Eye (1991) **5,** 181–185

Temperature Modulating Action of Choroidal Blood Flow

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Summary

A major physiologic role for the high flow choroidal vasculature is to help maintain a stable temperature in the macula. The choroidal vasculature modulates tissue temperature in the macula via both active and passive mechanisms. The active mechanisms involve a reflexive increase in choroidal blood flow in response to light. The neuro-anatomical pathways mediating this reflexive mechanism have been demonstrated to involve the supra-chiasmatic and the Edinger-Westphal nuclei.

The importance of the thermal environment for the retina has been neglected in looking for causes of retinal disease. The observations on the ability of the choroidal circulation to modulate the thermal environment of the macula should excite further study of the role of temperature and the ability of the eye to dissipate light-generated heat on macula disease.

The major component of ocular blood flow is the choroidal circulation. It accounts for 85% of all ocular blood flow.[1] Per gram of tissue, the choroid has four times the volume of blood flow found in the renal cortex.[1] The choroid is structured so that a dense network of small blood vessels with a large surface area, ostensibly for the exchange of oxygen and other nutrients, is immediately adjacent to the outer retinal layers. Unlike other vascular beds however, the oxygen content of venous blood in the choroid is unusually high, about 95% of that found in the arterial blood.[2] The small amounts of oxygen extracted from blood as it flows through the choroid thus suggests a role for the choroidal circulation in addition to that of providing oxygen and other nutrients to the outer retinal layers.[3]

All biological systems are sensitive to the temperature environments in which they function. The eye's visual activity, of necessity, exposes the retina to a thermally labile environment. Between 25% and 33% of all light entering the eye is absorbed by melanin in the retinal pigment epithelium and choroid.[4] The conversion of a portion of this light energy to heat, as in the case of photocoagulation, can produce temperatures capable of coagulating proteins. A major physiologic role for the large volumes of blood flowing through the choroid is to help maintain a stable temperature environment for the retina.[3]

The temperature-modulating action of the choroidal circulation was demonstrated in a series of experiments in the cynomolgus monkey eye.[3] A thermistor probe mounted inside the tip of a 23-gauge needle was inserted through the pars plana and positioned under direct observation in the macula or a peripheral retinal site. Choroidal blood flow was varied by altering intraocular pressure via a cannula inserted into the anterior chamber and connected to a reservoir of saline. Temperature measurements were made with the eye exposed to ambient room illumination or a moderate intensity light source.

Under most circumstances, the choroid

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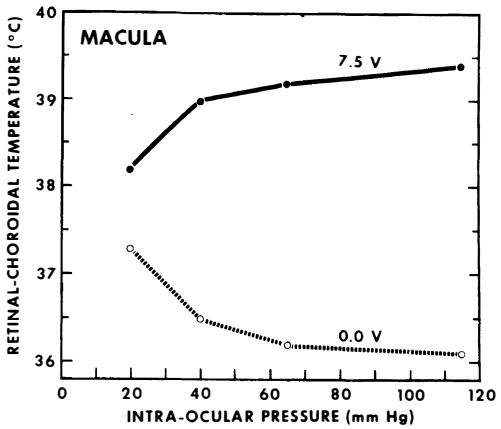

Fig. 1. *Macular temperature as a function of IOP at different light intensities. Each value was measured not less than one minute after alteration of the pressure at which time the temperatures had equilibrated. Exposed to the 7.5 V light source, increasing the IOP produces an increase in temperature (upper solid line). In contrast, increasing the IOP produces a decrease in tissue temperature when the eye is exposed only to background room illumination (lower dashed line).*[3]

helped maintain the temperature of the retina at or near central body temperature. A decrease in choroidal blood flow produced by increasing intraocular pressure decreased local tissue temperature (Fig. 1). When the eye is exposed to a light source, the absorption of light by the RPE and the choroid can raise local tissue temperatures above body temperature. Under these circumstances, the choroid now acts as a heat sink, dissipating heat by convection and conduction through the blood stream. Exposing the monkey eye to the light source and then decreasing choroidal blood flow by increasing intraocular pressure produced a marked increase in local

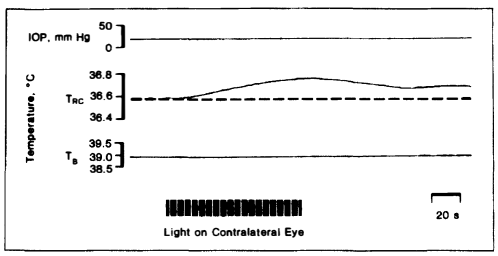

Fig. 2. *Light-stimulated reflexive increase in retinal-choroidal temperature (Trc). IOP indicates intraocular pressure and Tb body (rectal) temperature. Light exposure of contralateral eye occurred during period indicated by hatched bar.*[6]

tissue temperature, underscoring the importance of the choroidal vasculature in dissipating heat generated by the absorption of light. These changes were noted only in the macula and not in the retinal periphery. The presence of these changes only in the macula illustrates the intensified light-generated thermal loads in the macula produced by the focussing of light by the eye's optical system.

