The orbital cerebrospinal fluid pressure (CSFP) represents the true counter-pressure against the intraocular pressure (IOP) across the lamina cribrosa and is, therefore, one of the two determinants of the trans-lamina cribrosa pressure difference (TLPD). From this anatomic point of view, an elevated TLPD could be due to elevated IOP or abnormally low orbital CSFP. Both experimental and clinical studies have suggested that a low CSFP could be associated with glaucomatous optic neuropathy in normal-pressure glaucoma. These included monkey studies with an experimental long-term reduction in CSFP, and clinical retrospective and prospective studies on patients with normal-pressure glaucoma.

Since the choroidal blood drains via the vortex veins through the superior ophthalmic vein into the intracranial cavernous sinus, anatomy suggests that the CSFP could influence choroidal thickness. A population-based study revealed that thicker subfoveal choroidal thickness was associated with higher CSFP. Since the central retinal vein passes through the orbital CSF space, anatomy suggests that the retinal venous pressure should be at least as high as the orbital CSFP. Other experimental, clinical or population-based studies suggested an association between higher CSFP and higher retinal venous pressure and wider retinal veins. Consequently, a higher estimated CSFP was associated with arterial hypertensive retinopathy (with respect to the dilated retinal vein diameter and higher arterial-to-venous diameter) and with the prevalence, severity and incidence of diabetic retinopathy.

Physiologically, CSFP was related with higher IOP. The influence of the CSFP on the episcleral venous pressure and/or a regulation of both CSFP and IOP by a center in the dorsomedial/perifornical hypothalamus may be responsible for this. In summary, the CSFP may be an overlooked parameter in ocular physiology and pathology. Abnormal changes in the CSFP, in particular in relationship to the IOP, may have pathophysiologic importance.