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The computation of temperature rises in the human eye induced by infrared radiation

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Abstract. Long-term industrial exposure to low levels of infrared radiation has for many years been associated with the development of cataracts; the injury mechanism is widely held to be thermal. A finite element model of the human eye is employed to calculate the temperature rises experienced by the intraocular media when exposed to infrared radiation. The model is used to calculate transient and steady-state temperature distributions for various exposure times and a range of incident irradiances. The effect of the eye's natural cooling mechanisms on the heating is investigated. Specific absorption rates in the infrared irradiated eye are presented. For a radiation source of 1500 °C, absorption of radiant energy by the iris and the lens combined with conduction of heat from the anterior regions is found to be responsible for increases in the lens temperature of 1-2 °C, but under extreme exposure conditions the temperature rises are found to be substantially higher.

1. Introduction

The mechanism of infrared cataractogenesis is not clearly formulated. Historically, infrared cataracts (also known as glass blowers' or furnaceman's cataracts) appear to have resulted from exposure to infrared radiation. Reports in the early part of this century (Robinson 1903, 1907, Legge 1907) suggested that prolonged exposure (often over a period of years) to levels of infrared radiation exceeding environmental levels led to a prevalence of cataracts among glass workers. This resulted in radiation cataract being scheduled as an industrial disease in the United Kingdom under the 1908 Workmen's Compensation Acts for workers exposed to the glare of molten glass. In 1921 the work of Healy caused the Act to be extended to include cataract due to the glare of, or by rays from, molten or red-hot metal.

The ocular media absorb an increasing amount of the radiant energy incident upon the cornea for increasing wavelengths in the near infrared (760-1400 nm). At wavelengths in excess of 1400 nm water absorbs heavily and the cornea and aqueous humour absorb all incident radiation; beyond 2500 nm the cornea is effectively the sole absorber (Boettner and Wolter 1962). The energy absorbed by one tissue raises the temperature of that tissue, and, by conduction, those of the other ocular tissues. The heating by conduction and the direct absorption of near-infrared radiation by the interior structures is thought to play a role in the development of some opacities of the lens.

Differing theories have been put forward for the aetiology of infrared cataracts. There are two widely held conventional theories that advocate a thermal injury mechanism. The theory due to Vogt (1919) holds that the direct absorption of radiant energy by the lens leads to the formation of the cataract. The opposing theory (Goldmann

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1933a, b) argues that the radiant energy is absorbed in the iris and converted into heat which is then conducted to the adjacent epithelium of the lens. In recent years the debate has continued (Wolbarsht 1980, Pitts and Cullen 1981) and the development of infrared cataract remains a subject of controversy (Wolbarsht 1982, Waxler and Hitchins 1986).

The objective of the current study was the development and employment of a thermal mathematical model of the human eye to calculate the temperature distribution in the eye both before and as a result of exposure to infrared radiation. A detailed knowledge of the temperature changes in the eye resulting from exposure to infrared radiation is an important input to discussions about safety standards for exposure to infrared radiation. The model that is used in this paper was presented in the preceding paper (Scott 1988, hereafter referred to as I), which described the application of finite element techniques in the calculation of steady-state and transient temperature distributions in the human eye, and presented results for the normal (unexposed) human eye. In the present paper the results of applying the model to the infrared irradiated eye under a range of exposure conditions are reported. The computed temperatures given in this paper may be combined with those in I to determine the temperature rises attributable to exposure to infrared radiation. Values of the specific absorption rate within the eye when exposed to infrared radiation are also presented.

2. The heat transfer model

The thermal mathematical model of the human eye employed in this study is described in I. The parameters and constants required in the model are assigned the values which were chosen as control parameters in I, where the importance of uncertainties and discrepancies in these control values was also considered.

In the model of the normal unexposed human eye the eye was divided into six regions and it was assumed that no heat was generated within the eyeball. When the eye is exposed to infrared radiation the internal heat source term in the bioheat transfer equation will be non-zero. Assuming the radiating source is a black body the heat source H ($\mathrm{W\,m^{-3}}$) at a point (r, z) belonging to region I ($1 \leq I \leq 6$) of the eye is of the form

$$H(r, z) = \int_{\lambda_{\min}}^{\lambda_{\max}} f(r) S_I(r, z; \lambda) \mu_I(\lambda) \exp(-\mu_I(\lambda) x)\, \mathrm{d}\lambda \tag{2.1}$$

where $f(r)$ is the focusing factor, $\mu_I(\lambda)$ is the absorption coefficient for ocular tissue I, $\lambda_{\min}$, $\lambda_{\max}$ are the minimum and maximum wavelengths of the source (m), x is the distance travelled by the radiation in the tissue I (m) and $S_I(r, z; \lambda)$ is the irradiance of the point (r_I, z_I) where the radiation passing through (r, z) intersects the anterior boundary of tissue I ($\mathrm{W\,m^{-3}}$).

