca et al.² test the efficacy of a promising compound. In light of their visual function results, and ours, it may be worthwhile to assess the sensitivity and accuracy of the pattern or flash ERG in trauma patients.

References

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