STUDIES IN OCULAR RIGIDITY*

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RECENT developments in tonography and applanation tonometry have caused greater attention to be focused on the relation of the elastic properties of the coats of the eye and of ocular rigidity to the intra-ocular pressure.

It is now fairly well established that the coefficient of ocular rigidity, K , is not a constant, as Friedenwald (1937) had suggested, but that it varies with the intra-ocular pressure. In animal eyes, several workers, including Perkins and Gloster (1957a) and Hosni (1960), have found that K generally increases with the pressure in the eye. In human eyes, MacDonald (1955), Gloster and Perkins (1959), Goodside (1959), Prijot and Weekers (1959), Ytteborg (1960a, b), and Drance (1960) have all found that K bears an inverse ratio to the intra-ocular pressure. Friedenwald (1937) originally suggested that K depends on the initial volume of the eyeball, and more recent observations by Phillips and Quick (1960), Ytteborg (1960c), and Sampson and Girard (1961) have confirmed this.

To find out whether variations of rigidity in the individual eye were due primarily to changes in the elastic properties of the outer coat of the eye or to changes in the intra-ocular blood vessels, Hosni (1960) undertook a series of experiments in rabbits and cats. He observed that in animals the coefficient of ocular rigidity rose with the intra-ocular pressure up to 50 cm. saline and then fell slightly. Hosni further found that alteration in the volume of the intra-ocular vascular bed did not appreciably affect the value of K or its variations with pressure. Finally, he demonstrated alterations in ocular rigidity when enucleated eyeballs were immersed in solutions of different osmolarities, a fall in the water content of the tissues being associated with lower values for K and *vice versa*. He did not, however, obtain any significant changes in pressure or in K as a result of haemodilution in the rabbit. Hosni thus concluded, as had Macri, Wanko, and Grimes (1957), that changes in rigidity depended primarily on the elastic properties of the coats of the eye, and that intra-ocular vascular changes had a negligible influence. Ytteborg

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(1960c), on the other hand, found that the coefficient of ocular rigidity was higher in the enucleated than in the living human eye, while Prijot and Weekers (1959) and more recently Prijot (1961) have made similar observations. These workers therefore suggested that the vascular bed of the eye alters with the intra-ocular pressure, and thereby exerts an important influence on ocular rigidity.

Clinical observations on the effect of hydration on the living human eye have been chiefly directed towards an assessment of the effect of haemodilution and water drinking on the intra-ocular pressure. Leydhecker (1950) studied the electrical conductivity of blood to assess the state of haemodilution and found that the maximum decrease in conductivity always coincided with the maximum ocular tension. However, Yonebayashi (1959) and Pistocchi and Tusini (1960), who estimated the degree of haemodilution during the test by a different method, concluded that rises in intra-ocular pressure were not coincident with haemodilution. Recently, Galin, Aizawa, and McLean (1961) directly measured the osmolarity of blood during the water test. They found that the glaucomatous eye was a sensitive osmometer, yet it did not show constant and reproducible alterations in ocular rigidity with changes in pressure. Stepanik (1958) used the method of differential tonometry, and found that the changes in rigidity during the water-drinking test were not significant. Drance (1960) also came to the same conclusion. Draeger and Müller (1960), however, observed that the ocular rigidity fell during the water-drinking test, and they attributed this result to the increase in the intra-ocular pressure rather than to the hydration of the ocular tissues.

These observations suggest that ocular rigidity, besides varying from eye to eye, may also vary in the same eye under different conditions, and that such variations could be attributed in the main to two factors: firstly, alterations in the state of the vascular bed of the eye, and secondly, changes in the physical properties of the cornea and sclera. The present study was therefore undertaken in order to elucidate the influence of alteration of the properties of the corneo-scleral envelope caused by hydration and dehydration, upon the rigidity of the human eye.

Material and Methods

(1) EXPERIMENTS *in vitro*

Human eyes enucleated *post mortem* and rejected for corneal grafting were used; most of them were obtained 24–72 hours after death.

The corneo-scleral envelope was prepared as follows: the extra-ocular muscles, together with conjunctiva and episcleral tissue, were dissected away; the eyeball was carefully supported in one hand while an 8-mm. trephine hole was made over the stump of the optic nerve; the vitreous, lens, retina, and uveal tract were gently removed through the opening, leaving the cornea and sclera intact.

