A Model for the Mechanism of Optic Nerve Sheath Fenestration

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- The mechanism by which optic nerve sheath fenestration relieves papilledema associated with increased intracranial pressure has not been clearly defined. A model was constructed to determine if the induction of fluid flow along the nerve sheath and through the fenestration could account for a reduction in pressure around the optic nerve, as might be expected according to Bernoulli’s equation of fluid dynamics. The model states that as the velocity of a fluid increases, the pressure it exerts decreases. The model simulated fluid spaces corresponding to the intracranial vault, chiasm, and optic nerves. The unfenestrated model showed direct transmission of elevated intracranial pressure to the nerves, consistent with the production of papilledema. When one nerve was fenestrated, fluid flow along the nerve was initiated and pressure in that nerve sheath dropped substantially. In addition, pressure in the unfenestrated sheath dropped due to fluid communication across the chiasm. These lower intrasheath pressures were consistent with the bilateral resolution of papilledema after unilateral fenestration. The reduced intrasheath pressures persisted even when the intracranial pressure was again elevated to pathologic levels as long as active fluid flow continued along the nerve sheaths. Thus, induction of cerebrospinal fluid flow along the optic nerve sheath by fenestration appears to locally reduce the pressure around the optic nerve, in spite of persistently elevated intracranial pressure. This is in accordance with what would be predicted by Bernoulli’s equation.


Clinical experience and recent articles have substantiated the efficacy of early optic nerve sheath fenestration in the management of visual loss related to increased intracranial pressure (ICP), especially in pseudotumor cerebri. Many generalized symptoms, such as nausea, vomiting, and headache, are also improved by the procedure, at least temporarily. A bilateral effect has been noted after unilateral fenestration.

Two mechanisms of action have been proposed. One is that the nerve sheath fenestration allows filtration of cerebrospinal fluid (CSF) with a reduction in ICP. The other mechanism is that the operation produces fibrosis, which isolates the nerve from the increased ICP. Filtration seemed the most reasonable mechanism to account for the rapidity of onset of improvement, the improvement in generalized symptoms, and the bilateral effect. However, observations of persistently increased ICP and the return of generalized symptoms postoperatively raised questions about this mechanism.

The possibility arose that the fenestration generates CSF flow adjacent to the nerve with a concomitant reduction of local pressure around the nerve while ICP remains elevated. Bernoulli’s equation of fluid dynamics would seem to account for this phenomenon. The equation, simplified, states that hydrostatic pressure decreases when the velocity of the fluid increases. We constructed a model to test this hypothesis.

MATERIALS AND METHODS

Intracraniat volume is estimated to be 1900 cm³ and the optic nerve sheath diameter is approximately 4 mm. The length of the optic nerve from the posterior globe to the chiasm is about 50 mm.

A vessel of approximately 1900 cm³, representing the intracranial volume, was obtained. This vessel was sufficiently high to generate a fluid pressure at its base of approximately 210 mm H₂O, consistent with elevated ICP seen in pseudotumor cerebri. A manometer was connected at the vessel base to measure the ICP (Fig 1).

Previous studies have suggested a fluid communication of the two optic nerves via the chiasm. Thus, at the base, fluid entered an optic nerve-sized "Y" connector, simulating the chiasm. Immediately distal to the chiasm, plastic tubing of 4 mm in diameter was used to simulate the optic nerves. Each tube measured 60 mm. Manometers were placed just distal to the chiasm along each nerve to monitor the fluid pressure "seen" by each nerve. Clamps were placed at the end of each tube to create a closed three-compartment subarachnoid system, consisting of the brain and two optic nerve spaces. A 30-mL syringe filled with fluid was placed at the top of the large vessel such that once the fluid was initially decreased slightly by "fenestration," the ICP could be again elevated to pathologic levels and maintained while fluid was filtered through the nerve tubes.

Several simulations were made, including transmission of ICP to the nerves in the closed system, a unilateral optic nerve sheath fenestration with secondary reduction of ICP, unilateral optic nerve sheath fenestration with return of ICP to elevated levels, and bilateral optic nerve sheath fenestrations.
Fig 1.—Model for pressure reduction in optic nerve sheath fenestration.

RESULTS

In the closed system, the ICP of 210 mm H₂O was transmitted to the “optic nerve” manometers. This represents the unfenestrated steady state of elevated ICP, as in pseudotumor cerebri (Fig 2). When the clamp on one optic nerve was released, simulating fenestration, ICP began dropping slowly to 200 mm H₂O, but the fenestrated optic nerve sheath pressure dropped rapidly to 55 mm H₂O. The pressure in the “unfenestrated” nerve sheath dropped to 160 mm H₂O, a drop of 40 mm H₂O (Fig 3).

Dye infusion into the unfenestrated sheath showed turbulent flow at its “chiasm” junction, then flow of dye downstream along the fenestrated nerve. Dye instilled in the cranial bottle showed no evidence of directional flow after simulated nerve sheath fenestration, except for the fluid within 1 cm of the chiasm opening. Dye in this small, localized area moved slowly into the chiasm and down the fenestrated nerve sheath.

When the cranial bottle was refilled to the 210-mm level, pressure in the fenestrated nerve remained at 55 mm H₂O while the pressure in the unfenestrated nerve rose to 170 mm H₂O, continuing the 40-mm H₂O reduction in pressure that this system allowed (Fig 4). When the second nerve sheath was fenestrated, the pressure in that nerve also dropped to 55 mm H₂O, in spite of maintaining ICP at 210 mm H₂O (Fig 5).

