No. 12

THE CAMBRIDGE GLAUCOMA LETTER

published by THE CAMBRIDGE GLAUCOMA FOUNDATION, INC.

OUTFLOW

II

The Optic Disc

The blood circulates through the eye in flaccid vessels as through a pressurized sphere. Where the intravascular pressure is less than the intraocular pressure, the blood vessels are collapsed. Where the intravascular pressure is greater than the intraocular pressure, the blood vessels are distended. Stresses within the vessel walls are proportionate to the difference between the extravascular and intravascular pressures. These stresses, depending on their direction, may collapse a vessel or dilate it. They may also, less obviously, lengthen the vessel and under some circumstances bring about its concomitant constriction.

One often observes an increase in the ratio of the width of the retinal veins to that of the arteries. Such apparent widening of the veins may be a consequence of engorgement and distention, as when outflow is obstructed by venous thrombosis. Apparent widening of the veins may, however, also result from arterial insufficiency, when inadequate filling of the veins results in their partial collapse. The recognition that with impairment of the circulation the veins in the retina may collapse also provides an explanation for arteriovenous nicking. At the a-v crossing, the rigidity of the overlying artery prevents tangentially oriented collapse of the vein. Instead there may be radially oriented collapse as the vein passes under the artery, presenting the profile of the lesser diameter of the elliptical cross section of the vein to the observer and giving the appearance of a localized constriction.

The walls of the retinal vessels, be they arteries, capillaries, or veins, are subjected to stresses proportional to the difference between the intravascular and intraocular pressures. Intuitively it is immediately apparent that if the intravascular pressure is greater than the extravascular pressure, the diameter of the vessel will tend to expand. What is not so readily apparent is that increased intravascular pressure may bring about not only dilation but also elongation of the vessel, which, if its ends are tethered, will become tortuous. Furthermore, if the elastic modulus, i.e. the resistance to circumferential expansion is disproportionately high, then the stretching of the vessel may actually produce circumferential contraction of its walls and narrowing of its lumen.

We distinguish deformations that are promptly reversible upon removal of the inducing stress from deformations that persist long after the stress has been removed. For example, when digital pressure on the globe causes the collapse of a segment of vein on the optic disc, the deformation is reversible, because as soon as the finger is removed, the intraocular pressure falls to its prior value; the collapsed vein again fills with blood and recovers its prior shape. Here we need only look to the balance of forces exerted by the intravascular and extravascular pressures for an explanation of the phenomenon that we see. On the other hand, the tortuosity of an artery or vein that has been subjected to increased intraluminal pressure is reversed very slowly if at all when the intravascular pressure is reduced or the extravascular pressure increased. In this instance we may not attribute the morphologic changes that we see solely to the elastic characteristics of the tissue. Nonetheless it is axiomatic that the pressure induced stress does produce some deformation, however small, and we may ask whether such small deformation, if it persists day after day and week after week, might not suffice to determine a pattern of tissue growth that ultimately produces the observed configuration. We do not know what effects protracted mechanical stress will have on the structural development of tissue. For the time being, we note the similarity between the deformation expected if the tissue were elastic, and the observed non-elastic change, whatever its nature may be.

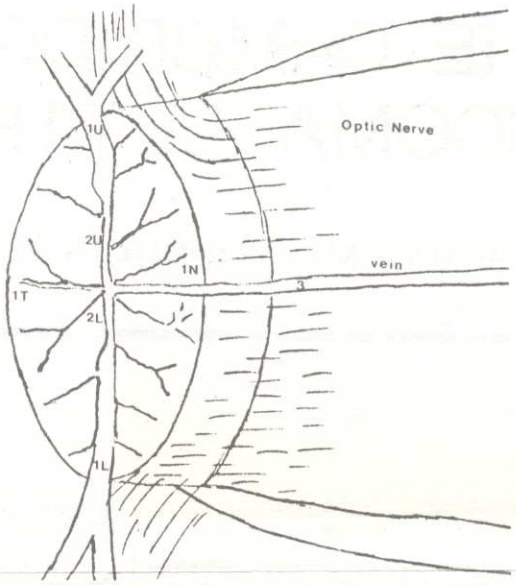

FIGURE 1 - Schematic representation of retinal vein leaving the eye at the optic disc. At point 1U and 1L, the vein is distended because intravenous pressure exceeds intraocular pressure. The external (intraocular) pressure does not express the contents of the vein at these points because of the downstream resistance at points 2U and 2L. The intraocular pressure does express the contents of the vein at point 2U and 2L, because the pressure within the optic nerve (point 3) is very low. As the intraocular pressure expresses the contents of the vein, its cross-section becomes smaller and the resistance to flow becomes greater. From the circumstance that the length of segment 2U is greater than 2L, one infers that either the flow through 2U is less or that it is less tightly compressed than 2L, giving it a lower resistance per unit length, since the pressure gradient across 2U must be the same as the pressure gradient across 2L. If intraocular pressure increases, resistance to flow increases because the vein becomes more tightly compressed or because the collapsed segment becomes longer, or both. If intraocular pressure decreases, the converse is the case. Thus the collapse of the vein creates in effect a self-adjusting pressure reducing valve.

