The Influence of Cerebrospinal Fluid Pressure on the Lamina Cribrosa Tissue Pressure Gradient


Purpose. To measure the tissue pressure gradient through the optic disk and to determine the relationship between intraocular, cerebrospinal fluid, and retrolaminar tissue pressures. The relationship of optic nerve subarachnoid space pressure to intracranial cerebrospinal fluid pressure also was explored.

Methods. Micropipettes coupled to a pressure transducer were passed through pars plana and vitreous to enter the optic disk in the anesthetized dog. Using a micromanipulator, pipettes penetrated the optic disk in steps while pressure measurements were taken. In some animals, pipettes also were passed into the optic nerve subarachnoid space. Lateral ventricle cerebrospinal fluid pressure, intraocular pressure, and arterial blood pressure were measured concurrently, and the effect of raising CSF pressure was explored.

Results. Retrolaminar tissue pressure was largely dependent on the surrounding cerebrospinal fluid pressure, which was on average 8.6 ± 3.5 mm Hg (SD, n = 8) higher, and was independent of intraocular pressure. Most (85% ± 15% [SD, n = 8]) of the pressure drop between intraocular pressure and retrolaminar pressure occurred across the anterior 400 μm of disk tissue. When the intraocular pressure was 21 mm Hg and the cerebrospinal fluid pressure was zero, retrolaminar tissue pressure averaged 7 mm Hg and the translaminar pressure gradient was 3.08 ± 0.29 mm Hg/100 μm tissue (SD, n = 3). Optic nerve subarachnoid space pressure was equivalent to lateral ventricular pressure.

Conclusions. These results show that cerebrospinal fluid pressure largely determines retrolaminar tissue pressure; hence, along with intraocular pressure, it is of major importance in setting the translaminar tissue pressure gradient. Results also demonstrate hydrostatic continuity between the optic nerve subarachnoid space and the lateral ventricle. That the translaminar pressure gradient can vary independently of intraocular pressure may be of importance in understanding the pathophysiology of glaucoma. Invest Ophthalmol Vis Sci. 1995;36:1163-1172.
Within the human optic disk, the retinal ganglion cell axons are grouped into approximately 230 bundles and pass through the scleral foramen to form the optic nerve. This region is known as the lamina cribrosa and is comprised of successive perforated sheets of connective tissue and intervening glial tissue.

The optic nerve leaves the eye, an environment with an elevated hydrostatic pressure, and passes to the brain while continually surrounded by meninges and cerebrospinal fluid (CSF). The IOP in humans averages 16 mm Hg. The optic nerve merges with brain parenchyma at the chiasm, and parenchymal pressures are at or just above the adjacent CSF pressure. It has been presumed that optic nerve axons experience an abrupt pressure change as they leave the eye at the level of the lamina cribrosa. A tissue pressure gradient, if present, will induce mechanical stress in a direction transverse to the pressure gradient (LaPlace’s Law), within the lamina cribrosa collagenous beams.

The determinants of the translaminar pressure gradient are IOP, retrolaminar tissue pressure, and the axial thickness of the lamina cribrosa. Little is known about retrolaminar tissue pressure and the factors that influence it. Because CSF surrounds the optic nerve, its pressure may influence retrolaminar tissue pressure, as postulated by Volkov. Indirect evidence and one concurrent measurement by Zaren and Hedges in the cat suggest that optic nerve subarachnoid space (ONSAS) pressure is equal to intracranial CSF pressure at the same vertical level. Measurements from six human cadavers indicated that ONSAS pressure was proportional to CSF pressure in the lateral ventricle. The proportionality constant varied from approximately 0.5 to 1.0, varying with site (along the optic nerve), eye, and specimen. This confirmed that fluid flow between the two sites occurred, but significant resistance with respect to flow might have been present.

Hydrostatic continuity along the spinal cord subarachnoid space was demonstrated by Kronig and Gauss in 1907 (discussed by Davson) and from the spinal cord to the lateral ventricle in the human by Smyth and Henderson. The pressure difference between any two points in the CSF is caused by the gravitational effect on the fluid height difference. More recently, Magnaes, measuring CSF pressures in humans, found that 68 out of 72 (92%) had zero pressure between the occipital prominence and vertebral body C7 while the subject was sitting. This range had a mean vertical distance of 140 mm, equivalent to 10 mm Hg hydrostatic pressure change. The occipital prominence is at the same level as the eye in humans. Thus, it is likely that in most humans when either standing or sitting, the intracranial CSF pressure is zero, or subatmospheric.

