Intraocular and optic nerve sheath hemorrhage in cases of sudden intracranial hypertension

PAUL J. MULLER, M.D., AND JOHN H. N. DECK, M.D.

Department of Pathology, Toronto Western Hospital, Toronto, Ontario, Canada

The eyes of 23 patients with sudden intracranial hypertension were studied at post-mortem. Intraocular hemorrhage had occurred in 37% and optic nerve sheath hemorrhage in 87%. Expansion of the optic nerve sheath, particularly the fusiform retrobulbar portion, was a consistent finding. The subdural space of the optic nerve sheath bore the brunt of the hemorrhage which sometimes communicated with perivascular intradural hemorrhages. Optic nerve sheath hemorrhage is shown to result from rupture of dural and bridging vessels of the optic nerve sheath; this we conclude is subsequent to optic nerve sheath dilatation caused by the transmission of intracranial pressure through the subarachnoid communication between the optic nerve sheath and the intracranial cavity. Intraocular hemorrhage is the result of retinal venous hypertension and rupture brought on by obstruction of both the central retinal vein and the retinochoroidal anastomosis.

KEY WORDS • increased intracranial pressure • intraocular hemorrhage • optic nerve sheath hemorrhage

Intraocular and optic nerve sheath hemorrhages have long been recognized as a complication of sudden intracranial hypertension, and their pathogenesis has been frequently reported.\(^1\) \(1,4,8,9,11-13,16-19,21,22\) Intraocular hemorrhages occur in approximately 20% of the patients who suffer a subarachnoid hemorrhage (SAH), and are of prognostic significance;\(^1,11,15,18\) an overall mortality of 25% is reported with SAH, but this rises to 50% when intraocular hemorrhage is present.\(^11\) Furthermore, vitreous hemorrhages may significantly impair vision in patients who survive the intracranial hemorrhage.\(^2,20\)

Controversy about the pathogenesis of these intraocular and optic nerve sheath hemorrhages has centered on the following questions: 1) How are the effects of intracranial hypertension transmitted to the orbit? Is the pressure transmitted via the venous or the subarachnoid connections linking the intracranial cavity and the orbit? 2) What is the origin of optic nerve sheath hemorrhages? Are they simply the result of propagation of intracranial subarachnoid blood through the subarachnoid space of the optic canal or caused by local rupture of vessels within the optic nerve sheath?

We have undertaken the following study to resolve these questions. We also investigated the relationship between cavernous sinus flow and intracranial pressure in the cadaver; the details of this study will be the subject of a separate report.\(^14\)
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Material and Methods

Postmortem examinations were carried out on 46 eyes from 23 patients with sudden intracranial hypertension and, as controls, on 24 eyes from patients with no evidence of intracranial hypertension. At autopsy, the posterior half of the globe of the eye and the intraorbital optic nerve were removed intact and fixed in formalin. In selected cases, the optic canal was also removed in continuity with the intraorbital optic nerve. The maximum retrobulbar diameter of the optic nerve and the diameter 1 cm proximal to the globe were measured. Subsequent to imbedding, the specimens were stained with Mallory's trichrome, hematoxylin and eosin (H & E), and H & E plus Luxol fast blue. All cases of intracranial hypertension had pathological confirmation of cerebral compression; these included four cases of severe cerebral trauma, eight of massive spontaneous intracerebral hemorrhage, nine of ruptured berry aneurysm, and two of internal carotid occlusion resulting in massive postinfarction cerebral swelling without hemorrhage.

Results

Of the 46 eyes from cases of sudden intracranial hypertension, 40 (87%) had optic nerve sheath hemorrhage and 17 (37%) had intraocular hemorrhage. The intraocular hemorrhages were retinal in 16, preretinal in 10, and vitreous in four. The majority of the retinal and preretinal hemorrhages were located at or near the optic disc, but some were more peripheral (Fig. 1). Gross inspection of the optic nerves and their sheaths revealed a bluish discoloration and a distension of the ampullary retrobulbar expansion which is normally present immediately behind the globe of the eye (Fig. 2). The average diameter of the midpoint of the retrobulbar distension in cases of sudden intracranial hypertension was 1.1 mm greater than in the controls. The diameter of the optic nerve sheath 1 cm behind the globe was 0.4 mm greater in the hemorrhagic cases than in the controls.