(a) **Relationship of Rigidity to Pressure.**—A special Perspex connector, similar to the metal flange used by Grant (1958), was utilized to make the preparation watertight and to allow entry of fluid into the corneo-scleral envelope (Figs 1 and 2).

PERSPEX FLANGE

FIG. 1.—Cross-section of Perspex connector.

FIG. 2.—Corneo-scleral envelope clamped to connector, and closed off temporarily with a metal stopper.

The preparation, which will henceforth be referred to as the "eyeball", was carefully filled with the perfusing solution, saline or Dextran alternately, ensuring that no air bubble was included. It was then connected by means of short polythene tubes and 3-way taps to a reservoir, an Agla micrometer syringe, and a Sanborn 267B pressure transducer. As shown overleaf in Fig. 3 (diagram) and Fig. 4 (photograph of apparatus in use), the "eyeball" was suspended and completely immersed in the required solution, the temperature of which was maintained at 26° C. and the pH adjusted to 7.4. The solutions used were 0.9 per cent. saline, and 6 per cent. Dextran ("Intradex"—Dextran solution B.P.—Glaxo); the pressure-volume determinations were done in one solution and again after the "eyeball" had been washed and immersed in the other solution for 3–4 hours. 10 μ l. fluid was injected into the "eyeball" at different pressure levels. About fifteen such increments were injected for each set of readings.

FIG. 3.—Schematic representation of perfusing apparatus.

FIG. 4.—Bird's-eye view of "eyeball" photographed from above, showing its connexions to the transducer, syringe, and recorder.

(b) Measurements of Corneal and Scleral Thickness

(i) *Corneal Thickness.*—The corneal thickness was measured with the pachometer (Maurice and Giardini, 1951) before and after the removal of the ocular contents.

After the rigidity had been determined for an eye, the corneal thickness was again measured by means of an electric micrometer as follows: a disc 8 mm. in diameter was trephined from the centre of the cornea and placed on the tray of the instrument under a very light piece of metal (Fig. 5); the micrometer gauge was gently lowered on to its surface, and, at the moment of contact before any pressure was exerted on the tissue, the neon indicator bulb would light up; the electricity supply was from a 120 V dry battery.

FIG. 5.—Electric micrometer, with indicator bulb. N.B. A metal piece placed in the tray prevents any twisting of tissue with the end of the micrometer gauge

The “micrometer” readings could be used as a comparison with the pachometer readings for a given eye.

(ii) *Scleral Thickness.*—As described above, discs of sclera were also measured with the electric micrometer after the thickness of the tissue had been altered by immersion in saline or Dextran respectively.

(2) **WATER-DRINKING TEST.**—In addition to the experiments *in vitro*, the water-drinking test was carried out in 26 patients (46 eyes) with suspected chronic simple glaucoma, measuring the ocular rigidity before and during the test.

Results

(1) EXPERIMENTS *in vitro*

(a) **Relationship of Rigidity to Pressure.**—Characteristic tracings obtained on the Sanborn recorder for an “eyeball”, using saline and Dextran respectively, are shown in Figs 6 and 7 (opposite). The coefficient of ocular rigidity was calculated using the formula:

$$K = \frac{\text{Log}_{10} P_2 - \text{Log}_{10} P_1}{\Delta V_E}$$

where P_1 is the initial pressure at each level of the reservoir, P_2 is the final pressure at each level of the reservoir, extrapolated to zero time, P_{av} is the mean of P_1 and P_2 , and ΔV_E is change in ocular volume. The total volume of fluid injected each time was 10 μ l.

The values of K and average pressure (P_{av}) were recorded for each “eyeball”, using saline and Dextran alternatively, and tests for significance were applied to the calculated values, as well as to the coefficient of correlation which was negative in every case.

The pooled data for eight “eyeballs” were then analysed for possible differences between the values obtained for saline, and those for Dextran (hypertonic) (Fig. 8, overleaf). In every case the values for K were higher in Dextran than in saline. Table I shows that the differences were highly significant.

