Corneal exposure
Sodium concentration in aqueous and lens changes

Lloyd J. Bronson, and Moshe Lazar**

Reversible corneal exposure cataracts have been observed in many rodents. The postulated mechanism has been one of transcorneal evaporation of water increasing the concentration of the aqueous and osmotically affecting the lens. In this experiment, changes in the colligative properties of hamster aqueous were measured as related to corneal exposure and the development of lens opacity. A rise of sodium concentration of aqueous was found to parallel the appearance and progression of lens opacity.

Key words: exposure cataract, aqueous concentration, sodium ion, colligative properties, reversible opacity.

The occurrence of corneal exposure cataracts in hamsters, mice, rats, and guinea pigs has been reported by several authors.1-3 The postulated mechanism has been one of trans-corneal evaporation of water resulting in an increased concentration of aqueous humor and its subsequent osmotic action on the lens membrane. However, no direct determinations of changes in concentration of aqueous have been made. Sodium ion comprises 95% of the cations present in aqueous humor, so that measurement of changes in the levels of this ion would appear to substantially reflect changes in the total colligative properties of the aqueous.4 The present experiment was designed to determine the effect of corneal exposure on sodium concentration of the aqueous, and the relationship of this to the development of lens opacities.

Methods and results

Adult Syrian Golden hamsters were anesthetized with 15 mg. of sodium pentobarbital injected intraperitoneally. Pupils were dilated with 10 percent phenylephrine drops. Eyes were proptosed and lids were maintained in an open position by retraction with tape.

In one group of animals, one eye was taped shut and one exposed. Aqueous samples were obtained from both eyes at 8, 20, 40, and 60 minutes of exposure. Immediately prior to collection of aqueous, the fornix and periorbital skin were dried with tissue. Animals were held upside down with traction on the lids maintaining proptosis. Corneas were punctured with a No. 25 gauge needle, resulting in the formation of a droplet of aqueous suspended from the corneal surface. Samples were collected with 10 μl pipettes and sodium concentration was measured by flame photometry. Only one aqueous sample was obtained...
Fig. 1. Sodium concentration of aqueous as related to corneal exposure.

from each eye. Samples of blood were obtained by cardiac puncture to determine serum sodium concentration. The results are presented in Fig. 1. Each point represents an average of ten eyes.

Sodium concentration of aqueous before exposure was 155 mEq per liter and that of serum, 148 mEq per liter. Cataracts were seen to develop after approximately eight minutes of exposure, the opacity initially appearing along the anterior suture lines. Aqueous sodium concentration at this time was found to have risen to 180 mEq per liter. Anterior chambers were noted to be progressively shallower and globes progressively softer as exposure proceeded. At 20 minutes of exposure, the opacity had progressed so as to involve the entire anterior surface of the lens and sodium concentration was found to be 209 mEq per liter. At 40 minutes, the aqueous sodium concentration was 254 mEq per liter, and at 60 minutes it had risen to 325 mEq per liter. At this time the cataracts were very dense, though still limited to the extreme anterior surface of the lens. In all fellow eyes taped shut, the aqueous sodium concentrations remained within the normal range (150 to 160 mEq per liter). These determinations were carried out simultaneously with those of exposed eyes and served as controls.

In another group of animals, both eyes were exposed for 60 minutes, at which time a sample was taken from the left eye, while the right eye was taped shut so as to allow reversal of lens opacity. Aqueous samples were then taken from right eyes at first sign of reversal, at completion of reversal, and 24 hours after reversal.

In eyes thus closed, the first sign of clearing of the lens occurred in 15 minutes, and consisted of the appearance of small holes or areas of translucency in the opacity, rather than a uniform reversal. Sodium concentrations of samples drawn at this time had fallen to 207 mEq per liter.

Total reversal of opacity occurred approximately 75 minutes after closure of eyes, at which time sodium aqueous concentration averaged 177 mEq per liter. Samples obtained 24 hours after reversal showed the return of sodium concentration to normal, pre-exposure levels.

Discussion

Goldman and Rabinowitz postulated that open lids allow evaporation of water through the cornea, causing an increase in aqueous concentration and an osmotic action on the lens, which may be the cause of the opacity. Although changes in aqueous concentration have not hitherto been directly measured, there is much indirect evidence supporting this view. Rodent lenses incubated in vitro in hypertonic solutions develop cataracts which are completely reversible. Fraunfelder and Burns noted that factors facilitating the rate of evaporation, such as increasing the width of the palpebral fissure or blowing hot air across the cornea, increased the rate of development of opacity. Conversely, factors opposing evaporation, such as ap-
application of an air-tight cup or air-tight layers of silicone high-vacuum grease, delay or prevent cataract formation.

In this experiment, changes in concentration of aqueous sodium ion were monitored as corneal exposure and subsequent cataract formation were allowed to proceed. We found an increase of sodium concentration of 34 per cent at the first sign of lens opacity and of 100 per cent at one hour of exposure. These findings, and the observation that the anterior chambers became progressively shallower as exposure proceeded, suggest that transcorneal evaporation and increase in the total aqueous concentration parallels the production of the observed lens opacity. Upon closure of the eye, a return of sodium concentration toward normal, a deepening of the anterior chamber, and a concomitant reversal of the opacity tend to support this view. It seems likely that the sodium is exerting an osmotic effect on the lens, and this is consistent with previous speculation as to the cause of these cataracts.

REFERENCES