In addition to the passive ability of the choroidal vasculature to dissipate a light-generated heat load, there also exists a centrally mediated reflexive mechanism by which choroidal blood flow can be increased in response to light. This reflexive mechanism has also been studied in the monkey eye.[5,6] Temperature measurements were taken from either the macula or the scleral surface of the cynomolgus monkey, while the fellow eye was exposed to a moderate intensity light source. Light exposure of the fellow eye produced an increase in tissue temperature in the non-light exposed eye (Fig. 2). The increase in tissue temperature resulted from a reflexive increase in choroidal blood flow. The reflexive increase in choroidal blood flow in the non-light exposed eye was confirmed using a hydrogen washout technique (Fig. 3).[6]

The light-generated reflexive increase in choroidal blood flow has also been observed in the human eye.[7] Taking advantage of the thinness and relative avascularity of the sclera and conjunctiva, choroidal blood flow

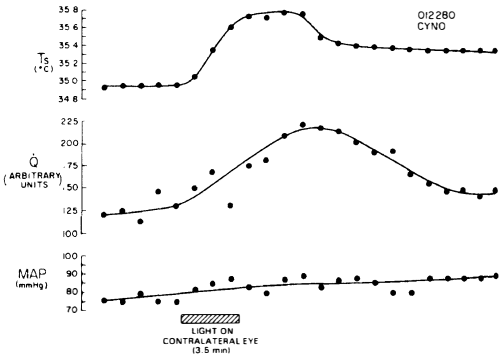

Fig. 3. *Reflexive increase in choroidal blood flow after light stimulation of contralateral eye. Ts indicates scleral surface temperature; Q, choroidal blood flow measured by hydrogen washout technique; and MAP, mean ocular arterial perfusion pressure. Light exposure of contralateral eye occurred during period indicated by hatched bar.*[6]

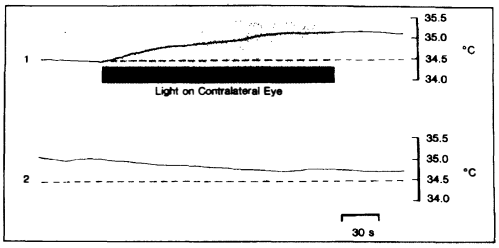

Fig. 4. *Scleral surface temperature recorded in normal human volunteer with flat thermistor probe inserted into superior conjunctival cul-de-sac, with temperature measuring surface lying against bulbar conjunctiva in superior temporal quadrant. Hatched bar indicates contralateral eye exposed to nonfocussed light of indirect ophthalmoscope set at 6.5 V.*[7]

measurements were made by measuring changes in bulbar conjunctival temperature (Fig. 4). The eye in which temperature measurements were made was occluded while the fellow eye was exposed to the light of an unfocussed indirect ophthalmoscope one metre away. Interestingly, the magnitude of the increase in choroidal blood flow was greatest in the human experiments.

Neuro-anatomical work in the pigeon has added further support for a centrally mediated mechanism for increasing choroidal blood flow. Gamlin *et al.* have demonstrated a pathway from the supra-chiasmatic nucleus to the contralateral Edinger-Westphal nucleus and then via the ciliary ganglion to the choroid.[8] Electrical stimulation of the pathway produces an increase in choroidal blood flow.[9] Other studies recently demonstrated that light can trigger this pathway to produce an increase in choroidal flow.[10]

These observations taken together make a strong argument for both a passive and active role for the choroidal circulation in maintaining a stable temperature environment for the retina, an environment that is particularly important in the macula.

Changes in choroidal blood flow are not the only way in which the eye modulates retinal tissue temperatures. Pupillary constriction in response to light effectively helps to moderate temperature changes in the macula. Additionally, the anatomical structure of the posterior segment vasculature acts passively to dissipate temperature. Usually in portions of the body that are exposed (such as an extremity) and have increased heat loss to the environment, blood flow is organised so that the warmer arterial blood travels adjacent to and in an opposite direction from the cooler venous flow. The arterial flow warms the returning venous blood, thereby helping to maintain core body temperature. In the eye, arterial and venous blood flow in the posterior segment course in the same posterior-to-anterior direction, further dissipating heat loss from the posterior segment.