The irradiance $S_I(r, z; \lambda)$ is calculated using the equation

$$S_I(r, z; \lambda) = (P/\sigma T_B^4) E(\lambda) \tilde{S}_I(r, z; \lambda) \tag{2.2}$$

where P is the irradiance incident on the eye ($\mathrm{W\,m^{-2}}$), T_B is the temperature of the black body source (K), σ is Stefan's constant ($\mathrm{J\,m^{-2}\,K^{-4}}$), $E(\lambda) = c_1 \lambda^{-5} [\exp(c_2/\lambda T) - 1]^{-1}$, with c_1 and c_2 Planck's radiation constants, and $\tilde{S}_I(r, z; \lambda)$ is the fraction of radiation incident on the cornea reaching the point (r_I, z_I).

The heat source $H(r, z)$ may be time dependent. For example, the effect on the temperature distribution of exposure to the radiating source for a prescribed interval

of time t^* may be required. In this case $H(r, z, t)$ is given by equation (2.1) for $0 < t \leq t^*$, and $H(r, z, t) = 0$ for $t > t^*$.

3. Evaluating the heat source

To evaluate the heat source H at a point (r, z) within the eye when exposed to infrared radiation factors such as the pupil size, the eyelid shielding, the image size, the transmission properties of the ocular media, the minimum and maximum wavelengths of the radiation emitted by the source, together with the source temperature and the irradiance incident on the eye must be known for inclusion in equation (2.1). These factors are considered separately.

3.1. Pupil size

For wavelengths less than 700 nm the iris absorbs more than 80% of the radiation incident upon it (Wolbarsht 1980). For these wavelengths the radiant energy transmitted to the posterior regions of the eye is proportional to the area of the pupil. For the normal dark-adapted eye, the pupil diameter ranges from approximately 7 to 8 mm; for outdoor daylight the pupil diameter contracts to about 1.6 mm (Sliney and Wolbarsht 1980). A value of 3.2 mm is employed as the control pupil diameter.

3.2. Eyelid shielding

The eyelids afford protection to the eyes. The heat source is corrected for shielding effects by excluding the contribution made by radiation passing through the eyelids. The further closed the eyelids are the lower the value of the heat source within the irradiated eye. The control eyelid aperture is taken to have a diameter of 12 mm.

Under normal conditions the human (male) eye blinks on average once every 2.8 s and is closed for 0.3 s, or approximately 10.7% of waking time (Adler 1970). The time taken for the steady-state temperature distribution to be reached is of the order of minutes (see § 5), therefore blinking should be taken into account when computing transient and steady-state solutions in the eye when exposed to infrared radiation. A given blinking rate can be incorporated into the mathematical model by reducing the intensity of the radiation incident on the anterior corneal surface by a corresponding amount. It is anticipated that when the eye is exposed to infrared radiation the blinking rate will increase above the normal rate to protect the eye, but precise data is unavailable. Consequently the normal blinking rate of 10.7% will be used as the control blinking rate; in § 4.2 the effect of an increase in the blinking rate is considered. When the eyelids are fully or partially closed the blood flow in the eyelids assists in the cooling of the irradiated eye. The eyelid blood flow is not included in the model because of the lack of reliable data.

3.3. Image size

Typical retina image sizes for a variety of conventional optical sources have been tabulated by Sliney and Wolbarsht (1980, p 135). A control image size of 1 mm is employed in the eye model. This value is appropriate for the image of a blast furnace opening and may be varied with each radiating source under consideration.

3.4. Transmission data

The transmission properties of the cornea, aqueous humour, lens and vitreous humour of radiation of wavelengths in the range 300–2500 nm are taken from the study by Boettner and Wolter (1962). Beyond 2500 nm effectively all incident radiation is absorbed by the cornea. For radiation of wavelength exceeding 2500 nm the cornea is assumed to have the same absorption properties as water; absorption coefficients are calculated using the optical constants of water presented by Rusk *et al* (1971). In the range 300–1000 nm absorption of infrared radiation by the iris is high; beyond wavelengths of about 1000 nm the iris absorbs only slightly. Absorption data for the iris are taken from Wolbarsht (1980).

The total loss by reflection and scattering is assumed to be 5% (Hartridge and Hill 1915).

3.5. The source temperature

For workers in different occupations who are exposed to infrared radiation the temperature of the radiating source will vary. These sources include molten glass and metal. Keatinge *et al* (1955) provide some estimates of the molten-metal temperatures to which iron workers might be exposed. These lie in the range 1250–1500 °C. Langley *et al* (1960) state that in a study of furnaces to which glass blowers were exposed the temperature in the furnace was approximately 1500 °C. A control source temperature of 1500 °C is used in the eye model.

3.6. Irradiance incident on the eye

The investigations performed by Dunn (1950) and Keatinge *et al* (1955) into the incidence of infrared cataract in industrial workers in the 1950s included measurements of the irradiance incident upon the eyes of workers in the glass industry and in iron rolling mills. For glass workers, irradiances at eye level in the approximate range 140–1700 $W\,m^{-2}$ were recorded, depending on the particular task being performed. Much higher irradiances from 2000 to around 6000 $W\,m^{-2}$ were recorded for iron mill workers. Values of irradiance within these ranges are used as control parameters.

