The reported incidence of traumatic optic neuropathy (TON) ranges from 1.5% to 4% following head trauma, and the management of such injuries remains controversial. Original treatment options included observation or optic canal decompression through a frontal craniotomy. Later surgical approaches involved external or transantral ethmoidectomy and, more recently, a combined transconjunctival and intranasal endoscopic approach. In 1982, Anderson et al proposed the use of high-dose corticosteroids for TON based on the observation that such treatment was effective in reducing traumatic central nervous system edema. Although the spinal cord and optic nerve are very different structures, many believed that the Second National Acute Spinal Cord Injury Study in 1990 provided further support for corticosteroid intervention within the first 8 hours of optic nerve injury, spurring the advocacy of rapid intervention in the form of a specific dose of methylprednisolone sodium succinate alone or in combination with optic nerve decompression surgery. However, following the second optic nerve injury, most patients’ vision fell to the pretreatment level of the first injury, and subsequent management of the second injury with corticosteroids and/or optic canal decompression provided little or no vision return. In contrast, optic canal decompressions performed for 91 primary TON injuries resulted in 82.4% having some degree of vision improvement. Patients with TON may have a second optic nerve insult, and vision recovery from the second event may be limited regardless of primary treatment choice.
Table 1. Patients With Second Traumatic Optic Neuropathy Injuries

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Mechanism of Trauma</th>
<th>Visual Acuity</th>
<th>Original Treatment</th>
<th>Time to Second Injury, y</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>T1/T2</td>
<td>T1i</td>
<td>T1f</td>
<td>T2i</td>
</tr>
<tr>
<td>1</td>
<td>MVC/MVC</td>
<td>20/200</td>
<td>20/25</td>
<td>20/400</td>
</tr>
<tr>
<td>2</td>
<td>Assault/assault</td>
<td>CF</td>
<td>20/20</td>
<td>NLP</td>
</tr>
<tr>
<td>3</td>
<td>Trauma/assault</td>
<td>20/100</td>
<td>20/50</td>
<td>20/70</td>
</tr>
<tr>
<td>4</td>
<td>MVC/other</td>
<td>CF</td>
<td>20/40</td>
<td>CF</td>
</tr>
<tr>
<td>5</td>
<td>MVC/assault</td>
<td>20/400</td>
<td>20/40</td>
<td>NLP</td>
</tr>
<tr>
<td>6</td>
<td>Assault/MVC</td>
<td>20/100</td>
<td>20/50</td>
<td>20/200</td>
</tr>
<tr>
<td>7</td>
<td>Assault/MVC</td>
<td>20/70</td>
<td>20/30</td>
<td>NLP</td>
</tr>
<tr>
<td>8</td>
<td>Assault/MVC</td>
<td>20/200</td>
<td>20/30</td>
<td>20/400</td>
</tr>
<tr>
<td>9</td>
<td>Assault/other</td>
<td>CF</td>
<td>20/200</td>
<td>CF</td>
</tr>
<tr>
<td>10</td>
<td>Trauma/MVC</td>
<td>20/70</td>
<td>20/40</td>
<td>NLP</td>
</tr>
<tr>
<td>11</td>
<td>Trauma/MVC</td>
<td>20/200</td>
<td>20/50</td>
<td>20/400</td>
</tr>
<tr>
<td>12</td>
<td>MVC/assault</td>
<td>20/100</td>
<td>20/50</td>
<td>20/200</td>
</tr>
</tbody>
</table>

Under the assumption that the type of intervention may not make much difference in ultimate vision recovery, recent TON management debate has focused on the safety of treatment options. That is, if all treatments provide roughly the same outcome, should we not choose the safest treatment? This thinking and our efficacy analysis, however, may be shortsighted, lacking consideration of optic nerve resilience against further insult. Herein, we present a case series of catastrophic, unrecoverable vision loss from second optic nerve insults following initial vision recovery from TON, suggesting increased optic nerve vulnerability and raising further questions of the stability of regained vision following TON and which treatment(s) might provide the most optic nerve protection against future injury.