On the surface of the disc we observe the dynamics of venous outflow from the retina. We cannot, to be sure, measure the intravenous pressure directly, but we are in a position to make reliable inferences about it. In the first place, we know that since the venous wall is flaccid, the intravenous pressure of the distended vein within the globe must be no less than the intraocular pressure of which we can make reliable measurements. In the second place, we know that in the cavernous sinus at the base of the brain the venous pressure is negligible, and may, when the patient is upright, even be negative. The venous pressure in the optic nerve immediately posterior to the lamina cribrosa is likely to be almost as low. On the optic disc, therefore, there is within a fraction of a millimeter a precipitous drop in pressure, from the 12 or 15 or 20 or 25 mm. Hg. or whatever the intraocular pressure happens to be, to a very low value, perhaps five or three mm Hg. The drop in pressure is associated with correspondingly large resistance to flow. Resistance is defined as the quotient of pressure difference divided by flow. Given a constant volume of flow per unit time, the large pressure difference indicates a proportionately large resistance.

The mechanism by which flow resistance on the disc is increased can be readily seen. With the ophthalmoscope, one can observe that on the optic disc the increase in resistance to flow is brought about by the pressure induced collapse of the retinal veins. The vein collapses, of course, from a disparity between intravascular and extravascular, i.e. intraocular pressures. The intravascular pressure is low because of a lack of down-stream resistance. There is literally nothing, no resistance, no friction, no back pressure, to prevent the blood in the retinal vein from draining freely into the veins behind the globe. That is why on the disc there collapses a segment of vein sufficient in length to establish a channel of high resistance to flow which will in effect maintain the intravascular pressures within the eye at above intraocular pressure. It is this resistance which holds back, as it were, the contents of the venous bed in the retina, and prevents its being expressed by the intraocular pressure. Since the volume flow per unit time remains the same, the velocity of flow will increase as the cross section of the vein becomes smaller.

It is instructive now to observe, how this collapsed segment of vein which serves as a reducing valve, responds to changes in the various forces to which it is subject. By lightly placing a finger over the lateral rectus muscle and making pressure on the sclera, one may increase the intraocular pressure, and simultaneously looking through the ophthalmoscope one notes that the segment of collapsed vein becomes longer, up to a point, beyond which additional pressure will cause no further extension of the collapse of the vessel. In other eyes even the initial increase of intraocular pressure brings about no change in the length of the compressed vein. In all eyes, as intraocular pressure increases, so does the resistance to venous drainage. This increase in venous outflow resistance comes about by two mechanisms which act independently or in combination: 1. As the flat-

tened vein is yet further compressed, its cross-section may become even smaller. 2. Segments of hitherto distended vein may collapse, thereby increasing the overall extent of flattened vein.

An analogous phenomenon may be observed in those eyes which have spontaneous pulsation of veins on the disc. There the fluctuation in the length of the collapsed segment is caused by pulsations in the intraocular pressure. It is the arterial pulse of the choroid which propagates through the aqueous and vitreous, contained within the relatively inelastic sclera, inducing small periodic fluctuation of intraocular pressure. This in turn changes venous dynamics in just the manner we have described and produces spontaneous pulsation of the retinal veins.

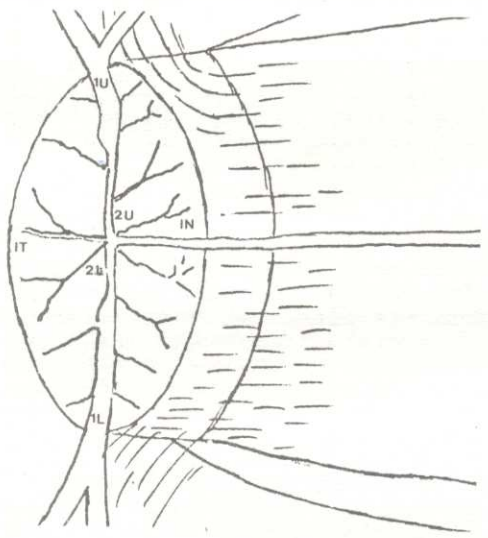

FIGURE 2 - Venous drainage on the glaucomatous optic disc. The upper and lower poles of the disc drain into more peripheral segments of retinal vein at 1U and 1L. These peripheral segments are distended with blood whose pressure is at or above the intraocular pressure. As the intraocular pressure rises, so does the pressure at 1U and 1L. On the other hand, venous drainage of the temporal and nasal poles, 1T and 1N, is into segments of retinal vein (2U and 2L), which are already collapsed by intraocular pressure. The venous pressure at 2U and 2L is substantially lower than intraocular pressure. Perhaps even more important, the venous collapse at 2U and 2L insulates this segment of vein and its tributaries from increases in intraocular pressure. The estimates of venous pressure assume an intraocular tension of 30 mm Hg. The highest venous pressures are expected in those areas of the disc that are must susceptible to glaucomatous damage. In the areas most resistant to glaucomatous damage, venous pressure remains low inspite of elevation of intraocular tension.