3.7. Minimum and maximum wavelengths

The equation (2.1) for $H(r, z)$ requires λ_{min} and λ_{max} to be specified. At the lower end of the spectrum the thermal contributions of visible and near-ultraviolet radiation are included by assigning $\lambda_{min} = 300$ nm; including wavelengths below 300 nm has an insignificant effect on the heat source (2.1). The absorption by the cornea of all wavelengths exceeding 2500 nm is known to lead to sharp increases in the corneal temperature which can be sufficient to cause corneal burns. However, corneal damage from infrared radiation is rarely seen from industrial exposures since levels of radiation high enough to inflict damage give immediate and severe corneal pain which quickly activates the eye's aversion responses. The development of infrared cataracts is commonly attributed to exposure to near-infrared radiation. For the control source temperature of 1500 °C the wavelength corresponding to the maximum radiation intensity is $\lambda \simeq 1630$ nm. In the mathematical model it is sufficient to choose $\lambda_{max} = 12\ \mu m$ since contributions to the heat source term (2.1) from wavelengths beyond 12 μm are negligible.

4. The steady-state temperature distribution

The results which are presented in the following sections were computed using the NAG Finite Element Library on a grid of 456 four-noded isoparametric quadrilateral elements, with a total of 496 nodes (see I). The grid was designed to have a large number of elements in the anterior regions of the eye where the changes in temperature are expected to be most rapid and of the largest magnitude.

The integral (2.1) was discretised using the composite trapezoidal rule with a wavelength interval $\delta\lambda$ of 25 nm.

The temperature distribution within the eye is illustrated by the temperature profile along the axis of symmetry. The axis of symmetry was chosen since it is least influenced by the eye's cooling mechanisms. The association between exposure to infrared radiation and the formation of cataracts implies that it is of interest to consider the temperature elevations experienced in the eye at the anterior and posterior poles of the lens and at a point on the posterior surface of the iris in contact with the lens; these points are denoted AP, PP and IR, respectively. The calculated temperatures at these points are tabulated and, in the case of transient solutions, plotted against time.

4.1. Results for a range of incident irradiances

Using the control parameters discussed in § 3 the steady-state temperature distribution is calculated for the incident irradiances $P = 280$, 1400 and 1700 W m^{-2}. These values correspond to the exposure reported by Dunn (1950) of a glass ball maker, the average exposure received by a glass worker and the exposure of a glass finisher, respectively. In addition, using the data of Keatinge *et al* (1955), the steady-state temperatures are calculated for an 'iron rougher' (a worker on an iron roughing mill) by assigning $P = 2100$ W m^{-2}. The temperature profiles along the axis of symmetry are plotted in figure 1, and the computed temperatures at the points AP, PP and IR are tabulated in table 1.

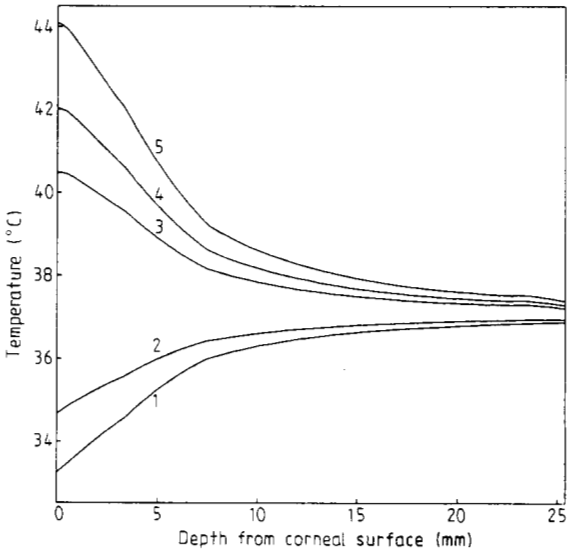

Figure 1. The steady-state temperature profiles for different occupations (1: normal eye; 2: ball maker; 3: average glass worker; 4: glass finisher; 5: iron rougher).

Table 1. The steady-state temperatures for a range of incident infrared irradiances.

Incident irradiance ($W m^{-2}$)	AP (°C)	PP (°C)	IR (°C)
0	34.6	36.0	35.2
280	35.6	36.4	35.9
1400	39.6	38.1	38.8
1700	40.6	38.6	39.6
2100	42.0	39.2	40.6

The theory of cataractogenesis forwarded by Vogt (1919) holds that the direct absorption of infrared radiation by the lens results in the development of cataracts, whereas that of Goldman (1933a, b) argues that the absorption of radiant energy by the iris combined with conduction of heat to the lens is responsible. It is therefore of interest to consider the amount of energy absorbed by the different structures of the eye. The specific absorption rate (SAR) is defined to be

$$\text{SAR} = H/\rho \ (\text{W kg}^{-1}).$$

Calculated values of the SAR are given in table 2. The SAR has a maximum value at the mid anterior corneal surface and then decreases along the pupillary axis through the cornea, aqueous humour, lens and vitreous humour. The heavy absorption by the iris of incident radiation of wavelengths less than 1000 nm is reflected in the high values of the SAR in the iris compared with those in the lens.

