Retinal ischemia is a common cause of visual impairment and blindness. At the cellular level, ischemic retinal injury consists of a self-reinforcing destructive cascade involving neuronal depolarisation, calcium influx and oxidative stress initiated by energy failure and increased glutamatergic stimulation. There is a cell-specific sensitivity to ischemic injury which may reflect variability in the balance of excitatory and inhibitory neurotransmitter receptors on a given cell. A number of animal models and analytical techniques have been used to study retinal ischemia, and an increasing number of treatments have been shown to interrupt the “ischemic cascade” and attenuate the detrimental effects of retinal ischemia. Thus far, however, success in the laboratory has not been translated to the clinic. Difficulties with the route of administration, dosage, and adverse effects may render certain experimental treatments clinically unusable. Furthermore, neuroprotection-based treatment strategies for stroke have so far been disappointing. However, compared to the brain, the retina exhibits a remarkable natural resistance to ischemic injury, which may reflect its peculiar metabolism and unique environment. Given the increasing understanding of the events involved in ischemic neuronal injury it is hoped that clinically effective treatments for retinal ischemia will soon be available [1)].

1)
Osborne NN, Casson RJ, Wood JP, Chidlow G, Graham M, Melena J. Retinal ischemia: mechanisms of damage and potential therapeutic strategies. Prog Retin Eye Res. 2004 Jan;23(1):91-147. Review. PubMed PMID: 14766318.