Ischemic Optic Neuropathy After Lumbar Spine Surgery

David M. Katz, MD; Jonathan D. Trobe, MD; Wayne T. Cornblath, MD; Lanning B. Kline, MD

Objective: Study of clinical features of ischemic optic neuropathy (ION) developing as a complication of multilevel lumbar spine surgery.

Design: Review of all cases of ION that developed within 2 weeks of spine surgery at two academic institutions from 1990 to 1992, and a review of adequately reported cases of ION after other nonophthalmic procedures.

Results: Four new cases are reported in patients who ranged in age from 41 to 65 years. All four had undergone uneventful but prolonged (8 to 9 hours) spine surgery, during which blood pressure was deliberately maintained between 85 and 100 mm Hg systolic and 45 to 65 mm Hg diastolic to reduce bleeding. Hemoglobin values fell 30 to 78 g/L during surgery. Arteriosclerotic risk factors, including systemic hypertension, diabetes, coronary artery disease, and smoking, were present in three cases. There was no evidence of orbital soft-tissue injury, retinal artery occlusion, or other neurologic deficits. The combination of hypertension and anemia has been noted in most of the 30 previously well-documented cases of ION after other nonophthalmic procedures.

Conclusions: Multilevel lumbar laminectomy should be added to the list of procedures that may produce ION as an isolated complication. Deliberate hypotension maintained for long operative periods in patients with arteriosclerotic risk factors may be the cause.

(Arch Ophthalmol. 1994;112:925-931)

Infarction of the optic nerve (ischemic optic neuropathy [ION]) usually occurs as an acute, spontaneous event in middle-aged and elderly individuals.1-5 Occlusion of one or more small ciliary arterioles in the optic nerve head is responsible, and hypertension and other arteriosclerotic risk factors are often documented.

A similar condition has been reported as an isolated complication of cardiopulmonary bypass procedures,6-15 bilateral radical neck dissection,16-21 various abdominal procedures,22-34 therapeutic phlebotomy,35 hemodialysis,36 cardiac arrest,37 hip surgery,38 partial pneumonectomy, mitral valvulotomy,40 cholecystectomy,41 parathyroidectomy, and coronary angiography.39 In many cases, perioperative hemorrhage and hypotension have been implicated; in some cases, there is no explanation. Visual loss after recurrent gastrointestinal tract and uterine hemorrhage, reported since the time of Hippocrates,35,38,39 has also been attributed to infarction of the optic nerve.22-25,28,40,41

We describe four patients who developed ION after uneventful multilevel lumbar laminectomy or fusion. This complication has not, to our knowledge, been reported after spine surgery.

RESULTS

All patients complained of unremitting visual loss that had begun immediately or within days of spine surgery. Arteriosclerotic risk factors included hypertension, coronary artery disease, and diabetes mellitus in one patient, diabetes

See Patients and Methods on next page
The four patients (Table 1) were referred for consultative examination to the Neuro-ophthalmology Clinics of the University of Michigan, Ann Arbor (patients 1 through 3) and the University of Alabama, Birmingham (patient 4) between 1990 and 1992. There were three women and one man, ranging in age from 41 to 65 years.

mellitus and heavy smoking in one patient, and hypertension in another. The preoperative hemoglobin level was normal in all patients, who had undergone multilevel lumbar laminectomy or fusion in procedures that lasted from 8 to 9 hours without documented untoward events. Intraoperative blood pressure was maintained between 85 and 100 mm Hg systolic and between 45 and 60 mm Hg diastolic to reduce bleeding. In one patient, a hypotensive agent was used to lower blood pressure; in the other three patients, blood pressure drifted down as the result of general anesthesia and blood loss. Estimated blood loss varied from 0.5 to 2 L; the hemoglobin level decreased by 30 to 78 g/L. Two patients received blood transfusions of 1 to 2 U, but the operating teams acknowledged a reluctance to transfuse owing to a fear of human immunodeficiency virus contamination.

The visual loss occurred on the day of surgery in two patients, and 2 days and 12 days after surgery in the other two patients. Visual acuity was severely impaired in five of six affected eyes; the sixth eye had 20/40 visual acuity but had substantial visual field loss. Both eyes were affected in two patients. Improvement in visual acuity occurred in two eyes, but severe visual field defects persisted in five eyes with a follow-up period ranging from 2 weeks to 6 months (Figure 1). The optic discs appeared normal in one patient and were swollen in the other three patients (Figure 2). In three eyes of two patients, optic disc swelling was mild.

