gests that the interocular inhibition or "competition" mechanism postulated to occur in the cortex during visual development may be responsible for the difference between the monocularly and binocularly fogged stereoacuity decrements in the main experiment. As in the animal studies, contrast difference between the eyes' images seems to trigger inhibition of the contrast-deprived eye, while binocularly symmetric contrast loss does not.

The present results, together with related animal studies that demonstrate the extreme vulnerability of the developing visual system to contrast imbalance between the two eyes, hold a number of important clinical implications. First, they emphasize the need for early detection and treatment in infants and children of any condition leading to interocular contrast imbalance if normal binocular vision is to develop. Second, they add weight to the question of whether monocular occlusion therapy for amblyopia improves visual acuity at the price of degrading binocular vision potential. Findings in the cat suggest that such occlusion during the "sensitive period" (ie, the neural plasticity interval when an amblyope would be treated) causes an irreversible loss of stereopsis. Alternative amblyopia therapies aimed at better maintenance of binocular vision have been proposed, such as graded vision reduction rather than total occlusion of the sound eye or binocular occlusion. Successful results with some patients are reported. However, clinical experience has found graded occlusion ineffective at treating deep amblyopia, and animal experiments indicate that prolonged binocular occlusion may lead to deprivation effects (though it also may extend the sensitive period). Thus, whether these or other techniques will succeed in maintaining binocular vision in most amblyopes while improving monocular visual acuity remains to be determined.

Key words: amblyopia, image contrast, interocular inhibition, stereoacuity


References

3. Ingram RM and Walker C: Refraction as a means of predicting squint or amblyopia in preschool siblings of children known to have these defects. Br J Ophthalmol 63:238, 1979.

Proptosis and Increase of Intraocular Pressure in Voluntary Lid Fissure Widening

Robert A. Moses, Patricia E. Carniglia, Walter J. Grodzki, Jr., and Jonas Moses

The eyeball proptoses about 0.5 mM and increases in intraocular pressure (IOP) about 2 mmHg with voluntary widening of the lid fissure. The findings probably result from the retraction of the upper lid into the orbit, thus increasing the volume of orbit contents and forcing the eyeball forward. Decrease in IOP with repeated tonometry may result, in part, from decay of the increased pressure induced by lid fissure widening. Invest Ophthalmol Vis Sci 25:989-992, 1984

When the eyelids voluntarily are opened widely, the eyeball moves forward perceptibly and downward slightly. No proptosis is seen, however, if the lids are opened passively, as by retractors. The mechanism of the globe movement with voluntary lid fissure widening has been attributed variously to relief of lid pressure on the eyeball or to displac-
ment of orbital soft tissues by the upper lid retracted into the orbit. If the former of these explanations is correct, the intraocular pressure (IOP) should decrease with voluntary lid fissure widening; if the latter is correct, the IOP should increase with voluntary lid fissure widening when orbital pressure is increased and the globe is forced forward against the restraint of the rectus muscles.

Nineteen volunteers of both sexes between the ages of 21–26 participated as subjects and were seated with head erect. Informed consent was obtained from each subject prior to participation in the study.

Materials and Methods. Photography: A bite-bar was extended forward 50 cm to carry a fixation target at eye height, and laterally, at right angles to the line of vision, to carry a camera focused on the corneal profile (Fig. 1). A light pipe illuminated the skin of the nose so that the lids and cornea were photographed in profile (Fig. 2). The entire assembly was counter-balanced over a pulley so that there was no force exerted on the subject's head when gripping the dental wax impression with his or her teeth. Photographs were taken in "normal" primary gaze and after the command to "open your eyes wide." After the subject released the bite-bar, a millimeter scale was brought into focus and photographed at the same camera setting. Measurements of the films at ×4 total magnification were from the corneal apex to the edge of the film frame, the shadow of a part of the camera and, hence, fixed to the dental impression.
The distance between the lids was measured from the angle between lower lid margin and sclera to the upper lash line (where the upper lashes insert into the lid skin) since the angle between upper lid margin and cornea or sclera often was obscured by lashes.