Some of the optic nerve sheath hemorrhages consisted of small aggregates of erythrocytes, while, in others, densely packed hemorrhage completely filled the subdural and subarachnoid compartments of the optic nerve sheath (Figs. 3 and 4). Hemorrhage was almost always more dense in the subdural than in the subarachnoid space (Fig. 5). The subarachnoid blood tended to be maximal in the ampullary retrobulbar segment of the optic nerve sheath, but was also present in all other segments, including that traversing the optic canal. Intradural hemorrhages were common and were demonstrated microscopically in over half of the optic nerve sheaths; if more sections had been taken, the incidence of intradural hemorrhage would no doubt have been even higher. In many cases, hemorrhages occurred in multiple foci. In some, the intradural hemorrhage had ruptured into the subdural space of the optic

![Fig. 1. Hemisected ocular globe showing large central preretinal hemorrhage and scattered punctate retinal hemorrhages (arrows).](image1)

![Fig. 2. Lateral view of posterior portion of globe and intraorbital optic nerve sheath showing an expanded retrobulbar dilatation (arrows) and bloody discoloration of the optic nerve sheath.](image2)
Fro. 3. Intraorbital optic nerve and sheath transected 15 mm behind the globe of the eye showing gross hemorrhage between the optic nerve and its dural sheath.

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nerve sheath (Fig. 6); in two cases it had ruptured externally to cause extravasation of blood into the orbital adipose tissue.

Discussion

Ballantyne,\textsuperscript{1} in 1943, proposed that intraocular and optic nerve sheath hemorrhage resulted from "a sudden rise in intracranial pressure, causing a stasis in all the venous channels that drain the tissues of the eye and orbit." We have shown in a cadaver model, in which a carotid cavernous fistula was fashioned, that even at the extremes of intracranial hypertension (250 mm Hg) cavernous sinus flow is maintained at 50\% of the control level at an input pressure of 50 cm H\textsubscript{2}O; the cavernous sinus is not obstructable.\textsuperscript{14}

Walsh and Hedges\textsuperscript{21} expanded Ballantyne's thesis by their suggestion that increased in-
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The systemic blood pressure. Later Hayreh and Edwards found a good correlation between ophthalmic vein pressure and CSF pressure but the elevation of ophthalmic vein pressure was not dramatic; they noted that the retinal capillary network and venous system easily withstand 20 mm Hg pressure since this is the normal intraocular pressure. Furthermore, the monkey has no significant ophthalmopterygoid anastomosis, and cannulation of the vein to measure its pressure directly reduces the ophthalmofacial connections. Therefore, elevation of the ophthalmic vein pressure by induced intracranial hypertension in the rhesus monkey is of dubious significance.

Thus, the hypothesis that intracranial venous hypertension is transmitted to the eye and orbit, which results in rupture and hemorrhage, seems to be refuted by the absence of occlusion of the cavernous sinus, the lack of significant elevation of the ophthalmic vein pressure in experimentally induced intracranial hypertension, and the nature of the venous drainage of the eye and orbit. Alternative hypotheses are available. They all depend on the transmission of blood, fluid, or pressure from the intracranial cavity to the optic nerve sheath through the subarachnoid communication in the optic canal.

The venous drainage of the eye in its orbit is illustrated in Fig. 7 and has been reviewed in detail by Last. The ophthalmic vessels drain into the cavernous sinus and the facial and pterygoid veins. The central retinal vein always has a major connection with the superior ophthalmic. Thus, any venous obstruction at the level of the cavernous sinus would not be expected to affect the central retinal vein, whose outflow would escape via the ophthalmic vein into the facial and pterygoid vessels. Furthermore, transmission of venous hypertension from the cavernous sinus into the orbital vessels would be dissipated by the same route before significant retinal venous hypertension occurred.

Direct measurements of ophthalmic vein pressure in the rhesus monkey have been made at various levels of intracranial pressure. Hedges, et al. found a rise in ophthalmic vein pressure when the intracranial pressure approached the level of the systemic blood pressure.
The fact that hemorrhage of the optic nerve sheath is not simply the extension of intracranial subarachnoid blood through the optic canal is borne out by two observations. Intraocular and optic nerve sheath hemorrhages have been reported in cases of sudden intracranial hypertension in which there has been no intracranial bleeding; in our series, one of the four eyes from two patients with cerebral swelling secondary to internal carotid artery occlusion had optic nerve sheath hemorrhages. Moreover, the subdural and not the subarachnoid compartment of the optic nerve sheath bears the brunt of the hemorrhage.