There were no other clinical signs of ischemia. Brain images were normal in the three patients in whom they were obtained.

Two factors appeared causal in our four patients: (1) anemia induced by blood loss, which the operating teams were reluctant to remedy by transfusion because of the risk of human immunodeficiency virus contamination, and (2) intraoperative hypotension maintained deliberately to minimize blood loss. Arteriosclerotic risk factors were documented in three of four patients. The contribution of prone positioning during surgery is unknown, but no patient had evidence of swelling of orbital symptom by an ill

Table 1. Four Cases of Ischemic Optic Neuropathy After Lumbar Spine Surgery

<table>
<thead>
<tr>
<th>Patient/Age, y/Sex</th>
<th>Procedure</th>
<th>Vascular Risk Factors</th>
<th>Complications or Causative Factors</th>
<th>Intraoperative BP, mm Hg</th>
<th>Hemoglobin, g/L</th>
<th>Estimated Blood Loss, L</th>
<th>Fluid Replacement</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/49/F</td>
<td>L3-5</td>
<td>HTN</td>
<td>Intraoperative hypotension</td>
<td>85-100/50-60</td>
<td>134/104</td>
<td>2.0</td>
<td>1 U PRBC, 3 L NS</td>
</tr>
<tr>
<td>2/65/F</td>
<td>L3-4</td>
<td>None</td>
<td>Intraoperative hypotension</td>
<td>90-95/50-55</td>
<td>140/80</td>
<td>0.5</td>
<td>3.8 L LR</td>
</tr>
<tr>
<td>3/60/F</td>
<td>L3-4</td>
<td>NIDDM, HTN, CAD</td>
<td>Intraoperative hypotension</td>
<td>95-105/45-55</td>
<td>164/86</td>
<td>2.0</td>
<td>5.5 L LR, 0.5 L GS, 0.5 L HS</td>
</tr>
<tr>
<td>4/41/M</td>
<td>L4-5-S1</td>
<td>IDDM, smoking (50 pack-y)</td>
<td>Deliberate intraoperative hypotension</td>
<td>85-100/50-60</td>
<td>153/117</td>
<td>2.0</td>
<td>2 U WB, 7 L LR, 0.25 L HS</td>
</tr>
</tbody>
</table>

*BP indicates blood pressure; MR, magnetic resonance; HTN, hypertension; NIDDM, non-insulin-dependent diabetes mellitus; CAD, coronary artery disease; IDDM, insulin-dependent diabetes mellitus; PRBC, packed red blood cells; NS, normal saline; LR, lactated Ringer's solution; CS, cell saver; HS, heparin; WB, whole blood; CF, counting fingers; F/U, follow-up; HM, hand motions; and NLP, no light perception.
†Preoperative/postoperative.
of orbital soft tissues or central retinal artery occlusion features associated with direct ocular compression by an ill-fitting headrest.\textsuperscript{12–14}

In our review of the 30 adequately documented cases of ION associated with various systemic procedures (Table 2), hypotension and perioperative blood loss were the factors most credibly implicated as the cause of visual loss. Among the 19 cases of ION after coronary artery bypass graft surgery,\textsuperscript{5–9,11–13} significant hypotension was documented in seven cases, denied

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure1.png}
\caption{Visual fields of patients 1 (left), 2 (center), and 3 (right).}
\end{figure}

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure2.png}
\caption{Optic discs of patient 2.}
\end{figure}