Because of considerable methodologic difficulties, there have been only two attempts to measure retrolaminar tissue pressure and no attempts at measuring the lamina cribrosa pressure gradient. Ernest and Potts performed the first optic nerve tissue pressure measurement in the cat using a 30-gauge needle connected to a pressure transducer, through which fluid was constantly infused (0.3 μl/second) to prevent the tip from blocking. The needle passed through the cornea-lens periphery into the optic nerve head. Intraocular pressure was maintained at 30 to 40 mm Hg through a cannula inserted into the anterior chamber. Average optic nerve tissue tension from seven animals was 14 mm Hg. The pressure gradient and position of the needle in the tissue were not measured. Hedges and Zaren used six cats in whom, by a lateral orbitotomy, a 30-gauge needle was inserted into the retrobulbar portion of the optic nerve 2 to 3 mm from the globe. In one, the optic nerve head was penetrated through the vitreous with a 30-gauge needle. Needles were not constantly perfused, but 50-μl flushes were given if needle tip blockage occurred. Optic nerve tissue pressure measurements ranged from 2 to 8 mm Hg, with a mean of 6 mm Hg. A rise in optic nerve tissue pressure was demonstrated, induced by CSF pressure elevations of 40 mm Hg, in the retrobulbar experiments but not in the transvitreal experiment. Our study was designed to measure the site and size of the lamina cribrosa pressure gradient, the retrolaminar tissue pressure, and its relationship to CSF pressure using highly localized measurements of tissue pressure. The relationship between optic nerve subarachnoid space CSF pressure and intracranial CSF pressure also was explored. Our method used micropipettes coupled to a low-compliance transducer, with the aim of minimizing the fluid fluxes across the tip into the tissue, to reduce tissue trauma. The degree of tissue trauma is thought to affect tissue pressure recordings artefactually, as shown by Wiig and Noddeland who compared micropipette and wick in catheter techniques.

Dogs were chosen as the experimental animal because they have well-developed lamina cribrosa, unlike the analogous rudimentary structures found in most smaller animals, and because they are susceptible to glaucoma.

METHODS

Tissue Pressure Transducer

Micropipettes were pulled from 1.2 mm OD (ID, 0.69 mm) borosilicate capillary glass tubing, producing a gentle final taper. A sharp single bevel was ground producing a 25° to 30° bevel with outer tip diameters between 19 μm and 30 μm. The distance from the tip to an outer diameter of 50 μm was 800 μm. The pipettes were filled with Ringer’s lactate solution. These...
parameters were chosen after pilot experiments on isolated pig eyes from a local abattoir, measuring response times in the vitreous and optic disk surface. Pipettes were fluid-connected to a 1.3 mm-diameter fiberoptic pressure transducer (Camino Labs; San Diego, CA) through a specially constructed perspex holder with two threaded ends, sealed with O rings (Fig. 1). A stainless steel tube side port was joined to the holder so that polythene tubing could be attached and small volume flushing could be performed while the pipette tip was in the vitreous. The three standard pressure transducers (Gould [Oxnard, CA] P23 ID) and the fiberoptic transducer were connected to a polygraph (Grass Instruments; Quincy, MA) and calibrated against a water-filled hydrostatic manometer before each experiment. The absolute pressure measurement error for each transducer (except arterial blood pressure) was ±1.0 mm Hg. The quoted compliance of the fiberoptic transducer is 0.04 pl/mm Hg. The measured compliance of our combined system is 1.0 nl/mm Hg.

Animals
Twenty-one mongrel dogs of varying breed mixture were used in this study, the housing and use of which conformed to the ARVO Statement for the Use of Animals in Ophthalmic and Vision Research. Nine were used to develop the experimental techniques. In three, there were no valid results because of problems described below. Valid results were produced in nine animal experiments.

Experimental Protocol
The dogs were suspended in a prone position, with front legs hanging down through a fenestrated sheet and the rest of the body supported in a sling. The head was fixed using a maxilla bite clamp connected to two magnetic stands.

The CSF pressure in the left lateral ventricle was monitored. A burr hole was drilled 1 cm off the sagittal midline halfway between the occipital prominence and a line between both lateral canthi. The left lateral ventricle was cannulated with an 18-gauge needle directed vertically through the burr hole, sealed and held in position with a skull screw (Camino Labs). The 18-gauge needle was connected to a pressure transducer. A lateral canthotomy was performed to improve exposure. An eye ring was sutured to the perilimbal conjunctiva and connected to another magnetic stand to stabilize the eye.

