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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 pathogeneses of visual disturbances in raised intracranial pressure.

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