\begin{table}[h]
\centering
\begin{tabular}{|c|c|c|c|c|c|c|}
\hline
Fluid Replacement & Latency of Visual Complaint & Eye Involved & Visual Acuity & Visual Field & Afferent Pupillary Defect & Optic Disc Appearance & Other Clinical Manifestations & MR Brain Imaging \\
\hline
1 U PRBC, 3 L NS & Same day & OU & Initial, 28/800 OD; final, 20/25 OD; CF OS; F/U time, 4 wk & Figure 1, left & None & Normal OU & None & Normal \\
\hline
2 d & OU & Initial, HM OD, 20/80 OS; final, HM OD, 20/30 OS; F/U time, 2 wk & Figure 1, center & OD & Pale swelling OU (Figure 2) & None & Normal \\
\hline
3.8 L LR & 12 d & OS & 20/15 OD, 20/40 OS & Figure 1, right & OS & Pale swelling OS & None & Not done \\
\hline
5.5 L LR, 0.5 L CS, 0.5 L HS & 1 d & OD & Initial, NLP OD, 20/20 OS; final, same; F/U time, 6 mo & Not done & OD & Pale swelling OD & None & Normal \\
\hline
2 U WB, 7 L LR, 0.25 L CS & & & & & & & & \\
\hline
\end{tabular}
\caption{Table 2: Summary of cases of ION associated with various systemic procedures.}
\end{table}
<table>
<thead>
<tr>
<th>Patient/Age, y/ Sex</th>
<th>Procedure</th>
<th>Vascular Risk Factors</th>
<th>Complications or Causative Factors</th>
<th>Latency of Visual Complaint</th>
<th>Eye Involved</th>
<th>Visual Acuity</th>
<th>Visual Field</th>
<th>Optic Disc Appearance</th>
<th>Other Clinical Manifestations</th>
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<tbody>
<tr>
<td>1/44/M M</td>
<td>CABG</td>
<td>CAD</td>
<td>Hypotension caused by hemorrhage</td>
<td>&lt;2 d OU</td>
<td>NR</td>
<td>Preserved inferior quadrants OD, absent OS OU</td>
<td>Pale swelling OU</td>
<td>None</td>
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<tr>
<td>2/61/M M</td>
<td>CABG</td>
<td>CAD</td>
<td>Hypotension caused by hemorrhage</td>
<td>&lt;2 d OD</td>
<td>NR</td>
<td>Superior altitudinal defect OS</td>
<td>Pale swelling OD</td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>3/61/M M</td>
<td>CABG</td>
<td>CAD, DM, HTN</td>
<td>Hypothermia (28°C)</td>
<td>&lt;2 d OD</td>
<td>NR</td>
<td>Superior altitudinal defect OS</td>
<td>Pale swelling OD</td>
<td>None</td>
<td></td>
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<tr>
<td>4/50/M M</td>
<td>CABG</td>
<td>CAD, DM</td>
<td>Hypothermia (28°C)</td>
<td>&lt;2 d OU</td>
<td>NR</td>
<td>Preserved temporal island OD, superior altitudinal defect OS</td>
<td>Pale swelling OU</td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>5/47/M M</td>
<td>CABG</td>
<td>CAD, DM, HTN</td>
<td>Hypotension caused by hemorrhage</td>
<td>&lt;2 d OU</td>
<td>NR</td>
<td>Preserved superior island OD, inferior altitudinal defect OS</td>
<td>Pale swelling OU</td>
<td>None</td>
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</tr>
<tr>
<td>6/50/M M</td>
<td>CABG</td>
<td>CAD</td>
<td>Hypothermia (28°C)</td>
<td>&lt;2 d OS</td>
<td>NR</td>
<td>Inferior altitudinal defect OS</td>
<td>Pale swelling OD</td>
<td>None</td>
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<tr>
<td>7/60/M M</td>
<td>CABG</td>
<td>CAD, HTN, bilateral ICA occlusion</td>
<td>Hypothermia (28°C)</td>
<td>7 d OD</td>
<td>NR</td>
<td>Superior altitudinal defect OS</td>
<td>Pale swelling OD</td>
<td>None</td>
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<tr>
<td>8/46/M M</td>
<td>CABG</td>
<td>CAD</td>
<td>Hypothermia (28°C)</td>
<td>2 d OU</td>
<td>Initial, CF; OU; final, same; F/U, 10 wk</td>
<td>Superior altitudinal defect OD, inferior altitudinal defect OS</td>
<td>Pale swelling OU</td>
<td>None</td>
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<tr>
<td>9/63/M M</td>
<td>CABG</td>
<td>CAD</td>
<td>NR</td>
<td>7 d OU</td>
<td>Initial, 20/400 OD, final, same</td>
<td>Superior altitudinal defect OD, central scotoma OS</td>
<td>Pale swelling OU</td>
<td>None</td>
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</tr>
<tr>
<td>10/56/M M</td>
<td>CABG</td>
<td>CAD, HTN, smoking</td>
<td>None</td>
<td>4 d OU</td>
<td>Initial, 20/400 OD, final, same; 20/20 OD, 20/30 OS; F/U, 2 mo</td>
<td>Superior altitudinal defect OD, inferior altitudinal defect OS</td>
<td>Pale swelling OD</td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>11/44/M M</td>
<td>CABG</td>
<td>CAD, HTN, smoking</td>
<td>None</td>
<td>4 d OS</td>
<td>Initial, 20/40 OD (amblyopia), 20/30 OS; final, same; F/U, 2 wk</td>
<td>Superior altitudinal defect OS</td>
<td>Pale swelling OS</td>
<td>NR</td>
<td></td>
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<tr>
<td>12/46/F M</td>
<td>CABG</td>
<td>CAD, HTN, DM, smoking</td>
<td>Innominate vein laceration</td>
<td>Same day OU</td>
<td>Absent OU</td>
<td>Normal OU</td>
<td>Facial and bilateral upper-extremity weakness, new lacunar infarct on CT</td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>13/54/M M</td>
<td>CABG</td>
<td>CAD</td>
<td>bilateral severe ICA stenosis</td>
<td>NR</td>
<td>2d OD</td>
<td>Nasal defect</td>
<td>Superior disc edema OD</td>
<td>None</td>
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</tr>
</tbody>
</table>

