DETERMINANTS OF CORNEAL TEMPERATURE*†

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THE surface temperature of the cornea is determined by adjacent ocular and extra-ocular tissues—an internal environment—and by conditions prevailing external to the body—an external environment. Any investigation of their effects is rendered more complex than would otherwise be the case in that the external environment is intermittently excluded during blinking so that, when the lids are closed, the thermal environment of the cornea is exclusively internal. The effect on corneal and pericorneal temperature of lid closure and opening (Braendstrup, 1952; Schwartz, 1964), environmental temperature (Schwartz, 1965), and inflammatory ocular disease (Huber, 1960) have been investigated previously. These authors measured temperature by contact; Zeiss (1930) also investigated the effects on corneal temperature of ocular inflammation but here a radiometric method was used. The purpose of this paper is to examine the various factors that determine corneal temperature and *inter alia* those that cause a difference in temperature between the right and left cornea of an individual.

Material and Methods

The instrument used for measuring corneal temperature is a bolometer (Fig. 1). This essentially consists of a balanced Wheatstone bridge, one radiation-sensitive arm of which is placed in the path of the spectrum emitted from the surface of which temperature is to be measured; this emitted radiation is "chopped" at 12.5 times a second and compared with that emitted by a built-in black-body radiator kept at a constant temperature. The radiation incident on the arm of the bridge produces a change in temperature and hence a change in resistance. The resulting change in current flow is amplified and recorded directly as a temperature reading on a dial (Fig. 2) which, at the calibration scale used, reads from 26 to 4°C. in units of 0.1°C. The bolometer is sensitive to infra-red radiation in the range of 1 to 25 μ .

The method of use is simple (Fig. 3). The bolometer is advanced to the surface whose temperature is to be measured and an almost instantaneous reading obtained on the dial. The distance between the bolometer and surface determines the extent of area of which the temperature is measured; with this particular instrument a separation of 0.5 cm. measures the temperature of an area having a diameter of 10 mm.

In taking measurements the subject was allowed to equilibrate in a room in which the temperature—measured with an air thermistor probe (Fig. 4)—was kept between 18 and 26°C., ambient conditions which require no special equipment. In addition, draughts were excluded as far as possible and any direct artificial radiation source (sun-light, radiator, electric light) to the subject's face excluded. A 15-minute period of equilibration was chosen as a reasonable com-

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FIG. 1.—The bolometer; the entry portal of the bolometer is within and smaller than the white ring.

FIG. 2.—Amplifier and recording dial.

FIG. 3.—Distance separating the cornea and the entry portal of the bolometer is approximately 0.5 cm.

FIG. 4.—Air thermistor probe.

promise between possible extremes. At the end of this period the subject was directed to look at a fixation target 8 ft away and voluntarily to retract both lids—thus exposing the whole cornea. Temperatures were then recorded, the average of three readings being calculated. Between each measurement the subject was directed to blink. If voluntary retraction left part of the cornea covered then the upper lid was manually retracted; this removes the skin of the lid from the entry portal of the bolometer, and allows the temperature of the whole cornea to be recorded (Mapstone, 1968a).

The factors investigated, together with the methods used, are listed below:

(1) *Environmental Temperature*.—The corneal temperatures of four subjects were recorded on seventeen separate occasions at 3- to 4-day intervals spread over a period of 8 weeks. All measurements were made in the late afternoon after a 15-minute period of equilibration.

(2) *Lid Retraction*.—The cornea was anaesthetized with Novesine and the subject left for 15 minutes. Corneal temperatures were then recorded, a lid speculum was inserted, and the cornea was exposed to the environment for 5 minutes. At the end of this period corneal temperatures were again recorded.

(3) *Lid Closure*.—Corneal temperatures were recorded after a 15-minute period of equilibration and the subject was then directed to keep both eyes closed for 5 minutes when corneal temperatures were again recorded.