Intraocular pressure was measured and controlled with two 25-gauge cannulas inserted through the peripheral cornea into the anterior chamber. One was connected by polythene tubing to a pressure transducer, the other to a suspended buret of Ringer’s solution to act as a hydrostatic pressure head. Thus, IOP was separately variable and recordable. The IOP was set at 15 to 25 mm Hg above CSF pressure. The pupil was dilated with 2% homatropine and 10% phenylephrine drops. A plano-concave corneal contact lens was applied with methylcellulose, and the retina and optic disk were observed with an operating microscope (D. F. Vasconcellos, Sao Paulo, Brazil). Temporal sclera was exposed, and 4 mm posterior to the limbus a sclerostomy passing through the pars plana was formed.

Tissue Pressure Measurements
After the removal of bubbles, the pipette–transducer assembly (Fig. 1) was placed in an arc micromanipulator. To avoid suction of air from capillary action, the side arm was not plugged until the pipette entered the vitreous.

Once the pipette was in the vitreous, the side arm plug was inserted, effectively flushing approximately
0.2 µl fluid through the pipette. All patent pipettes produced a pressure recording equal to that recorded concurrently from the anterior chamber, i.e., equal to IOP.

Pipettes were advanced and seen to indent the central optic disk surface, at which time there was often a transient rise in recorded pressure that settled to equal IOP. If the pressure remained elevated, a blockage was deemed to have occurred and the pipette was reversed into the vitreous and flushed by advancing the side arm plug. Further testing for the presence of tissue blockage was performed. At 100-µm depth into the disk tissue, IOP was reduced 5 to 10 mm Hg by lowering the hydrostatic pressure head. Blockage was deemed not to have occurred if a similar optic nerve tissue pressure change was recorded. The tissue response times at this step were used to calculate the response times, which averaged 6 minutes (see below).

When guaranteed unblocked, the pipette was then advanced into the optic nerve tissue in 100-µm steps (Fig. 2), to between 600- and 1000-µm tissue depth, before being withdrawn into the vitreous. In earlier experiments, we limited the penetration depth to 600 µm because of concerns about causing excessive tissue trauma that would affect subsequent penetration measurements. The pressure measurement after withdrawal into the vitreous was used as the zero depth measurement.

At each penetration depth, the pipette was kept in position until the measurement was stable. During every penetration, the pipette was maintained in one position for more than 15 minutes to see if the measurement was falling, which would have indicated a leak. Only those results not affected by pipette blockage or system leaks are presented here.

Four to five penetrations were attempted for each eye. On average, only one or two penetrations produced valid results for each dog. Note that the angle of entry into the optic nerve was oblique, averaging 65° (reducing the depth normal to the disk surface by 10%) (Figs. 2, 3).

Optic Nerve Subarachnoid Space Pressure

During the last penetration in each eye of six dogs, the pipette was advanced (Fig. 2, dotted line) until a sudden rapid reduction in pressure was recorded, between depths of 2.5 to 4.5 mm (varying with disk entry site). The response time in all cases was less than 30 seconds, compared with 6 minutes in optic nerve tissue. This is the time course of a fluid rather than a tissue pressure response, indicating that a fluid-filled space had been entered. Blood was not seen in the pipette after removal, indicating that a blood vessel had not been entered. In one case, the pipette tip broke in the tissue, and later dissection of the eye showed the tip to be in the ONSAS. In some cases, pial hemorrhages also were seen on histologic examination, indicating that the pipette tip had passed through pia mater.

Pressure measurements were taken while the pipette tip was in this cavity in six dogs, in four of which the CSF pressure (CSFp) was manipulated. In two dogs, CSFp was altered by inflation of a blood pressure cuff around the neck. In one, CSFp was altered by the injection of fluid into the lateral ventricle, and, in the other, the CSFp fluctuated with arterial blood pressure changes.

After completion of the experiment, the dogs were killed by lethal intravenous injection of pentobarbital, and the eyes were enucleated and immersion fixated in 10% formalin. The optic nerves were dissected and embedded in paraffin wax. Longitudinal...
sections from the center of the optic disk were taken and stained with hematoxylin and eosin.