**Table 2. Reported Cases of Ischemic Optic Neuropathy After Other Nonophthalmic Procedures**
### Table 2. Reported Cases of Ischemic Optic Neuropathy After Other Nonophthalmic Procedures* (cont)

<table>
<thead>
<tr>
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</thead>
<tbody>
<tr>
<td>1/4/64/M CABG[9]</td>
<td>CAD</td>
<td>NR</td>
<td>3 d</td>
<td>OS</td>
<td>initial, 20/40 OS; final, same; F/U, 7 d</td>
<td>Inferior altitudinal defect</td>
<td>Superior disc edema OS</td>
<td>None</td>
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</tr>
<tr>
<td>1/6/63/M CABG[9]</td>
<td>CAD, DM</td>
<td>None</td>
<td>5 d</td>
<td>OU</td>
<td>initial, 20/25 OS; final, same; F/U, 2 wk</td>
<td>Inferior altitudinal defects OU</td>
<td>Superior disc edema OU</td>
<td>None</td>
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</tr>
<tr>
<td>1/6/60/M CABG[5]</td>
<td>CAD</td>
<td>NR</td>
<td>2 d</td>
<td>OD</td>
<td>NR</td>
<td>Superior altitudinal defect</td>
<td>NR</td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>1/7/71/F CABG[6]</td>
<td>CARD</td>
<td>Hypertension postop</td>
<td>4 d</td>
<td>OU</td>
<td>NR</td>
<td>NR</td>
<td>Retinal emboli OS</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1/8/68/F CABG[6]</td>
<td>CARD, HTN</td>
<td>NR</td>
<td>Same day</td>
<td>OD</td>
<td>initial, HM OD</td>
<td>Preserved temporal island</td>
<td>Pale swelling OD</td>
<td>Retinal emboli OU</td>
<td></td>
</tr>
<tr>
<td>2/6/64/M bilateral s.n. neck dissection[9]</td>
<td>Smoking</td>
<td>Significant blood loss, hypotension</td>
<td>Same day</td>
<td>OU</td>
<td>initial, NLP OU; final, same; F/U, 4 mo</td>
<td>Absent OU</td>
<td>None</td>
<td>Facial edema, limited ocular movement, bilateral extensor plantar responses</td>
<td></td>
</tr>
<tr>
<td>2/8/71/M bilateral s.n. neck dissection[9]</td>
<td>Smoking</td>
<td>Hypertension, bilateral hemorrhage</td>
<td>Same day</td>
<td>OU</td>
<td>initial, NLP OU; final, same; F/U, 2 wk</td>
<td>Absent OU</td>
<td>None</td>
<td>Facial and eyelid edema</td>
<td></td>
</tr>
<tr>
<td>2/8/69/F staged bilateral s.n. neck dissection (2 mo)[9]</td>
<td>Smoking</td>
<td>Hypertension</td>
<td>3 d</td>
<td>OU</td>
<td>initial, NLP OU; final, same; F/U, 1 mo</td>
<td>Absent OU</td>
<td>None</td>
<td>Facial and conjunctival edema, proptosis, heart failure</td>
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</tr>
<tr>
<td>2/3/48/M staged bilateral s.n. neck dissection (1 y)[9]</td>
<td>HTN</td>
<td>None</td>
<td>Same day</td>
<td>OU</td>
<td>initial, NLP OU; final, same</td>
<td>Absent OU</td>
<td>Pale swelling OU</td>
<td>Facial edema, eyelid edema, increased intracocular pressure</td>
<td></td>
</tr>
<tr>
<td>2/4/16/F Total parathyroidectomy[9]</td>
<td>None</td>
<td>None</td>
<td>3 d</td>
<td>OU</td>
<td>initial, NR; final, 20/30 OD, 20/15 OS; F/U 1.5 y</td>
<td>Inferior altitudinal defect OD, superior altitudinal defect GS</td>
<td>NR</td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>2/5/62/M Drainage of infected hip prosthesis[9]</td>
<td>None</td>
<td>Shock during intubation</td>
<td>Same day</td>
<td>OU</td>
<td>initial, 20/400 OD, LP OS; final, 20/40 OD, HM OS; F/U, 1 y</td>
<td>Paracentral scotoma OD, absent GS</td>
<td>None</td>
<td>None</td>
<td></td>
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<tr>
<td>2/5/57/F Routine hemodialysis[9]</td>
<td>NR</td>
<td>Hypertension</td>
<td>1 d</td>
<td>OU</td>
<td>initial, 20/30 OD, 20/20 OS; final, same; F/U, 1 wk</td>
<td>Superior and inferior altitudinal defects OD, superior altitudinal defect OS</td>
<td>Pale swelling OU</td>
<td>None</td>
<td></td>
</tr>
</tbody>
</table>