**Tonometry:** A Perkins applanation tonometer equipped with a Kaufman reduced prism was used. With this prism, it is possible in most subjects to avoid touching the eyelashes while the lids are in habitual position; but in some subjects, the subject must be requested to open the eyes “a little.” These measurements were made on a separate occasion without the bite-bar-camera assembly. The subject was seated in primary gaze. Pressure was measured with lids in “normal” position (P1), with lids “wide open” (P2), and again with lids “normal” (P3). The change in IOP with lid widening, \( \Delta P = P_2 - (P_1 + P_3)/2 \). The average of pressures before and after lid fissure widening was used to compensate for the usual decrease in IOP with repeated tonometry.

In both procedures, the subject was instructed to breathe normally. There was no evidence at any time that any subject performed a Valsalva maneuver.

**Results.** Proptosis with voluntary palpebral fissure widening was corroborated in all subjects. The mean proptosis was 0.46 ± 0.22 (SD) mM with a mean increase in lid fissure width of 3.27 ± 1.37 mm (N = 19) (Fig. 3). IOP increased with lid fissure widening in 18 of 19 subjects, mean 1.88 ± 1.35 (SD) mmHg, N = 19 (Fig. 4). Increase in IOP was not correlated with degree of proptosis (\( r^2 = 0.01266 \)).

**Discussion.** It would appear that Birch-Hirschfeld’s explanation of ocular proptosis with voluntary lid fissure widening is probably correct; that the retracted upper lid occupies orbital space and forces the eye forward.

If lid fissure widening of 3.27 mM causes 0.46 mM of proptosis at least \( 4\pi(0.46)^2 = 208 \text{ mM}^2 \) volume must have been added to the orbit to displace an eye of 12-mM radius 0.46 mM. If the canthi are opposed diametrically on the surface of the globe, elevation of the lid 3.27 mM would expose a lunar area of globe approximately \( 2\pi^2 \theta = 2(12)^2 \times \frac{3.27}{12} = 78.5 \) mM\(^2\) (approximate since the angle \( \theta \) radians was approximated from fissure “height” in a straight line to the lash line rather than as a distance along the globe arc). If, indeed, the eye is proptosed by the volume of the lower lid segment of lid retracted into the orbit, we may calculate lid thickness. Lune surface area x lune thickness = 208/78.5 = 2.6 mM a plausible thickness of retracted lid.

The mean proptosis of 0.46 mM is accompanied by a mean increase in IOP of 1.9 mmHg. The authors have found that it requires from 23–78 g/mM, or on the average 38.9 g/mM, to displace the globe forward or backward, the force being linear to the displacement for ±1 mM displacement. (The mean force per mM displacement was omitted from the original report.) The globe displacement on lid fissure widening implies, therefore, that a force of some 0.46 mM × 38.9 g/mM = 17.9 g was exerted on the globe. This force over the cross-sectional area of the globe is a pressure of 17.9 g/4.5 cm\(^2\) = 4 gm cm\(^{-2}\) or 2.9 mmHg, 1 mmHg greater than the 1.9 mmHg observed. However, since change in IOP with lid fissure widening was measured in a separate procedure from the change in eye position with lid fissure widening, and a mean figure for force of ocular displacement obtained on other subjects is used, the agreement of the calculated and observed rise in IOP for a given eye displacement is satisfying.

The lack of correlation between degree of proptosis and rise in IOP is probably the result of the greater than three-fold variation in orbital stiffness mentioned above. Thus, a relatively great forward movement against weak retaining structures might induce less IOP rise than a smaller movement against stiffer restraints.

The fall in IOP with repeated tonometry has been explained partly by Kraukau and Wilke as due to tonographic effect, but the present results also might help explain the phenomenon, since we now have evidence that voluntarily opening the eyelids widely, as in common in tonometry, increases IOP appreciably. The fall in IOP then may be a decay of the augmented IOP, perhaps combined with a gradually decreasing effort to hold the eyes wide open as tonometry is repeated. The Kraukau tonometer employed by Krakau and Wilke has a relatively long narrow rod that contacts the cornea; thus, the subject need not widen his lid fissure for tonometry, IOP would not be increased, and no pressure decay curve would be obtained.