Consider also the effects on the length of collapsed vein of variations of retinal arterial flow. When flow through the vein increases, there is some small diminution in the length of the collapsed vein at its distal end. Conversely, as is often the case, when, as from arteriosclerotic obstruction of the feeding vessels, there is a diminution of flow, the length of collapsed vein increased. This relationship becomes obvious from purely algebraic considerations, inasmuch as the pressure gradient is the product of flow times resistance. If the pressure gradient is to remain the same, then when the flow per minute decreases, the resistance must increase. The resistance will increase either as a result of more complete or of more extensive collapse. Whether the change in resistance comes about as the result of more complete or more extensive collapse will be a function of the elastic properties of the vein. The less pliable, the more sclerotic the vein, the longer will be the segment required to bring about the requisite pressure gradient for any given amount of flow. Similarly, the lower the minute volume of flow, the longer will be the segment required to account for any given pressure gradient. We observe that when the flow diminishes beyond a point that is critical for any given value of wall stiffness, the reduction in pressure will no longer remain localized. The pressure will then be reduced in the entire venous tree. All the large veins of the fundus will then flatten, and their visible profiles will broaden as described in the preceding issue of the Glaucoma Letter.

These observations in physiology, in addition to their explanatory value for a-v nicking and the increased v/a ratio commonly seen in obstructive retinal vascular disease of all kinds, will also shed some light on the possible etiology of glaucomatous disc damage. Observation with the ophthalmoscope, readily confirmed with fluorescein angiography, reveals that the temporal and nasal poles of the disc, those areas in other words subserving the macula and the temporal field which are most resistant to glaucomatous damage, have a venous drainage through small horizontally coursing veins which enter the central retinal vein near the center of the disc in the proximity of the lamina cribrosa. The central vein in this area is almost always collapsed and the pressure within it is substantially lower than the intraocular pressure. Equally important is the circumstance that when the intraocular pressure rises, the venous pressure in this central segment of vein is unaffected. It is different with the venous drainage from the upper and lower poles of the optic disc. There drainage feeds into the superior and inferior retinal veins at or near the disc margin, where the retinal veins are distended with blood at or above the intraocular pressure. Therefore venous drainage from the

The Cambridge Glaucoma Letter
2 Sacramento Street
Cambridge, Mass. 02138

BULK RATE
U.S. POSTAGE
PAID
BELMONT, MASS.
PERMIT NO. 58290

"RQD 12-1-80"
Dr. and Mrs. Heinz Meyer
Route #2
Damascus, Va. 24236

upper and lower poles of the disc which show the greatest sensitivity to glaucomatous damage, occurs into a segment of vein at or slightly above the intraocular pressure. Occasionally, when intraocular pressure rises, the distended segment of vein into which drainage takes place collapses with a resulting fall in intravenous pressure. More commonly such collapse does not occur, and the venous pressure against which the upper and lower poles must drain, at minimum equals the intraocular tension. At least as important, when the intraocular pressure rises, unless, as is usually not the case, the area of collapse extends to this region, the pressure against which the capillary drainage from the upper and lower poles of the disc must contend, increases with the intraocular pressure.

Consider then what might happen to the venous circulation on the disc as the intraocular pressure is increased. The venous drainage from the temporal and nasal portions of the disc remains unaffected, inasmuch as it flows into a segment of retinal vein which is collapsed, and therefore under lower pressure, and insulated from any increase in intraocular tension. The venous drainage, on the other hand, from the upper and lower poles of the disc flows into branches of the retinal vein which are not usually collapsed either at normal or at elevated intraocular pressures. This fact means that the venous pressure in these segments is equal to or slightly higher than the prevailing intraocular pressure and, more importantly, that as intraocular pressure rises, the pressure in this segment of vein rises with it. At the upper and lower poles of the disc, as distinct from the temporal and nasal poles, the veins act like funnels which serve to conduct increases of intraocular pressure into the disc substance. It may not be coincidence that those areas of the disc most susceptible to atrophy and excavation from elevated intraocular pressure are the same areas in which elevated intraocular pressure induces increased venous pressure. It may also not be coincidence that the small splinter hemorrhages which are sometimes seen on the disc margin in open angle glaucoma are not unlike hemorrhages seen with venous obstruction elsewhere in the fundus.

One may make these observations without presuming to have discovered the "cause" of glaucomatous disc damage. The term "cause" is ambiguous, a trap which it is better to avoid. It is likely that several factors contribute to the glaucomatous destruction of the optic disc, and impairment of venous drainage may be one of them.

Ernst J. Meyer M.D.

* * * * *

The Cambridge Glaucoma Letter is published at unpredictable intervals by the Cambridge Glaucoma Foundation, Inc. If you wish to receive the Glaucoma Letter, it is free of charge, and if your address label does not yet bear a request date, such as "RQD 12-1-80", please write to us, or complete the coupon below and mail it to

The Cambridge Glaucoma Letter
2 Sacramento Street
Cambridge, Mass. 02138

(Telephone: 617-868-9307)

I would like to continue to receive the Cambridge Glaucoma Letter.

_____
name

_____
street

_____
city, state, ZIP