Alterations at the blood-retinal barriers in Kainic acid treated and optic nerve axotomized rats

by

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ABSTRACT

The purpose of this study was to investigate the alterations at the blood-retinal barriers in rats, by developing two unique models in the lab; first by kainic acid administration and second, by optic nerve axotomy. The animals were divided into two groups; one was injected 10 ul of 200 nM kainic acid intraocularly, while the other group was subjected to optic nerve axotomy. The former group was included saline injected and non-injected controls. Both techniques were carried onto one eye of each animal. The changes induced at the retinal barriers were traced by introducing a fluorescent drug i.e., ethidium bromide at 0.1, 0.5, and 1.0% concentrations intravenously into the animals. A survival time period after the intraocular injection of kainic acid and optic nerve axotomy was given ranging from 3-24 hrs. The circulating time for ethidium bromide was estimated to be 1-3 hrs. Cryostat sections of the retinal tissues of both experimental and control animals were made and then examined using a fluorescent microscope to study ethidium bromide uptake by different cellular layers of the retina.

It was revealed that the various cellular retinal layers demonstrated weak to strong drug activity as observed by the intensity of fluorescence. The uptake of ethidium bromide was mainly in the retinal pigment epithelium, outer nuclear layer, inner nuclear layer and, the ganglion cell layer. The optic nerve trauma resulted in a complete break
down of the retinal barriers as indicated by the rush entra-
ance of the drug in all the retinal cell populations. The
fluorescent uptake in both techniques was observed in the
same retinal cells. In the control animals, the route of
ethidium bromide uptake was opposite to that found in expe-
rimental animals as ethidium bromide was injected intraocu-
larly. Ethidium bromide uptake by different retinal cell
layers came as a result of alterations at the blood-retinal
barriers due to kainic acid and optic nerve trauma.