RESULTS

Figure 3 shows one optic nerve section in which the pipette penetrated to at least 600 \( \mu \text{m} \) depth. Relative to previously used methods, little tissue trauma was seen. Measurements from tissue fixed and stained after tissue pressure recordings revealed a scleral canal diameter of \( 1.45 \pm 0.18 \text{ mm} \) (mean \( \pm \text{SD} \), \( n = 5 \)) and a central lamina cribrosa axial width of \( 435 \pm 102 \text{ \( \mu \text{m} \)} \) (mean \( \pm \text{SD} \), \( n = 5 \)). The lamina cribrosa was situated across the central disk tissue at a depth from 100 to 600 \( \mu \text{m} \). The lamina cribrosa could be seen, comprising neural tissue and the transversely oriented, interrupted connective tissue bridging the scleral foramen. The lamina cribrosa axial thickness in this dog was 600 \( \mu \text{m} \).

Figure 4 shows the raw data from one optic nerve penetration in which the optic disk was penetrated in 100-\( \mu \text{m} \) steps to a depth of 1000 \( \mu \text{m} \) over 72 minutes. Tissue pressure, IOP, femoral artery blood pressure, and CSF pressure are plotted as a function of time. Response times were calculated from the time taken for 90\% of the tissue pressure drop to a new stable level, when IOP was reduced and the pipette was at a depth of 100 \( \mu \text{m} \). The mean 90\% response time was \( 5.9 \pm 0.76 \text{ minutes} \) (SD, \( n = 11 \)).

We used the micrometer depth readings directly and did not convert them to depth values normal to the plane of the disk surface for the following reasons: The pipettes, because of their tip size, tend to indent the inner limiting membrane for a variable distance before penetration. Once penetration occurs, the disk surface is seen to move up to and around the tip of the pipette. The maximum error in pipette placement is estimated to be 100 \( \mu \text{m} \) further than recorded depth. Also, the optic nerve in the scleral canal is slightly angulated toward the axis of pipette penetration. Thus, the use of an angulation correction factor would not add to the tissue depth accuracy.

Figure 5 shows the tissue pressure profiles from the first valid penetration in the left eye of eight dogs with varying IOPs. Retrolaminar tissue pressure was defined as the minimum pressure recorded between depths of 400 to 800 \( \mu \text{m} \). These data show a wide spread of retro-
laminar tissue pressures (RLTp) recorded with a mean of 14.3 ± 8.2 mm Hg (SD, n = 8).

Most of the pressure drop was demonstrated to occur over the first 400 μm of tissue depth. With each set of results from an animal, the mean percentage of the pressure drop from IOP to RLTp occurring over the first 400 μm tissue depth was calculated. The mean of the percent pressure drop over 400 μm for all experiments was 85% ± 15% (SD, n = 8).

To assess the gradient in the most physiological situation and to eliminate the confounding effect of different IOPs, a subset of the data was selected from three animals in which IOP was 21 mm Hg and CSF pressure was 0 to 2 mm Hg (Fig. 6). Retrolaminar tissue pressure averaged 7 mm Hg. The maximal gradient occurred at depths between 0 and 400 μm, averaging 3.08 ± 0.29 mm Hg per 100 μm tissue depth (SD, n = 3).

There was a strong linear relationship between retrolaminar tissue pressure and lateral ventricle CSF pressure (Fig. 7). These data are from the first penetration results in nine animals. Least mean square regression analysis (Microsoft Excel, Redmond, WA) was performed on these data points, giving a correlation coefficient of 0.93 (P < 0.01), y-intercept of 6.8, and gradient of 1.3. The gradient was not significantly different from 1.0 (t statistic, 1.5; df, 8). The y-intercept was significantly different from zero (t statistic, 4.3; df, 8; P < 0.003). Thus, CSF pressure is a major factor determining the retrolaminar tissue pressure and has high predictive value in determining RLTp in different dogs. Our data suggest the relationship RLTp = 1.3 × CSF + 6.8. Additionally, when CSFp was subtracted from RLTp, the mean of the resultant normalized retrolaminar tissue pressures was 8.6 ± 3.5 mm Hg (SD, n = 8), demonstrating a reduction in variance compared with the raw RLTp data (14.3 ± 8.2 mm Hg [SD, n = 8]).

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Data are from experiments in which more than one optic disk penetration was achieved. The "v" suffix indicates presumed vein penetration in optic nerve tissue.
Optic Nerve Tissue Pressure Gradients

To investigate the dependence of retrolaminar tissue pressure on IOP and CSFp within individual animals, data from animals in which valid multiple penetrations occurred were compared (Table 1). The "v" suffix indicates a presumed vein penetration (see below). The difference between RLTp and CSFp was consistent, with low variance within the same animal (mean variance, 0.5). As a comparison, the difference between IOP and RLTp showed much greater variation (mean variance, 24.5). Thus, within the same animal, CSF pressure changes were associated with equivalent changes in RLTp.