COMMENT

The bilateral effects of unilateral optic nerve sheath fenestration and the persistent relief from papilledema, in spite of a documented return of ICP to elevated levels, have posed troubling questions regarding the mechanism of action of optic nerve sheath fenestration. This model illustrates that Bernoulli’s equation of fluid dynamics may well explain this phenomenon. Bernoulli’s equation states that if an incompressible fluid is in streamline flow, then the quantity \( \frac{1}{2} \rho v^2 + \rho g h + P \) is constant at every point in the fluid. Thus, \( \frac{1}{2} \rho v^2 + \rho g h + P = \frac{1}{2} \rho v'^2 + \rho g h' + P' \), where \( \rho \) indicates the density of the fluid; \( v \), the velocity of the fluid; \( h \), the height above some reference level; \( g \), the acceleration due to gravity; and \( P \), the pressure exerted by the fluid.\(^\text{11}\) If the height is constant, as it would be for fluid moving virtually horizontally from the chiasm to the optic nerve head, the equation becomes \( \frac{1}{2} \rho v'^2 + P' = \frac{1}{2} \rho v^2 + P \), or the pressure decreases as the velocity of the fluid increases.

Previous studies have documented communications between intracranial CSF and the optic nerves, as well as between the optic nerves via the chiasm.\(^\text{11}\) These communications may be exaggerated in a chronic state of elevated ICP and are represented in the transmission of pressure from the cranial vessel to the optic nerve tubes in the steady-state unfenestrated model described above (Fig 2). There is virtually no organized fluid flow in this situation and the pressure is uniformly transmitted throughout the system.

When one nerve sheath is fenestrated (Fig 3), pressure within the cranial vessel induces CSF flow into the nerve sheath toward the fenestration. The opening from the cranial vessel into the chiasm represents a reduction in the cross-section area of the fluid column, so the fluid must accelerate, increasing its velocity \( vA = \text{constant} \), where \( A \) indicates cross-section area.\(^\text{11}\)
Bernoulli's equation tells us that the optic nerve surrounded by this more rapidly flowing CSF will be subjected to a substantially reduced pressure than prior to the induction of the flow by fenestration. That is, as velocity increases, pressure must decrease and the optic nerve sees a pressure drop while the ICP may remain high.

The chiasmal fluid communication between the nerves allows the flow of fluid into the fenestrated nerve sheath to cause a reduction in pressure transmission down the unfenestrated sheath. This is demonstrated by the turbulent dye flow at the chiasmal junction of the unfenestrated nerve and by a pressure reduction of 40 mm...
H₂O noted in that sheath compared with the ICP (Fig 4). Thus, chiasmal fluid flow may allow unilateral fenestration to reduce pressure in the unfenestrated side sufficiently to improve papilledema bilaterally. This bilateral effect will be variable, depending on the development of the chiasmal fluid communication, as has been noted clinically.²³

If the chiasmal fluid communication is not sufficiently developed or if the ICP is elevated to extremely high levels, the unfenestrated nerve may not see a large enough pressure drop to eliminate the papilledema on that side. Fenestration of the second nerve will then allow flow along both nerve sheaths, reducing the pressure around both nerves and helping to ensure bilateral relief of papilledema (Fig 5).

The model also explains that the eyes may be without papilledema or visual symptoms even if the ICP returns to elevated levels, as has been noted in the literature.²⁴ Immediately after fenestration, ICP may decrease to low levels due to acute filtration of CSF (Fig 3). Such a lowering of ICP will result in a relief of systemic symptoms, such as headache and nausea.

The pressure may remain low temporarily, but CSF production will likely increase, allowing ICP to slowly return to elevated levels. As the ICP increases, however, so does the pressure-driven flow of CSF along the nerve sheath and through the fenestration. This keeps the pressure around the nerves low and prevents a recurrence of papilledema (Fig 4).

While filtration through the fenestration may induce fairly rapid intra-sheath flow, this flow is small relative to the total CSF volume. Therefore, elevated ICP and generalized symptoms may return, but one would presume that as long as the fenestration is open, papilledema should not recur. Further support for this filtration concept is provided by the demonstration of “filtration cysts” around optic nerve sheath fenestration sites noted on magnetic resonance imaging.²⁵

If the fenestrations scar closed, one would expect papilledema and visual symptoms to recur, as ICP is again directly transmitted down the nerve sheaths (Fig 2).

This model is not a perfect representation of the three-compartment CSF-filled subarachnoid space. The fluid volume available to flow is overestimated since brain and nerve tissue filling these spaces are not represented. Further, variable resistances to flow caused by normal trabeculations of tissue in the fluid spaces are unaccounted for. These and other “simplifications” in the model make actual calculations of flow and pressure by Bernoulli’s equation impractical. However, this model does help us understand two important observations seen in patients with increased ICP who are treated with optic nerve sheath fenestration. It has shown that flow along one nerve may allow a reduction of pressure in the unfenestrated nerve, creating a bilateral effect. Further, the model illustrates that the CSF pressure seen by the nerve may be significantly lower than the ICP as long as the fenestration is open and flow along the nerve sheath is present. It appears, therefore, that filtration of CSF through an open fenestration site, rather than isolation of the optic nerve from ICP via scarring, is critical to the ability of optic nerve sheath fenestration to decrease papilledema.

This work was supported in part by That Man May See Inc, San Francisco, Calif.

References