(4) *Blinking*

(a) The left eye was anaesthetized with Novesine and, after 15 minutes, the temperatures of both corneae were measured. The lids of the left eye were then separated with a lid speculum and the exposed cornea irrigated with cold saline running at 160–180 drops a minute. The patient was recumbent during the irrigation, the duration of which was 10 minutes. The saline was running from a bottle suspended by a drip stand and had previously been frozen and then allowed partially to thaw. At the end of this period the speculum was removed, the patient allowed to blink, and the temperatures of both corneae recorded at half-minute intervals for half an hour.

(b) On another day the left cornea of the same subjects were irrigated under precisely similar conditions except that no speculum was inserted, *i.e.*, blinking was allowed. Corneal temperatures were recorded before and at half-minute intervals after irrigation.

(5) *Tears and Tearing*.—In the experiment outlined above, the right eye was not irrigated. The change in corneal temperature of this side was recorded.

(6) *Anterior Uveitis*.—Four patients with unilateral anterior uveitis were followed throughout the disease and the temperature increase of the cornea of the inflamed eye relative to that of the contralateral normal eye recorded.

(7) *Carotid Artery Disease*.—The corneal temperatures of four patients with proven carotid artery stenosis and one with a carotico-cavernous fistula were recorded.

(8) *Posterior Segment Pathology*.—The corneal temperatures of five patients with malignant melanoma of the choroid and one with a metastasis from a renal carcinoma were recorded.

Results

(1) *Environmental Temperature*.—Fig. 5 (overleaf) records the effect of a change in environmental temperature on right and left corneal temperatures in four normal subjects. The range of environmental temperatures investigated was 18° to 27°C. Table I (overleaf) records the gradients of these graphs, *i.e.*, the change in corneal temperature per degree change in environmental temperature over this 10° range.

FIG. 5.—Effect of environmental temperature change on corneal temperature.

(2) **Lid Retraction.**—Table II (opposite) shows the effect on corneal temperature of exposing ten corneae to the environment for a 5-minute period. All ten showed a fall in temperature with a range of 0.6 to 1.6°C. and a mean of 1.1°C. The fall in temperature is of the same order in the right and left corneae, so that the maximum temperature difference recorded between the two after exposure, was 0.3°C.

(3) **Lid Closure.**—Table III (opposite) shows the effect of lid closure for 5 minutes on the temperature of ten corneae. All showed a rise in temperature with a range of 1.1 to 2.2°C. and a mean of 1.5°C. As soon as the lids are opened after a period of closure, corneal temperature falls rapidly and the temperatures recorded in the Table are those of the first measurement

TABLE I
SLOPE OF REGRESSION LINES CALCULATED
FROM FIG. 5

Cornea	Right	Left
Fig. 1a	0.153	0.162
Fig. 1b	0.111	0.113
Fig. 1c	0.149	0.162
Fig. 1d	0.151	0.157
Mean	0.145	

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TABLE II
EFFECT OF LID RETRACTION ON CORNEAL TEMPERATURE (°C.)

Environmental Temperature	Initial Temperature		Final Temperature		Difference	
	R	L	R	L	R	L
23	34.7	34.5	33.5	33.2	1.2	1.3
19	35.0	35.0	34.4	34.2	0.6	0.8
24	35.1	35.1	33.5	33.5	1.6	1.6
21	34.4	34.6	33.3	33.4	1.1	1.2
20	34.1	34.2	33.2	33.5	0.9	0.7

TABLE III
EFFECT OF LID CLOSURE ON CORNEAL TEMPERATURE (°C.)

Environmental Temperature	Initial Temperature		Final Temperature		Difference	
	R	L	R	L	R	L
24	34.5	34.7	35.9	35.8	1.4	1.1
23	34.0	33.9	35.3	35.4	1.3	1.5
23	35.5	35.5	36.8	36.6	1.3	1.1
19	34.0	34.0	35.2	35.4	1.2	1.4
19	34.1	34.2	36.1	36.4	2.0	2.2

made. Again the temperature change is of the same order in the right and left corneae so that the maximum temperature difference between the two after lid closure is 0.3°C.