Methods

A retrospective medical record review was performed on patients referred to a single tertiary care oculoplastic practice and treated from 1994 through 2011 for second TON injuries. Patients in the same practice treated surgically for primary TON were also identified for outcome comparison. Institutional review board approval and informed consent from the patients were not required for a retrospective medical record review at the private tertiary referral practice.

All patients with primary or secondary injuries were provided a detailed review of the practice’s experience, as well as a summary of the literature to date, and then offered observation alone, high-dose corticosteroids, or high-dose corticosteroids and optic canal decompression. High-dose corticosteroid treatment consisted of one 1.5-mg/kg intravenous dose of methylprednisolone followed by 5.4 mg/kg/h for 72 hours if there was no improvement in vision, until vision improvement stabilized, or until surgery.

Snellen chart visual acuities were measured at presentation, twice daily while taking intravenous corticosteroids, on the day following surgery (if performed), and following completion of corticosteroids or surgery after 1 day, 1 week, 1 month, and 3 months. For the purposes of analysis, changes in visual acuities were assigned line improvement scores as a difference in the number of lines shown on a Snellen eye chart between visits. Beyond 20/400, counting fingers, light perception, and no light perception were each counted as additional “lines” of vision.

Using paired 2-tailed t tests, Snellen chart visual acuities, and assigned line improvement scores, pretreatment and post-treatment vision change following the first injury, pretreatment and posttreatment vision change following the second injury, posttreatment vision change after the first and second injuries, and posttreatment and pretreatment vision change after the first and second injuries were compared.

Optic canal decompressions were performed using a combined endonasal endoscopic and transconjunctival approach to achieve 120° to 180° of optic nerve exposure.

Results

A total of 131 patients were identified who were seen in the 18-year period from 1994 through 2011 for treatment of TON. Twelve patients had a previous incidence of TON and sought treatment for a second optic nerve insult. Among the 12 patients with secondary TON, 10 (83.3%) were men, the mean age was 42 years (range, 26-51 years), mean visual acuity at presentation was between 20/400 and counting fingers, mean length of time between TON events was 7 years (range, 2-13 years) (Table 1), mean loss in vision following the second insult was 5.6 lines (range, 1-11), and mean follow-up was 11.3 months (range, 1-26 months). Of these patients, 10 (83.3%) re-
ceived optic canal decompression, with 2 (16.7%) having a minimal vision improvement of 1 line and 2 (16.7%) having improvement of 2 lines. Two patients (16.7%) were treated with high-dose corticosteroids alone, neither of whom had any vision improvement.

One of the 12 patients (8.3%) with secondary TON had previously undergone attempted optic canal decompression after the first injury, but the decompression involved only a small anterior portion of the optic canal with roughly only 30° of radial decompression. Other treatments following the initial TON included methylprednisolone per the Second National Acute Spinal Cord Injury Study (5 [41.7%]), some other corticosteroid regimen (4 [33.3%]), and unknown treatment (3 [25.0%]).

Among the 12 patients with secondary TON, the most common mechanism of injury for their first TON event was aggravated assault (5 [41.7%]), motor vehicle crash (4 [33.3%]), and trauma (3 [25.0%]), whereas second injuries were ascribed to motor vehicle crash (6 [50.0%]), assault (4 [33.3%]), and minor trauma (2 [16.7%]) from a tether ball to the mid-glabella or banging the head against an open kitchen cupboard door.

Of the 115 patients with primary TON, 91 (79.1%) underwent surgical decompression, with 75 (82.4%) achieving improved vision with a mean of 5 lines of improvement and 74 (65.2%) of those with vision improvement after surgery having some improvement within the first 24 hours. In contrast, 24 patients (20.9%) with primary TON declined surgery and received corticosteroids alone. Only 10 (41.6%) had vision improvement with a mean of greater than 5 lines of improvement.