In addition to the heat loss from the posterior segment by convection in the choroidal circulation, there is heat loss via convection (and to a lesser degree conduction) through the vitreous and aqueous humours. This can be observed clinically by the movement of cells in the posterior and anterior chambers, with the cells rising in the warmer posterior portions of the eye and falling in the cooler anterior portions. All of these mechanisms act to modulate the unique temperature problems produced by the absorption of light in the posterior segment, problems which are particularly acute in the macula.

Until 1966 and the observations of Noell and his associates, all light-induced retinal damage was commonly thought to be thermal in nature.[11] The work of Noell, Ham, and others shifted the current focus of light-induced retinal damage to photochemical processes.[12] There is, however, a tightly bound synergism between thermal and photochemical processes. Even with this understanding, there still exists the notion that all pure thermal damage requires temperature elevations capable of producing coagulation of intracellular components, temperatures in the range of 10° to 20°C. What has been neglected is the sensitivity of cellular enzymatic systems to temperature changes.

Metabolically active tissues such as the retina may be sensitive to temperature increases far below that necessary to produce coagulation of intracellular proteins. In effect, temperature increases can act as a metabolic poison, producing intracellular changes which may only become clinically evident days, weeks,or years later. Vos calculated that a temperature rise in the range of 2° could produce the clinical findings of solar retinopathy.[13] Clinically, the delayed effects of temperature damage may explain the fre-

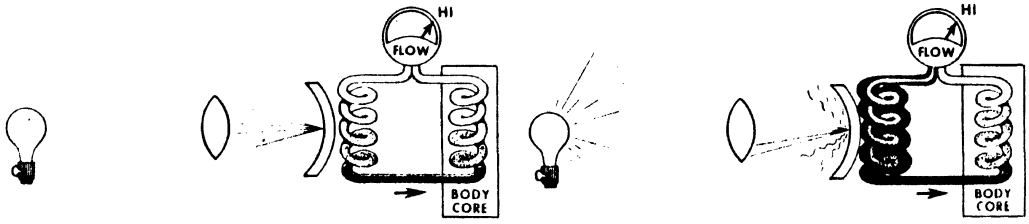

Fig. 5. *Model of function of the retinochoroidal circulation in normal animals and under conditions of thickened choroidal blood vessel walls. Left, focussing of light on the macula by the eye's optical system produces a local increase in tissue temperature. The high choroidal circulation, shown as a coil just behind the retina, absorbs the heat and stabilises local tissue temperature. The absorbed heat is dissipated by central body mechanisms (body core). Right, thickening of the blood vessel walls in the choroid decreases the efficiency of the choroidal vasculature in dissipating heat, allowing an increase in tissue temperature.*[3]

quent increase in size of photocoagulation lesions with time. The thermally-produced damage in photocoagulation has a Gaussian distribution with decreasing evidence of pathologic change toward the periphery of the lesion. There may exist a ring of retinal thermal damage which is not initially visible but which over time produces cellular death, which is clinically observable as an increase in size of the photocoagulation lesion.

The macula and posterior pole exist in a unique environment exposed to thermal and photochemical stresses. The eye has a number of anatomical and physiological mechanisms for dissipating the heat generated by the absorption of light. Yet temperature has been almost totally overlooked in the search for explanations for macular and other posterior pole diseases.

Pathologic studies of the eyes of patients with age-related macular degeneration have shown only minimal changes in the choroid. One change that has been observed is thickening of the vessel walls in the choroid.[14,15] Because of the high levels of blood flow in the choroid, it is unlikely that these changes would affect the delivery of oxygen and other nutrients to the outer retinal layers. These changes, however, could affect the ability of the choroidal circulation to dissipate heat generated by the absorption of focussed light as shown in Figure 5. These effects of thickening of the vascular wall are analogous to the decreased efficiency of an automobile radiator to act as a heat exchanger when, over time, its coils are thickened by corrosion.[3] A small increase in temperature over a long period of time could produce damage to the macula by a direct thermal effect or by amplifying photochemical damage. While this is speculative, the ability of the choroidal circulation to modulate the temperature environment of the macula should excite further study of the role of temperature in macular disease.

Keywords: choroid, choroidal blood flow, intraocular temperatures, macular blood flow.

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