(continued)
in four, and not reported in eight. Intraoperative hypotension often resulted from a complication during surgery, such as perforation of the innominate vein\textsuperscript{14} or other thoracic vessels.\textsuperscript{6} Where hypotension was denied or not reported, significant anemia or hypothermia was noted. Among the four patients whose hemogram was reported, the mean hemoglobin level decreased from 143 to 83 g/L. In the remaining five patients who lacked documented hypotension, anemia, or hypothermia, two also manifested emboli in the retinal arterioles, prompting the consideration of infarction as a possible cause. In four, and not reported in eight, optic nerve infarction was observed. Intraoperative hypotension often resulted from a complication during surgery, such as perforation of the innominate vein\textsuperscript{14} or other thoracic vessels.\textsuperscript{6} Where hypotension was denied or not reported, significant anemia or hypothermia was noted. Among the four patients whose hemogram was reported, the mean hemoglobin level decreased from 143 to 83 g/L. In the remaining five patients who lacked documented hypotension, anemia, or hypothermia, two also manifested emboli in the retinal arterioles, prompting the consideration of infarction as a possible cause. In four, and not reported in eight, optic nerve infarction was observed.

### Table 2. Reported Cases of Ischemic Optic Neuropathy After Other Nonophthalmic Procedures* (cont)

<table>
<thead>
<tr>
<th>Patient/Procedure</th>
<th>Vascular Risk Factors</th>
<th>Complications or Causative Factors</th>
<th>Latency of Visual Complaint</th>
<th>Eye Involved</th>
<th>Visual Acuity</th>
<th>Visual Field</th>
<th>Optic Disc Appearance</th>
<th>Other Clinical Manifestations</th>
</tr>
</thead>
<tbody>
<tr>
<td>27/57/M Mitrval valve-</td>
<td>CAD, congestive heart failure</td>
<td>Pneumothorax, difficult resuscitation after bypass</td>
<td>10 d</td>
<td>OU</td>
<td>Initial, CF OU; final, 20/20 OD, 20/20 OS; F/U, 3 mo</td>
<td>Initial, central scotoma OU; final, constricted field OU</td>
<td>Pale swelling OU</td>
<td>NR</td>
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<tr>
<td>28/52/M Cholecystec-</td>
<td>Wound hemorrhage</td>
<td>3 d and 9 d postop</td>
<td>48 d</td>
<td>OU</td>
<td>Initial, 20/20 OD, NLP OS; final, 20/40 OD, NLP OS; F/U, 3 mo</td>
<td>NR</td>
<td>Disc pallor OU (day 48)</td>
<td>NR</td>
</tr>
<tr>
<td>29/31/M Pulmonary lobec-</td>
<td>Hemorrhage, hypotension</td>
<td>1 d</td>
<td>OU</td>
<td>Left hemiparesis</td>
<td>Absent OU</td>
<td>Pale swelling OU</td>
<td>Left hemiparesis</td>
<td></td>
</tr>
<tr>
<td>30/64/F Coronary catheterization\textsuperscript{56}</td>
<td>CAD, HTN</td>
<td>NR</td>
<td>2 h</td>
<td>OD</td>
<td>Initial, 20/50 OD; final, 20/15 OD; F/U, 1 mo</td>
<td>Centrocoical scotoma</td>
<td>Pale swelling OD</td>
<td>Retinal emboli OD</td>
</tr>
</tbody>
</table>

* CABG indicates coronary artery bypass graft; CAD, coronary artery disease; DM, diabetes mellitus; HTN, hypertension; ICA, internal carotid artery; NR, not recorded; reoop, preoperative; postop, postoperative; CF, counting fingers; F/U, follow-up; NLP, no light perception; HM, hand motions; LP, light perception; and CT, computed tomography.