4.2. The effects of the eye's cooling mechanisms

In calculating the steady-state temperatures the parameters used in the mathematical model were assumed to be constant. In reality, as the temperatures within the eye increase, the eye will respond with protective cooling mechanisms, such as an increase in the blood flow rate, more frequent blinking and a higher evaporation rate. The oily layer of the precorneal tear film retards evaporation from the eye and if destroyed the rate of evaporation may increase by a factor of ten (Mishima and Maurice 1961). The cooling of the eye may be assisted further by a more rapid blinking rate. The control blinking rate may increase by a factor of three (Adler 1970). To give an indication of the effectiveness of these cooling mechanisms the steady-state temperature profiles for

Table 2. Calculated specific absorption rates (SAR) for a range of incident infrared irradiances (AC: mid anterior corneal surface; PC: posterior corneal surface; AP: anterior pole of lens; PP: posterior pole of lens; IR: posterior surface of iris; RT: intersection of pupillary axis and retina).

Incident irradiance ($W m^{-2}$)	AC ($\times 10^5$ W kg^{-1})	PC ($\times 10^2$ W kg^{-1})	AP ($\times 10$ W kg^{-1})	PP (W kg^{-1})	IR ($\times 10^2$ W kg^{-1})	RT (W kg^{-1})
280	0.214	0.435	0.39	0.878	0.446	0.236
1400	1.07	2.17	1.95	4.39	2.23	1.18
1700	1.30	2.64	2.37	5.33	2.70	1.44
2100	1.60	3.26	2.92	6.59	3.34	1.77

an iron rougher ($P = 2100\ \mathrm{W\ m^{-2}}$) with an evaporation rate of $320\ \mathrm{W\ m^{-2}}$ (calculated using data from Mishima and Maurice) and with a blinking rate of 20% are presented in figure 2.

Coates and Keatinge (1955) in their investigation into the incidence of lens opacities in workers in an iron rolling mill reported that although few of the workers at that time wore protection against the heat, most of them were observed to screw their eyelids up, thus reducing the corneal area exposed to the radiant energy. In figure 3

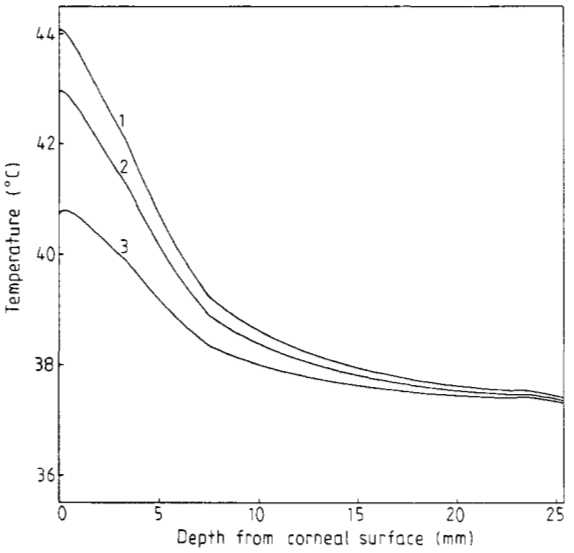

Figure 2. The effect of cooling mechanisms on the steady-state temperature profile of an iron rougher (1: control values; 2: increased blinking; 3: increased evaporation).

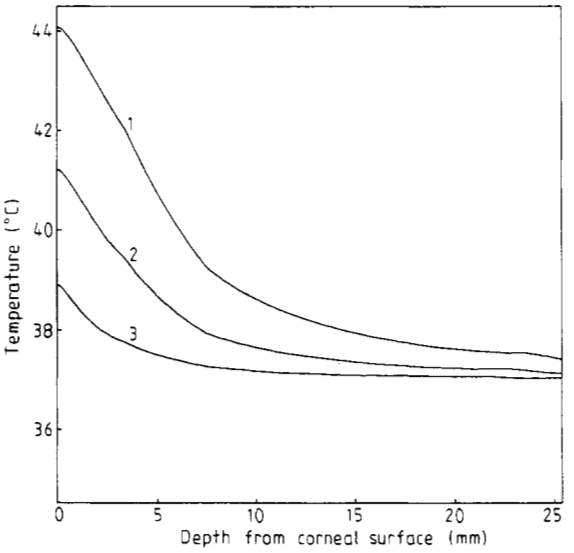

Figure 3. The effect of partial eyelid closure on the steady-state temperature profile of an iron rougher (1: eyelids fully open (eyelid aperture diameter 12 mm); 2: eyelid aperture diameter 6.5 mm; 3: eyelid aperture diameter 3.5 mm).

the temperature profiles are given for an iron rougher with eyelid aperture diameters of 12 (the control value), 6.5 and 3.5 mm. The corresponding temperatures at points AP, PP and IR are given in table 3. As the eyelids are progressively closed the central anterior to posterior ocular temperature gradient and the central to peripheral ocular temperature gradient are gradually reduced, and the temperature throughout the eye tends to the body-core temperature of 37 °C. With the eyelid aperture diameter of 3.5 mm and the control pupil diameter of 3.2 mm, very little radiation is directly incident on the iris. In this case it is seen that only the temperature in the anterior regions rises significantly above 37 °C and the temperature rises in the lens are substantially reduced compared with those for the unshielded eye. Note that if blood flow in the eyelids was incorporated into the mathematical model of the irradiated eye this would tend to further reduce the temperatures in the cornea and aqueous humour upon partial closure of the eyelids.