(4) **Blinking.**—Fig. 6 shows the effect of irrigating the left eye with saline, blinking being prevented by a speculum. In (a) the temperature fall is from 33.6 to 27°C., *i.e.*, 6.6°C. and in (b) the fall is from 33.6 to 28.1°C., *i.e.*, 5.5°C. The first temperature measured at the end of cooling was taken after removal of the speculum and after the subject had

FIG. 6.—Effect of irrigating left eye (a) and right eye (b) with ice-cold saline, blinking being prevented with a lid speculum. Air temperature 21°C.

reformed his pre-corneal film by blinking. If the temperature was taken with the speculum in place, then the reading was that of the cold irrigating saline; blinking wipes this away and allows the true surface temperature to be recorded. The time taken for the corneae to regain their initial temperatures was 24 minutes in Fig. 6*a* and 18 minutes in 6*b*, most of the increase taking place in the first 10 minutes.

Fig. 7 shows the effect of cooling these same two corneae, but here blinking was allowed. The fall in temperature was 0.7°C. in (a) and 1.1°C. in (b). In the first 10 minutes after cooling, both these corneae became 0.9°C. warmer than before irrigation.

FIG. 7*a* and 7*b*.—Effect of irrigating left eye only with cold saline, blinking not being interfered with. Air temperature 23°C.

(5) **Tears and Tearing.**—In Fig. 6 the temperature of the right non-irrigated cornea was recorded. In both cases there is a rise in temperature, 1.5 and 1.4°C. In Fig. 7 the temperatures of both corneae increase after irrigation by 0.9°C. The common factor in both instances is an increase in the rate of tear production.

(6) **Anterior Segment Inflammation.**—Fig. 8 shows the temperature increase of the cornea of the inflamed eye relative to that of the opposite non-inflamed eye throughout the period of inflammation. The maximum relative temperature increase was 2.4, 2.2, 1.1, and 1.3°C. in *a*, *b*, *c*, and *d* respectively. Clinically, the temperature increase was correlated with the severity of the inflammation; if signs and symptoms were obtrusive then the cornea was hot, and as the eye became white and quiet the temperature difference disappeared.

FIG. 8.—Graphs of temperature difference between cornea of inflamed eye and contralateral normal eye, in four patients with unilateral anterior uveitis.

(7) **Carotid Artery Disease.**—Table IV (opposite) records the corneal temperature of five patients with impaired blood supply to one eye resulting from carotid artery pathology (proven by angiography). In all cases the cornea of the affected side had a lower temperature than that of the opposite side. The range was 0.8 to 1.3°C. (mean 1.1).

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TABLE IV
EFFECT OF CAROTID ARTERY PATHOLOGY ON CORNEAL TEMPERATURE (°C.)

Environmental Temperature	Lesion	Corneal Temperature		Difference
		Right	Left	
19	Stenosis Right ICA	32.6	33.4	0.8
22	Stenosis Left ICA	34.7	33.7	1.0
24	Stenosis Left ICA	35.0	33.8	1.2
19	Stenosis Left ICA	33.9	32.8	1.1
20	Carotico-cavernous Fistula Left	34.9	33.6	1.3

ICA = Internal carotid artery.

(8) **Posterior Segment Pathology.**—Table V records the corneal temperatures of six patients with posterior segment neoplasms. The maximum temperature difference between the right and left corneae was 0.3°C.

TABLE V
EFFECT OF POSTERIOR SEGMENT PATHOLOGY ON CORNEAL TEMPERATURE (°C.)

Environmental Temperature	Neoplasm	Corneal Temperature		Difference
		Right	Left	
19	MM Left	33.4	33.6	0.2
21	MM Left	34.2	33.9	0.3
17	MM Right	32.7	32.6	0.1
22	MM Right	35.4	35.5	0.1
20	MM Right	34.5	34.2	0.3
21	Metastasis Left	35.4	35.4	0.0

MM = Malignant melanoma.