In the patients with secondary TON, there was a significant improvement in lines of vision following the initial treatment (P = .004); however, following the second insult, vision typically fell to or below that seen after the first injury, and no substantive improvement was obtained following treatment for the second insult (P = .05).

### Discussion

The management of TON, a potentially devastating injury with risk for irreversible vision loss, remains controversial, with some recommending no intervention, others strongly advocating high-dose corticosteroids with or without optic canal decompression, and still others emphasizing a necessity of rapid, early treatment. Perhaps the wide disparity in vision recovery success reported with various interventions reflects in part not only the mechanism of the TON but also patient characteristics not yet elucidated. One such characteristic may be the “resilience” of the optic nerve, which could depend on multiple factors, such as overall microvascular health, a tendency toward regional edema, general nutritional status of the patient, or previous optic nerve injury. A related characteristic might be the amount of anatomic redundancy of undamaged optic nerve fibers that provide no perceptible increase in optic nerve function, which we term optic nerve reserve. Optic nerve reserve, or the ability to lose a degree of neuronal anatomy without losing nerve function, is well recognized throughout the body.

Frisén and Quigley explored the idea of ophthalmic neuronal reserve and showed that a patient may have 20/20 visual acuity despite losing more than 40% of retinal or optic nerve neural channels, whereas a visual acuity of 20/50 on the Snellen eye chart represented a 90% loss of neural channels. They suggested that below a certain threshold of neuronal channel loss, linear neural loss creates a nonlinear vision loss.

Herein, we reviewed all medical records of patients with TON treated by a tertiary care oculoplastic surgical practice in an 18-year period, and we were surprised to find that 12 of 131 patients (9.2%) had had documented ipsilateral optic nerve injury with vision loss. Following their first injury, all 12 patients had some degree of vision improvement, but 2 to 15 years later (mean, 7 years), each had a presumed second optic nerve insult, and vision fell to or below the loss incurred from the initial injury (Table 2). Furthermore, only 4 of 12 patients (33.3%) recovered any vision after treatment of their second injury, and in all patients, the returned vision did not exceed the vision measured directly after the first injury.

In comparing the outcome of these 12 patients with secondary TON with that of 115 patients with primary TON treated in the same practice, 10 of 12 patients (83.3%) with secondary TON elected optic canal decompression, whereas only 91 (79.1%) patients with primary TON underwent surgery. Nevertheless, among the patients with secondary TON, only 3 (25.0%) showed marginal vision improvement, whereas among those with primary TON, 82.4% demonstrated better vision postoperatively.

This finding raises several questions. First, did the patients with a second injury have optic nerve reserve loss during their first injury, making them more susceptible to further unrecoverable vision loss from a second injury, many of which appeared to be quite minor? Second, since none of the 12 patients with secondary TON received meaningful optic canal decompressions (1 patient received 30° decompression) in

<table>
<thead>
<tr>
<th>Interval</th>
<th>Analysis of Mean Lines of Vision</th>
<th>P Value</th>
<th>Interpretation of Vision Change</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>First injury pretreatment vs first injury posttreatment</td>
<td>.004</td>
<td>Improved</td>
</tr>
<tr>
<td>II</td>
<td>First injury posttreatment vs second injury pretreatment</td>
<td>&lt;.001</td>
<td>Worsened</td>
</tr>
<tr>
<td>III</td>
<td>Second injury pretreatment vs second injury posttreatment</td>
<td>.05</td>
<td>Unimproved</td>
</tr>
<tr>
<td>IV</td>
<td>First injury pretreatment vs second injury pretreatment</td>
<td>.01</td>
<td>Worsened</td>
</tr>
<tr>
<td>V</td>
<td>First injury pretreatment vs second injury posttreatment</td>
<td>.05</td>
<td>Unimproved</td>
</tr>
</tbody>
</table>