TABLE I
COMPARISON OF VALUES FOR K IN DEXTRAN AND IN SALINE

Mean K		No. of Observations	D.F.
For Dextran	0.02066	114	113
For saline	0.01679	110	109

Difference between means = 0.00387
Pooled standard error (both series) = ± 0.000805 with 221 d.f.
 $t = 4.80$ with 221 d.f.
 $p < 0.001$

Determining the relationship of pressure to rigidity (P_{av} and K) for the Dextran and saline values, we obtained the following equations:

For Dextran experiments: $K_D = 0.0316 - 0.00037 P_{av}$.

For saline experiments: $K_S = 0.0277 - 0.00036 P_{av}$.

These equations as well as the scatter diagram in Fig. 8 show that a negative correlation was obtained in both types of experiment.

(b) Corneal and Scleral Thickness

(i) **Comparison of Pachometer with Micrometer Readings.**—The values for corneal thickness measured with the pachometer were compared with

FIG. 6.—Tracings from an “eyeball”, using normal saline.
(a) Paper speed, 0.50 mm./sec. (b) Paper speed, 10.00 mm./sec.

FIG. 7.—Tracings from an “eyeball”, using 6 per cent. Dextran.
(a) Paper speed, 0.50 mm./sec. (b) Paper speed, 10.00 mm./sec.

FIG. 8—Scatter diagram for K at different levels of pressure. Pooled data from eight “eyeballs”, for saline and Dextran. The calculated regression line for each group is indicated.

those obtained using the micrometer on corneal discs. The values corresponded fairly well, as can be seen from the histogram (Fig. 9), and with the

FIG. 9—Histogram to show values for corneal thickness, measured with the pachometer and electric micrometer respectively.

exception of "eyeballs" Nos. 14 and 19, the micrometer readings were higher than those of the pachometer. This is what one would expect in view of the additional trauma applied to the tissues in trephining out the discs, and the consequent imbibition of fluid from the cut edges of the discs.

(ii) *Effect of Saline and Dextran on the Thickness of Cornea and Sclera.*—The mean values for the corneal thickness in saline and Dextran obtained with the pachometer are shown with their standard errors in Table II. Here we find that the cornea is significantly thinner after immersion in 6 per cent. Dextran than after immersion in normal saline.

TABLE II
CORNEAL THICKNESS IN SALINE AND IN DEXTRAN, MEASURED WITH THE PACHOMETER

Pachometer Readings	Corneal Thickness	
	Saline	Dextran
Mean Value (mm.)	1.170	0.615
Standard Error	± 0.013	± 0.021
No. of Readings	14	14

$t = 18.2$ at 26 d.f.

$p < 0.001$

The scleral thickness could be measured only with the micrometer, since optical methods are not satisfactory for this translucent tissue. We therefore measured discs of sclera in saline and in Dextran, and compared its changes of thickness with those of the cornea. The results of the comparison are given in Table III, in terms of their mean values.

TABLE III
MEAN VALUES OF THICKNESS OF DISCS OF CORNEA AND SCLERA (MICROMETER) IN SALINE AND IN DEXTRAN

Micrometer Readings	Corneal Thickness (mm.)		Scleral Thickness (mm.)	
	Saline	Dextran	Saline	Dextran
Mean Value	1.46	0.73	0.84	0.75
Standard Error	± 0.068	± 0.024	± 0.039	± 0.035
No. of Readings	21	21	21	21

$t = 10.56$ at 40 d.f.
 $p < 0.001$

$t = 1.73$ at 40 d.f.
 $p > 0.1$

These values show that the sclera also becomes thinner in Dextran than in saline, but that the change is neither so dramatic nor so significant as in the case of the cornea.

(iii) *Effect of Thickness of Cornea and Sclera on Coefficient of Rigidity, K.*—For each of the "eyeballs" the values for K which had been obtained at different levels of pressure (Fig. 8) were plotted in terms of the thickness of

the ocular coats. Fig. 10 shows this relationship as applied to corneal thickness, again comparing the behaviour in saline and in Dextran. The overall picture shows that the rigidity was higher when the cornea was thinner.

FIG. 10.—Ocular rigidity (K) related to corneal thickness in saline and in Dextran.

As we have already seen, sclera does not change in thickness as much as cornea when placed in solutions of different osmolarities; Fig. 11 (opposite) shows how these alterations in scleral thickness are related to the rigidity of the "eyeball". In this case also we find that the rigidity is higher when the sclera is thinner.