The influence of IOP on RLTp was assessed. Because IOP was set in relation to CSFp and because RLTp was related to CSFp, CSFp was subtracted from RLTp to assess a relationship independent of CSFp (Fig. 8). No significant relationship was found between IOP and the difference between RLTp and CSF pressure (correlation coefficient = 0.45).

Optic Nerve Vein Penetrations

In three animals, optic nerve vein penetration seemed to occur. In these animals, the recording showed a rapid fall in pressure at a depth between 300 and 800 μm to levels approximately 7 mm Hg above CSF pressure. The response times were less than 30 seconds, indicating that a fluid-filled cavity had been entered, and blood was seen in the pipettes after removal, indicating that an optic nerve blood vessel, presumably a vein, had been entered (Table 2). Figure 9 shows a presumed vein penetration, followed by ON-SAS penetration. In two dogs, other valid penetrations were produced (Table 1). These presumed venous pressure recordings had the same relationship to CSF pressure as did retrolaminar tissue pressure.

Optic Nerve Sheath and Lateral Ventricle Cerebrospinal Fluid Pressure

Raw data in Figure 9 demonstrate the rapid fall in pressure to 7 mm Hg (presumed vein entry, see below) and further penetration to 2 mm Hg in the ONSAS. Raw data in Figure 10 demonstrate the rapid fall in pressure on entering the ONSAS and subsequent similar alterations in ONSAS pressure (ONSASp) and lateral ventricular pressure produced by neck blood pressure cuff inflation. Combined data from six animals in which pressure measurements were recorded from the left lateral ventricle (LLVp) and optic nerve subarachnoid space (ONSASp) simultaneously are presented in Figure 11.

The results show a significant linear relationship between optic nerve subarachnoid space pressure and lateral ventricle subarachnoid space pressure. Least mean squares regression of these data points shows a y-intercept of -0.52 and a gradient of 0.96, with a significant correlation coefficient of 0.999 (P < 0.001). The hypotheses that the y-intercept was not significantly different from 0 (t-statistic, -1.47; df, 24) and the gradient was not significantly different from 1.0 (t-statistic, 1.6; df, 24) were tested with the Student's t-test and were upheld. Thus, optic nerve subarachnoid space pressure can be said to be equivalent to lateral ventricle pressure within the experimental error of the method used.

DISCUSSION

Judging from postexperimental histology (Fig. 3), less trauma was induced by our micropipette method than with the only other optic nerve tissue pressure method described in which histology was shown (Ernest and Potts). Ours are probably the most accurate and artifact-free measurements to date; however, our method is prone to leaks and blockages and has a slow response time, partially because of the use of a passive transducer.

The dog's globe and optic nerve dimensions are comparable to those of the human. The mean scleral canal diameter measured from these dogs was 1.45 mm versus 1.48 mm reported in the human. The subarachnoid space surrounds the optic nerve ending at the posterior limit of the sclera and lamina cribrosa.

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TABLE 2. Presumed Optic Nerve Vein Penetrations at Which Penetration Occurred

FIGURE 8. Relationship between corrected retrolaminar tissue pressure (RLTp - CSFp) and intraocular pressure in nine animals.
FIGURE 9. Tissue pressure (mm Hg) recording at a depth from 700 µm to 1500 µm. Pressure (mm Hg) ordinate versus time (minutes) abscissa. At 800 µm, the pressure fell to 7 mm Hg (open triangle), suggesting a vein was penetrated. At 1500 µm, a further decrease in pressure to 2 mm Hg occurred, suggesting cerebrospinal fluid penetration.

in dogs and humans. Dogs do not have a central retinal artery or vein. Branches from the posterior ciliary arteries and veins pass into the optic nerve tissue at the level of the sclera and turn anteriorly to emerge on the surface of the disk, forming usually incomplete vascular arcades around the small central cup. Retinal arterioles and venules emerge from these arcades. This vascular arrangement permits pipette penetration with a reduced risk of inducing hemorrhage. Dogs have a well-developed lamina cribrosa, with 10 to 15 sheets of collagenous connective tissue bridging the scleral canal. Average lamina pore size in the beagle ranges from 12 to 50 µm, similar to that in humans (10 to 100 µm). The lamina cribrosa axial thickness averaged 500 µm in the dogs used, compared with a mean of 237 µm in humans reported by Quigley et al; however, the connective tissue beams appear less dense (personal observations, 1993) in dog eyes than in human eyes. The dog optic nerve is myelinated and contains fewer axons than the human optic nerve (150,000 versus 1,010,000).