Table 3. The steady-state temperatures for an iron rougher with partially closed eyelids.

Eyelid aperture diameter (mm)	AP (°C)	PP (°C)	IR (°C)
12	42.0	39.2	40.6
6.5	39.4	37.9	38.4
3.5	37.8	37.3	37.4

4.3. The effect of blood flow

One deficiency of the existing mathematical model of the eye is the simplifying assumption made concerning blood flow. It is assumed that blood flow is concentrated in the sclera/choroid/retina region; separate blood flows in the iris and ciliary body are not included in the model. At present it is not clear how to incorporate the blood flow in the iris and ciliary body within the model (Lagendijk 1982, I). It was observed in I that the temperature distribution in the anterior regions of the eye is sensitive to uncertainties in the convective heat transfer coefficient h_s from the sclera to the surrounding body-core temperature. The coefficient h_s is dependent upon the blood flow rate in the sclera and it is anticipated that when the eye is exposed to infrared radiation this blood flow will provide effective cooling. A rise in temperature may result in an increase in the blood flow rate, which in turn would lead to an increase in the value of h_s. In an attempt to consider the effects of uncertainties in h_s and variations in the blood flow rate on the temperature distribution in the exposed eye, the steady-state temperatures for an irradiance $P = 2100\ \mathrm{W\,m^{-2}}$ have been calculated for the control value $h_s = 65\ \mathrm{W\,m^{-2}\,°C^{-1}}$ (Lagendijk 1982) and increased values $h_s = 90$ and $110\ \mathrm{W\,m^{-2}\,°C^{-1}}$. The results are presented in table 4. It is found that with this range of values for h_s the maximum variation in the temperature in the anterior regions is approximately 0.45 °C, while in the vitreous humour the maximum variation is less than 0.2 °C. These variations are greater than the corresponding variations of 0.25 and 0.1 °C, respectively, found for the unexposed eye but nevertheless may be regarded as being relatively small compared with the rises in temperature resulting from the exposure to infrared radiation.

Table 4. The effect of variations in the convective heat transfer coefficient on the steady-state temperature distribution with $P = 2100\ \mathrm{W\ m^{-2}}$.

h_s ($\mathrm{W\ m^{-2}\ °C^{-1}}$)	AP (°C)	PP (°C)	IR (°C)
65 (control)	42.05	39.22	40.58
90	41.79	38.99	40.30
110	41.65	38.87	40.15

5. The transient response

The steady-state temperature calculations provide information on the maximum temperatures experienced within the eye when exposed to a constant source of infrared radiation over a length of time sufficient for temperature equilibrium to be established. In practice, the steady-state temperatures may not give a realistic picture of the temperature distribution within the eye of a worker in an industrial environment since in general the worker will only be exposed intermittently. For example, at the beginning of the century it was reported that a 'finisher' in a glass factory would spend approximately 3 s looking directly into the glass furnace while warming his bottle and would then turn so that his back was towards the furance and spend about 21 s fixing the rim (Robinson 1903). The exposure of such a worker is constantly changing and modern working practices have probably been aimed at reducing the length of direct exposure time, as well as the levels of irradiance received by workers. To assess duration of exposure and the amount of infrared radiation to which workers today are exposed a series of time and motion studies would be needed. Nevertheless, it is informative to consider how quickly the temperature in the irradiated eye is elevated so as to determine the time interval which elapses before the steady state is achieved and the time taken for the eye to cool down to the normal unexposed state once exposure has ceased.

5.1. Exposure for 30 min

In figure 4 the temperatures at chosen points within the eye are plotted for an exposure time of 30 min with an incident irradiance $P = 1250\ \mathrm{W\ m^{-2}}$, followed by a cooling period ($P = 0$) of 30 min. It is observed that when the eye is irradiated the temperature in the anterior structures rises rapidly over the first 5-6 min; the rate of temperature increase then slows down and the steady state is achieved after approximately 20 min of exposure. Similarly, when the eye ceases to be irradiated at time $t = 30$ min, the temperatures initially fall sharply, with the normal temperature distribution restored after about 20 min.