(2) WATER-DRINKING TEST.—In the water-drinking tests none of the 26 patients showed a rise in intra-ocular pressure of more than 7 mm. Hg with the applanation tonometer. The ocular rigidity was determined from applanation and Schiötz readings in each of the 46 eyes, but it did not show any consistent relationship to the changes in pressure. The water-drinking test thus proved to be inconclusive as regards the effects of hydration on the rigidity of the eye.

Discussion

Our procedure differed from that employed by other workers measuring ocular rigidity *in vitro*, in that the isolated corneo-scleral envelope was used. This had the advantage that it was possible to bathe both surfaces of the cornea and sclera with the osmotically active solutions and so obtain a maximal effect. Also, direct measurements of the changes induced in corneal and scleral thickness are clearly more satisfactory than assessments of the uptake or loss of fluid by weighing the whole eye (Hosni, 1960), in which incalculable changes in the water content of the lens and vitreous may occur.

FIG. 11.—Ocular rigidity (K) related to scleral thickness in saline and in Dextran.

The behaviour of the preparations *in vitro* resembled that of the intact eye insofar as the coefficient of rigidity, K , had the same order of magnitude and a similar variation with intra-ocular pressure as those found clinically. Changing the osmolarity of the perfusing solution caused an alteration in the values for K , which were significantly higher in Dextran (hypertonic) than in 0.9 per cent. saline (isotonic) (see Table I and Fig. 8). In the absence of nervous and vascular influences, the changes in rigidity could be due only to alterations in the elastic properties of the coats of the preparation *in vitro*.

Our measurements of corneal thickness with the pachometer, as well as of discs of cornea and sclera, indicated without any doubt that these tissues were thinner in 6 per cent. Dextran than in 0.9 per cent. saline, although the changes were much more extensive in the corneal than in the scleral tissue (Figs 10 and 11). On the basis of these findings we are convinced that marked thinning of the corneo-scleral envelope by dehydration causes a significant increase in ocular rigidity.

It has been reported by various authors, including Remington (1957) and Wiegiersma (1955), that the thinner a material the higher is its rigidity; this applies not only to synthetic substances (*e.g.* cellophane) but also to biological materials such as muscular tissue. Moreover, in patients with hydration of ocular tissues due to thyrotropic exophthalmos, Weekers and Lavergne (1958) as well as Becker and Gay (1959) found that ocular rigidity was lower than normal.

It appears that the pattern of pressure-volume or rigidity relationships may be different in human and in animal eyes (Perkins and Gloster, 1957b; Macri, Wanko, and Grimes, 1957), and that this difference between species may account for the findings of Hosni (1960) regarding the relationship of ocular rigidity to thickness of the coats of the eye. The results of the present series of experiments are in general agreement with the findings in other tissues and with the clinical observations of Weekers and Lavergne (1958), and others.

The general conclusion to be drawn from this investigation is that gross changes in the hydration of the corneo-scleral envelope, accompanied by measurable alterations in corneal thickness, must occur before it is likely that there will be an appreciable modification of ocular rigidity. The inconsistent changes in ocular rigidity which occurred during the water-drinking test were similar to those found by Galin and others (1961). In future studies it would seem advisable to follow the course of haemodilution during the water-drinking test, as well as to assess the extent to which the changes in the composition of blood in certain pathological conditions modify the rigidity of the eye. At present it would seem that variations in the rigidity of the individual eye are attributable to changes in the intra-ocular vascular bed rather than to alterations in the physical properties of the cornea and sclera.

Summary

(1) There is some evidence that rigidity varies in the individual eye. These variations could be due either to changes in the elastic properties of the coats of the eye or to alterations in the intra-ocular vascular bed.

(2) Determinations of rigidity were carried out *in vitro* on corneo-scleral envelopes prepared from enucleated human eyes. The degree of hydration of the cornea and sclera was varied by bathing the tissues in solutions of different osmolarities; alterations in the water content were assessed by measurements of the thickness of the cornea and sclera.

(3) Reduction of the water content of the cornea and sclera caused an increase in ocular rigidity, while hydration of the tissues had the opposite effect. Gross changes in thickness were necessary before any significant changes occurred in rigidity.

(4) Water-drinking tests in a series of patients with suspected glaucoma gave negative results, and no consistent changes were found in ocular rigidity during the tests.

(5) It is concluded that alterations in the physical properties of the coats of the eye are unlikely to be responsible for variations in rigidity in an individual eye.

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