Discussion

The summated effect of the heat sources to the cornea determine its temperature. These sources are positive, producing a temperature increase, negative, producing a temperature decrease, or both negative and positive, capable of producing a rise or fall according to circumstances. In general the heat sources to a tissue are three:

- (1) Endogenous metabolic heat.
- (2) Exogenous metabolic heat convected *via* the vascular system.
- (3) Environmental heat, either from adjacent tissue—the internal environment, or from the environment external to the body.

Since the cornea is both metabolically torpid and avascular, its main sources of heat must be environmental. Fig. 9 (overleaf) shows that these sources are four in number, *viz.*, the external environment, the lids, tears, and the aqueous humour.

Fig. 5 shows that the result of a decrease in environmental temperature is to produce a linear drop in corneal temperature—a mean fall of 0.145°C. per degree fall in environmental temperature over the temperature range 18 to 27°C. Schwartz (1965) derived a regression line with a slope of 0.23 in rabbits; the discrepancy may in part be explained by the in-

FIG. 9.—Factors affecting corneal temperature.

ICA = internal
ECA = external
CCA = common

} carotid artery

Stippled areas represent vascular tissue.

frequent blinking of the rabbit. This fall in temperature or, what amounts to the same thing, loss of heat, occurs mainly by convection and radiation; conduction plays a small part since air is such a poor conductor of heat. Fig. 5 also shows that the effect of a drop in environmental temperature on the right and left corneae is the same, indeed, unless there were a large difference between the temperatures of the two corneae, there would be no reason to suppose that this should not be so. Hence this factor, whilst it may change the absolute value of a corneal temperature, should not produce a difference in temperature between the right and left corneae.

The effect of the lids on corneal temperature varies, the results indicating that with the lids open the temperature falls on average 1.1°C . over a 5-minute period and that with the lids closed it rises 1.5°C . again over a 5-minute period. Schwartz (1964) in rabbits and Braendstrup (1952) in man similarly found a temperature increase with lid closure. Opening the lids exposes the cornea to the environment and if environmental temperature is lower than corneal temperature, heat loss occurs by convection and radiation. With lid closure this loss is prevented and, in addition, the cornea is exposed to a new thermal environment—the vascular palpebral conjunctiva. This part of the lid is protected from cold conditions that may prevail in the external environment so that the perfusing blood has a temperature approaching that of the body core; Schwartz (1965) showed in rabbits that the temperature of the inferior fornix approached that of the rectum and, that it varied quantitatively like the rectal temperature with a decrease in environmental temperature. Since the fornix is usually hotter than the environment, heat must pass by conduction from the palpebral conjunctiva into the cornea.

In this context blinking may be regarded as interrupted lid closure or interrupted lid opening, and its effect will be a compromise between these two. Ploman, Engel, and Knutsson (1928) showed that during a blink the act of lid closure occupies 0.05 sec., the closure is maintained for 0.15 sec., and this is followed by lid opening taking 0.2 sec.; thus the whole blink lasts approximately 0.4 sec. Adler (1965) gave an inter-blink period of 2.8 sec. in men and 4.0 sec. in women. During the actual blink the whole of the cornea is not covered by the lid for 0.4 sec. but assuming this to be so for half of this period—0.2 sec.—then during waking hours a cornea is covered by palpebral conjunctiva rather

than exposed to air for $0.2/3.2 \times 100$ per cent. = 6 per cent. of the time, *i.e.*, during this period heat loss to the environment is prevented and heat is gained from the lids.

Blinking also agitates the pre-corneal tear film, propelling cold tears to the lacus lacrimalis and mixing the film with warm tears secreted at body-core temperature in the recess of the superior fornix. Thus with each blink a warm lacrimal secretion is layered across the cornea. Finally, the act of blinking in a still atmosphere alternately expels and then sucks in a new layer of air over the surface of the pre-corneal film, creating in effect a draught.

Fig. 6 shows the effect of blinking when the cornea is exposed to a severe thermal stress. When blinking was prevented corneal temperature fell by 6.6 and 5.5°C . (Fig. 6), whereas with blinking it fell by only 0.7 and 1.1°C . (Fig. 7). Thermally speaking, air is cold water "spread out thin" and it is reasonable to argue from this that the effects of cold air also are vitiated to some extent by blinking. The conditions of this experiment exclude any interference from tears—they are washed away by the irrigation—and thus make the interpretation more simple.