5.2. Exposure for 30 s

With $P = 1250\ \mathrm{W\ m^{-2}}$ the steady state was reached after 20 min of exposure but, as noted previously, workers are unlikely to be exposed constantly for such long periods. It is therefore of interest to consider the thermal effects of a much shorter exposure, followed by a cooling period. In figure 5 the temperatures are plotted for an exposure of 30 s with $P = 5000\ \mathrm{W\ m^{-2}}$, followed by a cooling period of 150 s. After exposure ceases the temperature on the corneal surface falls rapidly but the temperatures of the remaining eye structures initially continue to rise as heat is conducted from the cornea into the interior of the eyeball. For a 30 s exposure with $P = 5000\ \mathrm{W\ m^{-2}}$ the maximum

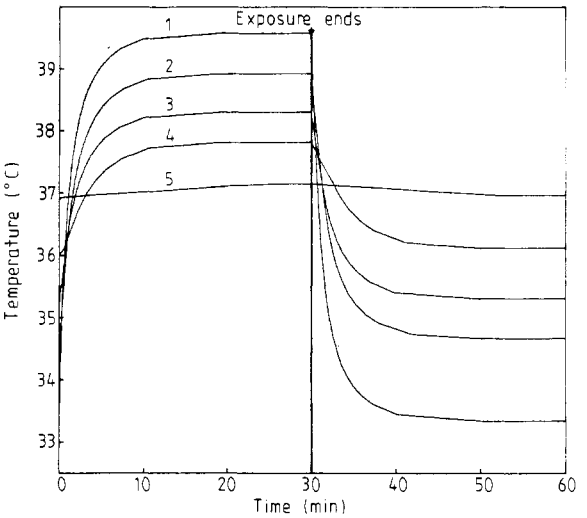

Figure 4. The transient temperatures within the eye for an exposure of 30 min with $P = 1250\ \mathrm{W\,m^{-2}}$ and a cooling period of 30 min. (1: mid corneal surface; 2: anterior pole of lens; 3: posterior surface of iris; 4: posterior pole of lens; 5: retina).

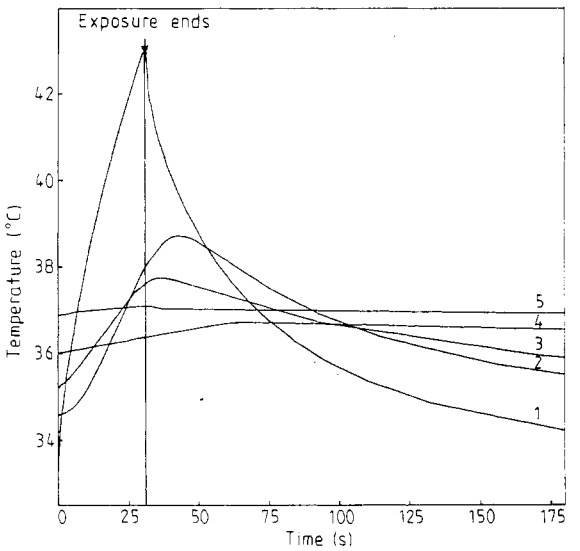

Figure 5. The transient temperatures within the eye for an exposure of 30 s with $P = 5000\ \mathrm{W\,m^{-2}}$ (1: mid corneal surface; 2: anterior pole of lens; 3: posterior surface of iris; 4: posterior pole of lens; 5: retina).

temperatures at the points AP, PP and IR and the times after exposure ceases at which they are achieved are given in table 5. It was found that after 20 min of cooling following the 30 s exposure the calculated intraocular temperature distribution was within 0.01 °C of the steady-state temperature distribution of the normal eye.

5.3. The effect of the body-core temperature

It was observed in I that for a normal unexposed eye the intraocular temperature distribution is sensitive to the body-core temperature. Workers who are exposed to

Table 5. The maximum temperatures and the times at which they are achieved following an exposure of 5000 W m^{-2} for 30 s (figures in parentheses are the normal eye temperatures).

Point in eye	Maximum temperature (°C)	Time (s)
AP	38.7 (34.6)	13
PP	36.8 (36.0)	55
IR	37.7 (35.2)	6

infrared radiation are likely to be performing work that causes their body-core temperature to rise above the control value of 37 °C. To investigate the effect on the temperatures in the irradiated eye of an elevation in the body-core temperature let ΔT represent the difference between the temperature in the normal eye and the exposed eye at time t for some given incident irradiance P. With the simplifying assumptions concerning blood flow and heat loss from the eye that have been made in the present thermal eye model, it can be shown that provided the body-core temperature remains the same during exposure as it was prior to exposure, ΔT satisfies a differential equation which does not depend directly upon the body-core temperature. Therefore, if the body-core temperature is constant throughout, the calculated temperature rises in the eye are independent of the body-core temperature. It follows that temperatures in the exposed eye are as sensitive to changes in the body-core temperature as those in the normal eye.

