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## Pathogenesis of optic disc edema in raised intracranial pressure



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### ABSTRACT

Optic disc edema in raised intracranial pressure was first described in 1853. Ever since, there has been a plethora of controversial hypotheses to explain its pathogenesis. I have explored the subject comprehensively by doing basic, experimental and clinical studies. My objective was to investigate the fundamentals of the subject, to test the validity of the previous theories, and finally, based on all these studies, to find a logical explanation for the pathogenesis. My studies included the following issues pertinent to the pathogenesis of optic disc edema in raised intracranial pressure: the anatomy and blood supply of the optic nerve, the roles of the sheath of the optic nerve, of the centripetal flow of fluids along the optic nerve, of compression of the central retinal vein, and of acute intracranial hypertension and its associated effects. I found that, contrary to some previous claims, an acute rise of intracranial pressure was not quickly followed by production of optic disc edema. Then, in rhesus monkeys, I produced experimentally chronic intracranial hypertension by slowly increasing in size space-occupying lesions, in different parts of the brain. Those produced raised cerebrospinal fluid pressure (CSFP) and optic disc edema, identical to those seen in patients with elevated CSFP. Having achieved that, I investigated various aspects of optic disc edema by ophthalmoscopy, stereoscopic color fundus photography and fluorescein fundus angiography, and light microscopic, electron microscopic, horseradish peroxidase and axoplasmic transport studies, and evaluated the effect of opening the sheath of the optic nerve on the optic disc edema. This latter study showed that opening the sheath resulted in resolution of optic disc edema on the side of the sheath fenestration, in spite of high intracranial CSFP, proving that a rise of CSFP in the sheath was the essential pre-requisite for the development of optic disc edema. I also investigated optic disc edema with raised CSFP in patients, by evaluating optic disc and fundus changes by stereoscopic fundus photography and fluorescein fundus angiography.

Based on the combined information from all the studies discussed above, it is clear that the pathogenesis of optic disc edema in raised intracranial pressure is a mechanical phenomenon. It is primarily due to a rise of CSFP in the optic nerve sheath, which produces axoplasmic flow stasis in the optic nerve fibers in the surface nerve fiber layer and prelaminar region of the optic nerve head. Axoplasmic flow stasis then results in swelling of the nerve fibers, and consequently of the optic disc. Swelling of the nerve fibers and of the optic disc secondarily compresses the fine, low-pressure venules in that region, resulting in venous stasis and fluid leakage; that leads to the accumulation of extracellular fluid. Contrary to the previous theories, the various vascular changes seen in optic disc edema are secondary and not primary. Thus, optic disc edema in raised CSFP is due to a combination of swollen nerve fibers and the accumulation of extracellular fluid.

My studies also provided information about the pathogenesis of visual disturbances in raised intracranial pressure.

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### Contents

1. Introduction ..... 110
2. Terminologies used for optic disc edema in raised intracranial pressure in the literature ..... 110

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3.	Review of the postulated theories	110
3.1.	Mechanical theories	110
3.2.	Non-mechanical theories	110
3.3.	Combination of a large number of factors mentioned above	110
4.	My studies on the pathogenesis of optic disc edema in raised intracranial pressure	110
4.1.	The sheath of the optic nerve	111
4.1.1.	Immediately behind the eyeball	111
4.1.2.	Posterior to the loose and bulbous part	112
4.1.3.	In the region of the optic canal	112
4.2.	Centripetal flow of fluids along the optic nerve	112
4.3.	Role of compression of the central retinal vein in optic disc edema in raised intracranial pressure	113
4.3.1.	Group 1	113
4.3.2.	Group 2	114
4.4.	Acute intracranial hypertension	116
4.4.1.	Its effect on the optic disc	116
4.4.2.	Its effect on ophthalmic and systemic blood pressures	116
5.	Chronic intracranial hypertension	117
5.1.	Method to produce chronic intracranial hypertension by space-occupying lesion	117
5.2.	Intra-cranial space-occupying lesions in rabbits	118
5.3.	Intra-cranial space-occupying lesions in rhesus monkeys	118
5.3.1.	Sites of the intracranial balloon in rhesus monkeys	118
5.3.2.	Cerebrospinal fluid pressure	118
5.3.3.	Optic disc and fundus changes	120
5.3.4.	Role of fluorescein angiography in optic disc edema	123
5.3.5.	The time interval between the rise of intracranial pressure and development of optic disc edema	124
5.3.6.	Resolution of optic disc edema after reduction of intracranial pressure	126
5.3.7.	Ipsilaterality of the fundus changes	126
5.3.8.	Effect of opening the sheath of the optic nerve on the optic disc edema	126
6.	Pathological studies	128
6.1.	In the first phase of the study	128
6.2.	In the second phase of the study	129
6.2.1.	Light microscopic study	129
6.2.2.	Horseradish peroxidase study	130
6.2.3.	Electron microscopic study	130
6.2.4.	Axoplasmic transport study	131
7.	Summary of my findings providing insight into pathogenesis of optic disc edema in raised intracranial pressure	133
7.1.	Invalidity of previous common theories	133
7.2.	Role of the raised CSF pressure in the optic nerve sheath	133
7.3.	Histopathologic findings	134
7.4.	Horseradish peroxidase findings	134
7.5.	Axoplasmic flow findings	134
7.6.	Electron microscopy findings	134
7.7.	Effect of optic atrophy on optic disc edema	134
7.8.	Stereoscopic color fundus photography and fluorescein fundus angiography findings	134
7.8.1.	Stereoscopic color fundus photography findings	134
7.8.2.	Stereoscopic fluorescein fundus angiography findings	135
7.9.	Role of optic nerve tissue pressure in optic disc edema in intracranial hypertension	137
7.10.	Role of intraocular pressure in optic disc edema in intracranial hypertension	138
8.	Pathogenesis of optic disc edema indicated by my studies	139
8.1.	Primary changes	139
8.2.	Secondary changes	139
8.3.	Conclusion	140
9.	Pathogenesis of visual disturbances in raised intracranial pressure	140
9.1.	Clinical studies	140
9.1.1.	Types of visual disturbances	140
9.1.2.	Fundus findings	141
9.1.3.	Progress	141
9.1.4.	Pathogenesis of visual disorders in clinical studies	141
9.2.	Experimental studies	142
9.2.1.	Types of visual disturbances	142
9.2.2.	Pathogenesis of visual disorders in experimental studies	142
10.	Conclusions and future directions	142
	Conflict of interest	143
	Acknowledgments	143
	References	143

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