We therefore propose that optic nerve sheath hemorrhage originates from the rupture of intradural vessels and bridging vessels running from the dura to the pia mater of the optic nerve (Fig. 8). The dilatation of the optic nerve sheath suggests this phenomenon, and the demonstration of perivascular intradural hemorrhages that communicate with the subdural space of the optic nerve sheath confirms it (Fig. 6).

Intraocular hemorrhages result from the obstruction of the central retinal vein. For retinal venous hypertension and subsequent hemorrhage to occur, the central retinal vein must be obstructed before it communicates with anastomotic channels which could re-route the retinal venous drainage. The obstruction must not only occur before the central retinal vein communicates with the superior ophthalmic, but it must also occur at the level of the retinochoroidal anastomosis. This choroidal drainage via ciliary vessels will allow adequate retinal venous drainage to take place through the retinochoroidal anastomosis when the central retinal vein is obstructed proximally. Hayreh tried unsuccessfully to produce a

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**Fig. 7.** Schematic diagram of the venous drainage of the eye and orbit (modified after Wolff).

**Fig. 8.** A schematic drawing of the intraorbital and intracanalicular portion of the optic nerve and its sheath. The intracranial subarachnoid space communicates with the optic nerve sheath subarachnoid space. The rupture of bridging vessels results in optic nerve sheath hemorrhage. The retinochoroidal anastomosis is depicted. CRV = central retinal vein; CRA = central retinal artery.
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hemorrhagic retinopathy in the rhesus monkey by tying off the central retinal vein at its exit from the optic nerve. Fujino, et al. on the other hand, produced intraocular hemorrhage by obstructing the central retinal vein in such a way as to eliminate the retinochoroidal anastomosis.

The effusion of clear or bloody CSF into the optic nerve sheath through the optic canal's subarachnoid communication with the intracranial cavity in cases of sudden intracranial hypertension results in compression of the central retinal vein and dilatation of the optic nerve sheath. The fusiform retrobulbar portion of the optic nerve sheath, which dilates the most, compresses and obstructs the retinochoroidal anastomosis that lies just anterior to the subarachnoid cul-de-sac; thus, the venous drainage of the eye is markedly reduced with subsequent retinal venous hypertension and hemorrhage. The effect of reduced retinal venous drainage on the retinal venous pressure may be compounded by the reflex sympathetic hypertension that commonly occurs in cases of severe intracranial hypertension. Good correlation between ophthalmic artery pressure and systemic blood pressure has been demonstrated, and a preferential blood flow into the ophthalmic artery during intracranial hypertension has been proposed.

The difference in the incidence of intraocular and optic nerve sheath hemorrhage (the latter is 2 to 3 times more common) is simply explained. The pressure within the optic nerve sheath may be sufficient to cause its dilatation and subsequent rupture of the optic nerve sheath vessels, but still be insufficient to obstruct both the central retinal vein and the retinochoroidal anastomosis. The retinochoroidal anastomosis, lying outside the subarachnoid space near the sclerodural junction, is not as susceptible to compression as is the central retinal vein within the subarachnoid space of the optic nerve sheath. As long as the retinochoroidal anastomosis is patent, retinal venous runoff by this route will prevent the generation of sufficient retinal venous pressure to cause rupture and hemorrhage.

The clinical correlate of optic nerve sheath hemorrhage is not clear. Walsh and Hoyt state that the sudden onset of visual blurring in some SAH cases may be due to hemorrhage within the optic nerve. We demonstrated no intraneural optic nerve bleeding. The visual blurring may be due to compression of the intraorbital optic nerve subsequent to increased pressure within the optic nerve sheath, an effect that may be compounded by the optic nerve sheath hemorrhage.

Summary

Intraocular hemorrhages are not uncommon complications of sudden intracranial hypertension. Optic nerve sheath hemorrhages occur in the majority of cases. We have examined the major pathogenetic theories as to the origin of these hemorrhages, and on the basis of gross and histological examination of 46 eyes from cases of sudden intracranial hypertension, as well as a cadaver cavernous sinus effusion study and review of the venous drainage of the eye and orbit, conclude that intraocular and optic nerve sheath hemorrhages result from the transmission of intracranial pressure into the optic nerve sheath through the subarachnoid communication in the optic canal and not via vascular routes.

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References


Address reprint requests to: J. H. N. Deck, M.D., Department of Pathology, Toronto Western Hospital, Toronto, Ontario, Canada.