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We thank John E. McGillicuddy, MD, and Vildan Mel- lin, MD, for their editorial comments.

Reprint requests to W. K. Kellogg Eye Center, 1000 Wall St, Ann Arbor, MI 48105 (Dr Trobe).
**REFERENCES**

9. Shaw PJ, Bates D, Cartlidge NEF, Heaviside D, Julian DG, Shaw DA. Early neuro-ophthalmic risk criteria. Several possibly liberally, several endarterectomies, and several endarterectomies of our patients were the only site of anemia. Several retinal disc edema is immediate postoperative day. The clinical reasons: that this edema was either or were perioperative; retinal edema was the only site where these retinal edema were.

<table>
<thead>
<tr>
<th>Author(s)</th>
<th>Title</th>
<th>Journal</th>
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<tr>
<td>Smith et al.</td>
<td>Retinal ischemia</td>
<td><em>Arch Ophthalmol.</em></td>
<td>1983</td>
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<tr>
<td>Levitan</td>
<td>Atrophy of the optic nerve following hemorrhage</td>
<td><em>Am J Ophthalmol.</em></td>
<td>1983</td>
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<td>Holmes</td>
<td>Loss of vision following hemorrhage</td>
<td><em>Br J Ophthalmol.</em></td>
<td>1983</td>
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<td>de Almeida</td>
<td>Blindness after hemorrhage</td>
<td><em>Ann Ophthalmol.</em></td>
<td>1983</td>
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**Other Clinical Manifestations**

<table>
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<th>Region</th>
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<tr>
<td>Retinal edema</td>
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<tr>
<td>Retinal hemorrhage</td>
<td>1983</td>
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<td>Retinal detachment</td>
<td>1983</td>
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</table>

**Vidal Malovan**

Center, 1000
Fluid Replacement

- 1 U PRBC, 3 L NS
- 3.8 L LR
- 5.5 L LR, 0.5 L CS, 0.5 L HS
- 2 U WB, 7 L LR, 0.25 L CS

Latency of Visual Complaint

<table>
<thead>
<tr>
<th>Fluid Replacement</th>
<th>Eye Involved</th>
<th>Visual Acuity</th>
<th>Visual Field</th>
<th>Afferent Pupillary Defect</th>
<th>Optic Disc Appearance</th>
<th>Other Clinical Manifestations</th>
<th>MR Brain Imaging</th>
</tr>
</thead>
<tbody>
<tr>
<td>Same day</td>
<td>OU</td>
<td>Initial, 20/800 OU; final, 20/25 OD, OS; F/U time, 4 wk</td>
<td>Figure 1, left</td>
<td>None</td>
<td>Normal OU</td>
<td>None</td>
<td>Normal</td>
</tr>
<tr>
<td>2 d</td>
<td>OU</td>
<td>Initial, HM OD, 20/80 OD; final, HM OD, 20/30 OD; F/U time, 2 wk</td>
<td>Figure 1, center</td>
<td>PD</td>
<td>Pale swelling OU (Figure 2)</td>
<td>None</td>
<td>Normal</td>
</tr>
<tr>
<td>12 d</td>
<td>OS</td>
<td>20/15 OD, 20/40 OS</td>
<td>Figure 1, right</td>
<td>CS</td>
<td>Pale swelling OS</td>
<td>None</td>
<td>Not done</td>
</tr>
<tr>
<td>1 d</td>
<td>OD</td>
<td>Initial, NLP OD, 20/20 OS; final, same; F/U time, 6 mo</td>
<td>Not done</td>
<td>PD</td>
<td>Pale swelling OD</td>
<td>None</td>
<td>Normal</td>
</tr>
</tbody>
</table>

Of human surgery in two the other red in five visual acuities were affected by optic nerve swelling from discs in the other patients. Brain swelling of orbital soft tissues or central retinal artery occlusion features associated with direct ocular compression by an ill-fitting headrest.

In our review of the 30 adequately documented cases of ION associated with various systemic procedures (Table 2), hypotension and perioperative blood loss were the factors most credibly implicated as the cause of visual loss. Among the 19 cases of ION after coronary artery bypass graft surgery, significant hypotension was documented in seven cases, denied