

Residual vision activation and the brain-eye-vascular triad: Dysregulation, plasticity and restoration in low vision and blindness - a review

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Vision loss due to ocular diseases such as glaucoma, optic neuropathy, macular degeneration, or diabetic retinopathy, are generally considered an exclusive affair of the retina and/or optic nerve. However, the brain, through multiple indirect influences, has also a major impact on functional visual impairment. Such indirect influences include intracerebral pressure, eye movements, top-down modulation (attention, cognition), and emotionally triggered stress hormone release affecting blood vessel dysregulation. Therefore, vision loss should be viewed as the result of multiple interactions within a "brain-eye-vascular triad", and several eye diseases may also be considered as brain diseases in disguise. While the brain is part of the problem, it can also be part of the solution.

Neuronal networks of the brain can "amplify" residual vision through neuroplasticity changes of local and global functional connectivity by activating, modulating and strengthening residual visual signals. The activation of residual vision can be achieved by different means such as vision restoration training, non-invasive brain stimulation, or blood flow enhancing medications. Modulating brain functional networks and improving vascular regulation may offer new opportunities to recover or restore low vision by increasing visual field size, visual acuity and overall functional vision. Hence, neuroscience offers new insights to better understand vision loss, and modulating brain and vascular function is a promising source for new opportunities to activate residual vision to achieve restoration and recovery to improve quality of life in patients suffering from vision loss.

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