The effect of the lids on corneal temperature is therefore four-fold: a passive prevention of heat loss during a blink, a more active heat transfer during the actual blink, a layering of warm tears across the surface of the cornea, and an air-conditioning effect caused by the movement of the lids. The total effect of these factors is difficult to assess, but only the movement of air can cause loss of heat to the cornea. This, however, will not be so if the environmental temperature is greater than body core temperature and evaporation from the pre-corneal film is prevented. Since there is no reason to suppose that the effect on the right cornea is in any way different from that on the left (lid closure and lid opening produced similar effects on both corneae), the lids cannot *per se* produce a difference between the temperatures of the right and left corneae, and, some other factor or factors must be responsible.

Tears are secreted in the recess of the superior fornix, protected from the external environment by the overlying lid. The temperature of this area approaches that of the body core (Schwartz, 1965) to which the temperature of the tears must also approximate. From here they pass, propelled by the blink, across the cornea to the medial canthus, giving heat both to the environment, and to the cornea as they pass. The effect of tearing on corneal temperature is shown in Figs 6 and 7; the temperature of the cornea of the eye not exposed to the cold saline rises by 1.4 and 1.5°C . (Fig. 6), and in Fig. 7 both corneae are seen to become 0.9°C . hotter than the initial temperature 10 minutes after irrigation had ceased. In the first instance tearing occurs in the non-irrigated eye as a result of the insult to the opposite eye, and in the second tearing is a direct result of the irrigation; the rise in corneal temperature is attributed to this.

Of the factors affecting corneal temperature, a rapid change (*i.e.*, in a matter of seconds) can result only from tearing since the other factors need some time to act. When taking temperatures with a bolometer, it is frequently observed that after the first few readings the measured temperature increases by anything up to 1.0°C . Zeiss (1930) also noted temperature fluctuations of similar magnitude. This is caused by tears reflexly produced either by voluntary lid retraction to expose the required amount of cornea, or by manipulation of the lids by the experimenter. The excess of tear production need not be gross, but, if additional corneal temperature readings are needed, then the subject must equilibrate for a further 15 minutes. However, if tear production is normal (sufficient to maintain

the integrity of the pre-corneal tear film), then there is no difference in corneal temperature as measured between blinks.

Tears may also cool the cornea by evaporation from the pre-corneal film. Mishima and Maurice (1961) have shown that the superficial oily layer of the pre-corneal film protects against evaporation from the middle fluid layer, its destruction in the rabbit increasing the rate of evaporation from $2.2\text{--}3.7\text{ }\mu\text{l./hr/cm}^2$ to $40\text{--}45\text{ }\mu\text{l./hr/cm}^2$ —up to a 20-fold increase. With the film intact this small quantity of evaporation cannot change the temperature to any great extent, but the effect must be a cooling one, evaporation abstracting the latent heat of vaporization of water. The rate of evaporation will also be affected by the humidity, but Schwartz (1965) in rabbits found this to be small—a 1 per cent. change in humidity producing a change in corneal temperature of 0.04°C .

The summated effect of tears is then firstly a cooling effect due to the evaporation of tears from the pre-corneal film, and secondly a heating effect due to the secretion of warm tears in the superior fornix and their subsequent passage across the front of the colder cornea. Again there is no reason to suppose that the effect of tears on corneal temperature will differ as between right and left corneae and, if tear production is normal, this factor alone can produce no difference in temperature between the two corneae of an individual. However, if tears are produced in excess, then irregular readings are obtained and differences of more than 0.5°C . occur between the two corneae.