5.4. The effect of the ambient temperature

The temperature distribution in the anterior structures of the normal human eye is sensitive to changes in the ambient temperature since the heat loss from the anterior corneal surface is dependent on the surrounding room temperature (see I). In an industrial environment such as a glass factory or iron rolling mill the ambient temperature is likely to exceed the control value of 20 °C employed in the present study. Moreover, the ambient temperature may be higher during exposure, for example, when the door of a blast furnace is open. Let T_1 and T_2 denote respectively the temperatures at time t for the normal and the irradiated eye (with the irradiance P given), and let $T_{amb}(I)$ and $T_{amb}(F)$ denote the ambient temperatures before and during exposure, respectively. Defining $\Delta T = T_2 - T_1$ it can be shown that ΔT satisfies a differential equation in which the ambient temperatures appear only in the corneal boundary condition

$$-k\partial\Delta T/\partial n = h_c\Delta T + \sigma\varepsilon(T_2^4 - T_{amb}(F)^4) - \sigma\varepsilon(T_1^4 - T_{amb}(I)^4).$$

The radiation heat transfer coefficient is defined as

$$h_r(T;\, T_{amb}) = \sigma\varepsilon(T^2 + T_{amb}^2)(T + T_{amb})$$

and over the range of ambient and tissue temperatures of interest $h_r\ (T;\, T_{amb})$ is approximately constant, i.e. $h_r(T;\, T_{amb}) \simeq h_r$. With this approximation the corneal boundary condition reduces to

$$-k\partial\Delta T/\partial n = (h_c + h_r)\Delta T + h_r(T_{amb}(I) - T_{amb}(F)). \tag{5.1}$$

If $T_{amb}(I) = T_{amb}(F)$ then it follows that the temperature elevations experienced in the eye when exposed to infrared radiation are independent of the ambient temperature.

However, if $T_{amb}(F) > T_{amb}(I)$ then the second term on the right-hand side of equation (5.1) is negative, which implies a reduction in the amount of heat lost by radiation from the corneal boundary and a corresponding increase in temperature in the anterior structures.

In table 6 the calculated temperatures for an exposure of 30 s with $P = 1250\ W\ m^{-2}$ are presented for the following ambient temperatures:

(i) $T_{amb}(I) = T_{amb}(F) = 20\ °C$
(ii) $T_{amb}(I) = T_{amb}(F) = 30\ °C$
(iii) $T_{amb}(I) = 20\ °C$, $T_{amb}(F) = 30\ °C$.

Comparing the results of (i) and (ii) confirms that the temperature rises within the eye are independent of the ambient temperature, provided the ambient temperature is constant throughout.

Table 6. The effect of the ambient temperature on the temperature rises experienced in the eye for an incident irradiance $P = 1250\ W\ m^{-2}$. (Figures in parentheses are the temperature rises.)

Exposure time (s)	T_{amb} (°C)	AP (°C)	PP (°C)	IR (°C)
0	20	34.57	36.01	35.20
30	20	35.41 (0.84)	36.10 (0.09)	35.81 (0.61)
0	30	35.80	36.51	36.11
30	30	36.64 (0.84)	36.60 (0.09)	36.73 (0.62)
0	20	34.57	36.01	35.20
30	30	35.61 (1.04)	36.11 (0.10)	35.98 (0.78)

5.5. Comparison with experimental data

A major difficulty in attempting to validate a thermal model of the human eye is the lack of experimental data available with which to compare the computed results. Data for 30 s exposure with incident irradiances of 1250 and 5000 $W\ m^{-2}$ were given by Goldmann (see Coates and Keatinge 1955); the data agree closely with the calculated temperatures. Goldmann reported that with a blast furnace temperature of 1460 °C and incident irradiance of 1250 $W\ m^{-2}$ the rise in temperature in the anterior chamber was 3 °C after 30 s exposure, while for an irradiance of 5000 $W\ m^{-2}$ the corresponding elevation exceeded 9 °C. The temperature rises on the mid corneal surface obtained using the finite element model are 2.5 and 9.7 °C for a 30 s exposure with $P = 1250$ and 5000 $W\ m^{-2}$, respectively.

6. The need for further data

The presented results are an attempt to quantify the temperature rises experienced by industrial workers exposed to infrared radiation. A lack of knowledge of how the eye responds to heating means that there are uncertainties in the computed results. In particular, it is difficult to assess the significance of the conduction of heat from the corneal surface on the lens temperature because of the uncertainties that exist in the

response of the cornea's cooling mechanisms to changes in the corneal temperature. Calculations have shown that increased evaporation from the anterior corneal surface combined with a more rapid blinking rate can produce substantial deviations in the temperature distribution in the anterior structures of the eye. In addition, partial closure of the eyelids reduces the area of the irradiated eye and limits temperature changes. A more sophisticated model would incorporate temperature-dependent parameters, but a lack of data available on the parameters prevents such refinements from being built into the present model.

A further difficulty is that of incorporating into the mathematical model separate blood flows in the iris and ciliary body. A sensitivity analysis has demonstrated that while the calculated temperatures are dependent on the heat transfer coefficient h_s from the sclera to the surrounding body core, the effect of uncertainties in h_s is relatively small compared with the magnitude of the temperature increases induced by exposure to infrared radiation. However, once the temperature in the irradiated iris exceeds 37 °C blood flow in the iris will assist in conducting heat away from the iris. Reliable data on human iris and ciliary body blood flows are needed before this effect can be examined.