**Key words:** intraocular pressure, eyelids, proptosis, lid fissure

From the Department of Ophthalmology and the Oscar Johnson Institute, Washington University School of Medicine, St. Louis Missouri. Supported in part by NEI grant EY-00256, National Institutes of Health, Bethesda, Maryland. Submitted for publication: January 17, 1984. Reprint requests: Robert A. Moses, MD, Department of Ophthalmology, Washington University School of Medicine, 660 S. Euclid Street, St. Louis, MO 63110.

**References**

Decrease of Anterior Ciliary Arterial Pressure with Increased Ocular Pressure

Go Sakimoto and Bernard Schwartz

The relationship of the ocular pressure to the anterior ciliary arterial pressure was studied by measuring the pressure of the anterior ciliary artery using a modified pressure chamber. Of the 40 subjects, 9 were normal, 16 were ocular hypertensive patients, and 15 were primary, open-angle, glaucoma patients. A significant negative correlation was found between the anterior ciliary arterial pressure and the ocular pressure such that the former decreases as the latter increases. These results support the concept that the blood flow in the anterior ciliary artery is from the inside of the eye to the outside. Invest Ophthalmol Vis Sci 25:992-995, 1984

In order to further investigate our previous finding that blood flowed from the inside of the eye to the outside in the anterior ciliary arteries,1 we examined the circulatory dynamics by measuring the pressure of the anterior ciliary artery in normal, ocular hypertensive, and glaucomatous subjects and determined the correlations between pressures in this vessel and ocular pressures.

Materials and Methods. Subjects: As defined previously,2 the study was comprised of normal subjects, ocular hypertensive and open-angle glaucoma patients attending the Outpatient Ophthalmology Service of the Tufts-New England Medical Center (Boston, Massachusetts). Informed consent was obtained from all subjects. None of the normal subjects or ocular hypertensive patients were taking ocular medications. All of the glaucoma patients were receiving antiglaucoma medication.

The pressure chamber method was used for the measurement of the anterior ciliary arterial pressure.2 A transparent plastic film (Saran Wrap) was used on the tip of the pressure chamber for measurement of the anterior ciliary arterial pressure, and a transparent latex membrane (Tonoﬁlm) was used for the measurement of the episcleral venous pressure.

First, ocular pressure and brachial blood pressure were measured with the patient seated. The Goldmann applanation tonometer was used for ocular pressure, and brachial blood pressure was measured in the standard fashion by sphygmomanometer. For the subsequent measurement of episcleral vein and anterior ciliary arterial pressures, the subject’s eye was anesthetized topically with 0.5% proparacaine and the subject was seated before the slit lamp. The anterior ciliary arteries were distinguished from the episcleral veins by wider caliber, more tortuous course, and slightly brighter color.2

The episcleral venous pressure was measured before the anterior ciliary arterial pressure, using the total collapse of the veins as the end point. The measurement was made on an episcleral vein at a point approximately 5 mM from the limbus in the temporal quadrant; the right eyes of 35 patients and the left eyes of four patients were measured. The diastolic arterial pressure was recorded as the first point at which some portion of the anterior ciliary artery showed pulsation. The pressure then was increased and the systolic arterial pressure was recorded as the point at which the artery collapsed totally. Pressure measurements were made on the anterior ciliary artery in the temporal and nasal quadrants approximately 5 mM from the limbus, in the right eye of 36 subjects and in the left eyes of four subjects. Only one eye was measured in each subject.

Ocular pressure was measured by one observer and anterior ciliary arterial and episcleral venous pressure by a second; each was masked to the measurements made by the other. Pressures for the episcleral veins and the anterior ciliary arteries were recorded as the average of three consecutive measurements. The median coefficients of variation (SD/mean \times percent) and, in parentheses, the 30th and 70th percentiles for the reproducibility of each of the sets of pressure measurements were: nasal systolic 5.4 (3.6, 6.6); nasal diastolic 7.5 (5.8, 11.7); temporal systolic 6.6 (5.6, 8.4); temporal diastolic 7.9 (5.1, 11.3); and episcleral venous pressure 7.6 (6.0, 11.1).