Summary

Glaucomatous optic neuropathy (GON) is characterized by progressive loss of retinal ganglion cells, including their axons, and by tissue remodeling of the optic nerve head. This is followed by visual field defects. Traditionally, diagnosis and treatment has been directed towards lowering the intra ocular pressure (IOP), which has been considered the most important risk factor. However, not all patients with glaucomatous damage have an elevated IOP. Thus, progression of GON may occur even at IOP in the low teens. These facts challenge the pathophysiological concept of glaucoma based only on IOP. Hence, disturbed ocular blood flow has been regarded as a potential risk factor of great interest.

In the present study, the hemodynamic changes in glaucoma patients (high and normal tension glaucoma) and in patients with ocular hypertension (OHT) has been illustrated. Also the angiopathic effect of certain glaucoma risk factors on ocular perfusion has been assissed to detect its role in pathogenesis of GON.

Among the many risk factors described, systemic hypertension (SHT), diabetes mellitus (DM) and myopia deserved special interest to be studied for the following reasons:

- They are often associated with glaucoma.
- Their role in glaucomatous damage or progression of the disease is debatable.
- Control of hypertension and DM may influence progression of the disease.

Furthermore, the authers assessed the relations between the measured hemodynamic parameters and certain clinical and investigational findings of glaucoma patients to define the predictive role of these circulatory parameters in diagnosing the glaucoma or assessment of its progression.

According to evidence-based medicine in 1993, the effectiveness of IOP reduction in glaucoma treatment had not yet been established. Today, the role of vascular factors in the management of glaucoma is in the same position as IOP was just over a decade ago.

The only vascular factor consistently meeting the criteria required for clinical consideration is diastolic perfusion pressure. There is currently no evidence supporting the role of ischemia in the clinical management of the disease, in spite of numerous small clinical findings supporting the role of vascular deficits and ischemia in glaucoma. Existing clinical research supports the

funding of a large-scale prospective ocular hemodynamic study in glaucoma.

Technology for the comprehensive assessment of vascular hemodynamics exists in the clinical research environment. The technology for metabolic measurements of retinal oxygen consumption is under development and will be applied in clinical research in the near future. Using these techniques, the literature contains numerous examples of altered blood flow in ophthalmic disease.

Current technologies cannot be used to determine whether altered blood flow is primary or secondary to IOP and/or optic nerve damage.

Longitudinal studies may provide better insight into the role of ocular blood flow deficits in disease progression, but at a great cost in time and materials.

As technology improves, more direct measurements may provide the answers to some basic questions:

_ How great a blood flow deficit is necessary to cause glaucomatous damage?

_ What is the threshold of perfusion deficit before nerve cell damage occurs?

_ How can they identify this threshold?

Measurement of metabolism may provide the answer. Several types of measurement would be of interest. Desired measurements include retinal oximetry, quantification of metabolites, reduction/oxidation potentials, or perhaps ATP/NADH_levels.

Using spectral analysis of reflected light, work is underway in the development of retinal oximetry, but a system suitable for clinical use is still years away. These measurements will allow us to fine-tune glaucoma medications as we gain understanding of how various drugs effect the environment in which neurons must survive.