The length of exposure time as well as the intensity of the incident radiation was found to be an important factor in determining the magnitude of intraocular temperature rises. For short exposure times the lens continued to heat up after exposure ceased as heat was conducted from the anterior tissues. The time that elapsed before the normal (unexposed) eye temperature distribution was restored was of the order of minutes. The exposure of an industrial worker will in general involve a repeated cycle of exposure followed by cooling. Depending on the exposure and cooling times and the incident irradiance, the eye may not cool down fully between successive exposures. As a result a detailed study of modern working practices and conditions is required before the temperature changes within the eye which are likely to be experienced today in an industrial environment can be accurately determined.

7. Conclusion

A thermal model of the human eye has been employed to determine changes in the intraocular temperature distribution resulting from exposure to infrared radiation. A range of different exposure conditions has been considered. With the control model parameters the calculated elevations in the lens temperature for short exposure times (steady state not achieved) were in the range 1–2 °C, with the highest lens temperature increases being in the anterior portion. These temperature rises of 1–2 °C are no more than occur in a normal eye when the body-core temperature and ambient temperature are raised significantly above the control levels of 37 and 20 °C, respectively (see I). However, if increased body-core and ambient temperatures are combined with an intense corneal irradiance while the eyelids are fully open, then the calculated temperature rises at the posterior pole of the lens exceeded 3 °C, with the temperature increases in the front of the lens being substantially higher.

At present it is unclear whether the important factor leading to the development of infrared cataracts is a temperature rise in the iris or in the lens, or both. Nor is it clear whether a brief very high temperature rise caused by a combination of extreme exposure conditions, or a more modest temperature rise experienced for a longer time (or a combination of the two), is significant in cataract formation. Without this knowledge it is not possible to conclude that the computed results support either of

the conventional theories due to Goldmann and Vogt. Nevertheless, the results obtained using the mathematical model demonstrate how quickly different parts of the infrared irradiated eye heat up and how long they take to return to their normal temperature distributions once exposure to infrared radiation has ceased. In addition, the results that were obtained for the partial closure of the eyelids (which shield the iris) go some way towards quantifying the extent to which lens temperature rises are caused by direct absorption of infrared radiation by the lens and how much they are due to the conduction of heat from the iris and anterior regions of the eye.

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Résumé

Calcul de l'élévation de température provoquée dans l'oeil humain par une irradiation infrarouge.

On a pendant trés longtemps associé l'exposition industrielle chronique à de faibles niveaux d'irradiation en infrarouge au développement de cataractes; on admet classiquement que le mécanisme de production des lésions est de nature thermique. Un modèle de l'oeil humain reposant sur le principe des éléments finis est utilisé par l'auteur pour calculer les élévations de température dans les milieux intraoculaires exposés au rayonnement infrarouge. Ce modèle permet de calculer les distributions de température transitoires, ou à l'équilibre, pour diverses durées d'exposition et une gamme d'éclairements énergétiques. L'auteur a étudié l'effet des mécanismes naturels de refroidissement de l'oeil sur l'élévation de température. Des valueurs de l'absorption spécifique dans l'oeil irradié aux infrarouges sont également présentées. Avec une source de rayonnement à 1500 °C, l'absorption d'énergie radiante par l'iris et le cristallin, combinée à la conduction de la chaleur provenant des régions antérieures, paraît responsable d'élévations de température du cristallin de l'ordre de 1 à 2 °C, mais dans des conditions d'exposition extrêmes, les élévations de température peuvent être notablement supérieures.

Zusammenfassung

Die Berechnung des Temperaturanstieges im menschlichen Auge durch Infrarot-Bestrahlung.

Lange Zeit dachte man, daß die Langzeit-Exposition des Auges mit niedrigen Dosen Infrarotstrahlung in der Industrie zur Entwicklung von Katarakten führt, wobei als Schädigungsmechanismus thermische Effekte angenommen wurden. Ein Modell des menschlichen Auges, bestehend aus endlich vielen Elementen, wurde verwendet zur Berechnung des Temperaturanstieges im intraokularen Medium bei Infrarotbestrahlung. Das Modell wurde verwendet zur Berechnung transienter und Gleichgewichtstemperaturverteilungen bei unterschiedlicher Expositionsdauer und verschiedenen Strahlungsdichten. Der Einfluß des natürlichen Kühlmechanismus des Auges auf die Erwärmung wurde untersucht. Spezifische Absorptionsraten im infrarot-bestrahlten Auge werden Angegeben. Für eine Strahlungsquelle von 1500 °C zeigte sich, daß die Absorption der Strahlungsenergie durch die Iris und die Linse zusammen mit der Wärneleitung aus den vorderen Bereichen des Auges für einen Temperaturanstieg in der Linse von 1-2 °C verantwortlich ist. Es stellte sich allerdings heraus, daß unter extremen Bedingungen die Temperaturanstiege wesentlich höher sind.

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