The final factor affecting corneal temperature is the aqueous humour. From the direction of the convection current in the anterior chamber, it follows that the aqueous must be heating the endothelial surface of the cornea and its ability to do this depends on the temperature difference between the two; *e.g.*, if the cornea is heated the convection current ceases and there is no further net transfer of heat (Duke-Elder, 1932). The positive source of heat to the aqueous is the anterior uveal tract, either endogenous heat produced by local tissue metabolism, or exogenous metabolic heat convected by the vascular system, the temperature of the perfusing blood approaching that of the body core. The temperature of the aqueous therefore depends on the difference between the quantity of heat lost to the cornea and the quantity gained from metabolic activity in, and the vascular supply to, the anterior segment. As shown above, the effect of the environmental temperature on right and left corneal temperatures, and presumably the right and left aqueous humour, is the same, *i.e.*, no temperature difference is produced between the two; similar considerations apply to the lids and tears. Further, there is no reason to suppose that the quantity of metabolic heat produced in one anterior segment should differ greatly from that in the other—the muscles of the iris and ciliary body of both eyes contracting in unison, whilst the quantity of heat produced by the ciliary epithelium cannot be large. Thus, the only heat source to the aqueous that can vary as between right and left anterior segments is the convected heat of the perfusing blood. From this it can be deduced that any difference between the temperatures of the right and left corneae is largely a reflection of differing blood supplies to their respective anterior segments. Consequently, if the blood supply to one anterior segment is increased, then the temperature of the ipsilateral cornea would be higher than that of its fellow, whereas if it were decreased, then it should be lower. The results of measuring corneal temperature in anterior segment inflammation and carotid artery disease show that with an increased blood supply—inflammatory vasodilatation—the corneal temperature increases by up to 2.4°C . (Fig. 8) relative to the opposite cornea, whereas if the blood supply to the anterior segment is decreased, as in carotid artery disease,

then the temperature of the ipsilateral cornea may be as much as 1.3°C . less than that of the opposite cornea (Table IV). These differences are considerably greater than normal—(left minus right = $+0.29$ or -0.39°C . = mean $\pm$ two standard deviations, Mapstone, 1968b).

The indications are that, if a temperature difference greater than normal exists between the right and left corneae, then there is an asymmetry in blood supply to the two anterior segments. However, there is one further factor that may affect corneal temperature and this must be considered before the above statement can be regarded as valid; this is the influence of the metabolism and blood supply of the posterior segment. Lawson (1957), Lawson, Wlodek, and Webster (1961), and Lloyd Williams, Lloyd Williams, and Handley (1960, 1961) have shown that the temperature of the skin overlying a carcinoma of the breast is up to 5°C . higher than that of the corresponding contralateral area. Furthermore, Lloyd Williams and others (1961) have shown that the skin over a malignant melanoma is hot. From this, it would be reasonable to suppose that the cornea of an eye harbouring a malignant melanoma would be hotter than that of the contralateral normal eye, but the results in Table V indicate that this is not so. A neoplasm can produce an increase in temperature either by an increased local blood supply, thus bringing more heat to unit mass of tissue in unit time, or by increased local tumour tissue metabolism. In relation to this, Lawson and Gaston (1964) have shown that the temperature of blood draining a breast neoplasm may be up to 3°C . hotter than that of the arterial supply. Both these facts seem to contradict the corneal temperature measurements in eyes with malignant melanomata, however; increasing the blood supply to a tissue need not necessarily result in a local rise in temperature.

Fig. 10a represents a section of tissue exposed to the external environment, unit mass of tissue being represented by the area in the box (not stippled). Suppose the temperature of the perfusing blood to be T and that of the extravascular tissue t . At equilibrium the heat lost to the environment is balanced by endogenous metabolic heat and heat convected by the blood. Fig. 10b shows the same tissue exposed to the same environmental temperature, but here the blood supply is increased. The temperature of the perfusing blood

FIG. 10.—Effect of increasing blood supply on surface temperature.

is still T , but in unit mass of tissue there is a greater volume than previously. Now since t is less than T there must be a greater quantity of heat in unit mass, *i.e.*, the temperature is increased to t' . This represents conditions existing on, or near to, the surface of the body, for example in the skin or the iris.