* Demonstrates the change in vision at the indicated time intervals. Taken across all patients with 2 injuries, vision following the second injury is worse than that seen following the first injury, and vision after treatment of the second injury is no better than the vision seen before treatment of the first injury.
the treatment of their first injuries, might a more complete can- nal decompression (120°-180°) be more protective against fur- ther vision loss? Third, should we be recommending constant safety eyeglass use with polycarbonate lenses and titanium frames for all patients who have had TON and re- gained vision to protect their uninjured eye since the eye with TON may be at higher risk for further vision loss? In several studies, including that by Frisén and Quigley, an instability of regained vision in patients with TON treated with cortico- steroids alone has been reported. Fourth, in practices where optic canal decompressions are offered as a treatment option, should patients with known previous optic nerve inju- ries be excluded? Fifth, if we dig deeply enough into patients’ histories, can we better identify and stratify those who have had a previous optic nerve injury and make further studies on TON management outcome analysis more meaningful? Although very intriguing, this study should be used pri- marily as a springboard for further thought and prospective in- vestigation since it has many potential flaws. First, the data may be confounded by the retrospective analysis, study size, and probable referral bias. Although 9.2% of our patients with TON had a previous optic nerve injury, 12 patients is nonethe- less a small cohort, and it remains unclear whether this high percentage is typical of a national average or reflects local re- ferral patterns of patients with profound vision loss to a ter- tiary care practice. In fact, given the retrospective character of this study, the true percentage of second injuries may be even higher if more in-depth medical history questioning had been done. This possibility is further highlighted by the fact that all previous TON events identified in our medical records in- volved the same eye injured a second time, raising the ques- tion of whether histories of contralateral optic nerve injury might have been neglected and missed. Furthermore, all pa- tients in this study with secondary TON had significant vi- sion return (mean, 4 lines; range, 2-9 lines) after their initial injuries. We suspect that this also indicates referral bias since patients who had previous TON and continuing severe vision loss may not have been referred after a second injury with rela- tively minor additional loss. Second, patients with primary TON and second-insult TON are not well-matched cohorts, with mean ages being 37 and 42 years and lines of vision return af- ter initial injury being almost 6 and 4, respectively. Third, only 1 patient with secondary TON had a return to 20/20 visual acu- ity after the initial injury. Possibly patients with 20/20 visual acuity may have greater preservation of their nerve reserve. Fourth, patients with primary or secondary TON were not stratified in our medical records according to suspected or known mechanism of TON, of which several have been proposed. Fifth, few if any of the patients in this study had rigorous color vision analysis, contrast sensitivity study, optical coherence tomography, electrophysiology, or formal vi- sual fields beyond 30° from fixation to best characterize optic function. Finally, our follow-up for patients with primary TON was only 7 months on average, so our hypothesis that 120° to 180° optic canal decompression may be protective against fur- ther optic nerve TON is conjecture since none of the 12 pa- tients had received such surgery.

Conclusions
To our knowledge, this is the first case series of patients with second-event TON injuries to the same eye. Despite the fact that a patient’s vision might return after an episode of TON, following a second optic nerve insult, high-dose cortico- roids and surgical decompression may not be beneficial. Regard- less of vision recovery, the question is whether patients with primary TON should be offered substantial optic canal de- compression since it might be partially protective against a sec- ond nerve injury. Nevertheless, given the possible lifelong vul- nerability of their injured eye, we strongly encourage all patients with past or current TON to always wear eyeglasses with polycarbonate lenses mounted in a titanium frame to pro- tect their other eye.

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Study concept and design: Soparkar.
Acquisition, analysis, or interpretation of data: All authors.
Drafting of the manuscript: Guy.
Critical revision of the manuscript for important intellectual content: All authors.
Statistical analysis: Guy, Soparkar.
Administrative, technical, or material support: Alford, Sami.
Study supervision: Soparkar, Alford, Patrinely, Parke.
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REFERENCES

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