Our pressure measurements demonstrate the dominant role that CSF pressure plays in determining retrolaminar optic nerve tissue pressure. Within the same animal under different IOP and CSF pressures, the retrolaminar tissue pressure was found to be at a constant level (mean, 8 mm Hg) above the CSF pressure in the surrounding subarachnoid space. Intraocular pressure is shown largely to determine anterior optic disk tissue pressure (Fig. 5) over the most anterior 100 µm of tissue.

This series of results also includes measurements of the pressure gradient across the optic disk. Although our optic disk pressure profiles between animals are difficult to compare because of variable IOP and CSF pressures, we have shown that 85% of the pressure drop from intraocular to retrolaminar tissue pressure occurs across the first 400 µm, i.e., over the anterior bulk (67% to 100%) of the lamina cribrosa. The maximal pressure gradient was 5.08 mm Hg per 100 µm tissue in this region in those animals with IOP of 21 mm Hg and CSF pressure from 0 to 2 mm Hg. Further posterior, relatively little pressure change occurs. Hence, it can be stated that the lamina cribrosa occupies the region across which most of the pressure gradient falls.

Our results demonstrate that CSF pressure in the canine optic nerve subarachnoid space and in the lateral ventricle at the level of the eye are identical, demonstrating hydrostatic continuity of the CSF along the canine optic nerve sheath.

Our experiments could not determine where the pressure drop between the retrolaminar tissue and the CSF occurs nor what factors are involved in maintaining this difference. This pressure difference may be due to inherent constrictive qualities of the pia mater.

FIGURE 10. Pressure recording from (a) lateral ventricle cerebrospinal fluid, (b) optic nerve tissue and ONSAS, (c) intraocular pressure, (d) arterial blood pressure. Pressure (mm Hg) ordinate versus time (minutes) abscissa. Tissue pressure recording (b) shows a sudden fall to zero at 2.5 mm depth (open triangle), indicating entry into the ONSAS. Large arrows indicate neck blood pressure cuff inflation (up) and deflation (down) of 25 mm Hg. Triangles indicate 5 mm Hg inflation. Note the equivalent pressures in the lateral ventricle and ONSAS. ONSAS = optic nerve subarachnoid space.
Optic nerve tissue pressure gradients

![Graph showing lateral ventricle pressure vs. ONSAS pressure](image)

**FIGURE 1.** Pressure from lateral ventricle and optic nerve subarachnoid space recorded simultaneously in six animals. Lines connect data from the same animal. Pressure is recorded in mm Hg.

Reported intraluminal vein pressures have been shown to be several millimeters of mercury above tissue pressures in the rabbit hind leg and human forearm and have been presumed to be equivalent to surrounding tissue pressure in the eye. The only direct measurements from ocular veins (cat retinal vein) suggest that the pressure is 7 to 10 mm Hg above IOP. Our presumed optic nerve venous pressures within the deep laminar and retrolaminar regions had the same relationship to CSF pressure as tissue pressure and likely were close to the surrounding tissue pressure.

If these experimental results in animals are extrapolated to humans, the following features are noted. The pressure gradient between the intraocular space and the retrolaminar tissue may occur across the lamina cribrosa in humans. In humans, if the retrolaminar pressure is greater than and largely determined by CSF pressure, it could have potential pathophysiological importance if lamina cribrosa beam tension or other hydrostatic pressure effects are important in the etiology of glaucoma. Small changes in IOP could have a proportionally greater effect upon the translaminar pressure gradient if the retrolaminar pressure is greater than atmospheric (zero) pressure. A translaminar pressure difference of 8 mm Hg when retrolaminar pressure is 7 mm Hg and IOP is 15 mm Hg will be doubled when IOP increases to 23 mm Hg. Individuals with low CSF pressure might have low retrolaminar tissue pressure and, hence, an increased translaminar pressure gradient. Conversely, those with higher CSF pressure might have a lower gradient. These factors could influence the development of normal tension glaucoma or the susceptibility of the optic nerve in ocular hypertension.

**Key Words**

dog, optic nerve, tissue pressure, cerebrospinal fluid pressure, glaucoma

**Acknowledgments**

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