Fig. 10c refers to a similar unit mass of tissue; here, however, it is not exposed to the ambient environment but is isolated in the surrounding body tissue. At equilibrium the temperature of the perfusing blood is T , as also is that of the extravascular tissue (there is no heat loss to the environment). Fig. 10d shows the same tissue but here the blood supply is increased, and it is obvious that no increase in temperature can occur since the blood has the same temperature as that of the extravascular tissue. These latter conditions apply to any tissue lying in a sheltered thermal environment, such as a neoplasm of the choroid. Consequently, it is to be expected—and Table V confirms this—that the increased blood supply to a vascular neoplasm would have little effect on corneal temperature. A corollary of this is that a reduction in blood supply to the posterior segment would likewise be expected to have little effect on corneal temperature.

There remains the problem of metabolic heat produced by tumour tissue; if this is to affect corneal temperature a gradient must be produced so that heat can flow anteriorly. However, the whole of the posterior segment of the eye is lined by retina, which has the highest rate of respiration of any tissue in the body and in common with embryonic tissue and tumour tissue can accumulate lactic acid both aerobically and anaerobically (Adler, 1965). This being so, no large temperature gradient can be produced by a malignant melanoma and no heat will flow; *i.e.*, corneal temperature will not be affected.

On the basis of the evidence detailed above it is concluded that, if a temperature difference greater than normal exists between the right and left corneae of an individual, the cause lies in the differing temperatures of their respective aqueous humours; this in turn is determined by differing blood supplies to their anterior segments. The lids, *normal* tear production, and the external environment cannot produce such an asymmetry, since both corneae are affected to the same degree by changes in these. Further, events in the posterior segment are unlikely to cause a temperature difference since this part of the eye provides a relatively stable thermal background to the anterior segment, with a temperature approaching that of the body core. If a significant temperature difference does exist then the cause is either an ischaemia of one anterior segment or a hyperaemia of the other.

Summary

The effect of the following factors on corneal temperature was investigated: environmental temperature, lids and blinking, tears and tearing, aqueous humour, anterior uveitis, carotid artery stenosis, and posterior segment neoplasm.

Evidence is adduced that if an abnormal temperature difference exists between the right and left corneae the cause must lie in an inequality in blood supply to the anterior segments. This may be due to an ischaemia of one or a hyperaemia of the other.

Events in the posterior segment of the eye do not affect corneal temperature to any measurable extent.

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REFERENCES

- ADLER, F. H. (1965). "Physiology of the Eye", 4th ed. Mosby, St. Louis.
- BRAENDSTRUP, J. M. (1952). Thesis cited by N. Ehlers (1965). "The Precorneal Film", *Acta ophthal. (Kbh.)*, Suppl. 81, p. 88.
- DUKE-ELDER, S. (1932). "Textbook of Ophthalmology", vol. 1, p. 457. Kimpton, London.
- HUBER, A. (1960). *Ophthalmologica (Basel)*, **139**, 351.
- LAWSON, R. (1957). *Canad. Services med. J.*, **13**, 517.
- and GASTON, J. P. (1964). *Ann. N.Y. Acad. Sci.*, **121**, 90.
- , WLODEK, G. D., and WEBSTER, D. R. (1961). *Canad. med. Ass. J.*, **84**, 1129.
- MAPSTONE, R. (1968a). *Exp. Eye Res.*, **7**, 237.
- (1968b). *Brit. J. Ophthal.*, **52**, 818.
- MISHIMA, S., and MAURICE, D. M. (1961). *Exp. Eye Res.*, **1**, 39.
- PLOMAN, K. G., ENGEL, A., and KNUTSSON, F. (1928). *Acta ophthal. (Kbh.)*, **6**, 55.
- SCHWARTZ, B. (1964). *Invest. Ophthal.*, **3**, 100.
- (1965). *Arch. Ophthal. (Chicago)*, **74**, 237.
- and FELLER, M. R. (1962). *Invest. Ophthal. (Chicago)*, **1**, 513.
- WILLIAMS, F. J. LLOYD, WILLIAMS, K. LLOYD, and HANDLEY, R. S. (1960). *Lancet*, **2**, 958.
- ZEISS, E. (1930). *Arch. Augenheilk.*, **102**, 523.