



# Macular Hemodynamic Responses to Short-term Acute Exercise in Young Healthy Adults

PIERRE FORCIER,\* HÉLÈNE KERGOAT,\*† JOHN V. LOVASIK\*

We investigated the effects of vigorous exercise on blood flow in the macular vasculature. The velocity and density of entoptically viewed leukocytes in the paramacular retinal capillaries were measured with an Oculix BFS-2000 blue field simulator in 18 healthy adults first at rest, and then after 20 min of exercise. Exercise typically increased the density of leukocytes with more variable effects on their velocity. When leukocyte velocity and density were factored together, macular blood flow increased only marginally after exercise. We conclude that retinal blood flow in the macula is subject to the influence of autoregulatory mechanisms presumably to sustain normal central visual function during increased systemic blood flow. © 1997 Elsevier Science Ltd

Blue field entoptoscope   Exercise   Leukocytes   Macular blood flow   Vascular autoregulation

## INTRODUCTION

Exercise transiently alters the circulation in muscular and respiratory systems by increasing the cardiac output and systemic blood pressure (BP; Lamb, 1984; Vander *et al.*, 1989; Lamb *et al.*, 1990). Homeostatic mechanisms redistribute blood flow throughout the body so as to maintain critical levels of oxygen in vital organs and increased perfusion of active muscles according to metabolic demand. Few studies in exercise physiology have focussed specifically on the relationship between exercise, ocular blood flow and visual function. Earlier studies examining the effects of exercise on vision demonstrated increased visibility of both patterned visual stimuli (Koskela, 1988) and diffuse light (Jones & Wilcott, 1977) after exercise. However, the most frequently reported effect of exercise on the oculo-visual system is that of decreased intraocular pressure (IOP) (Marcus *et al.*, 1970; Myers, 1974; Passo *et al.*, 1987; Ashkenazi *et al.*, 1992). A therapeutic application of this decrease in IOP after exercise was proposed by Passo *et al.* (1989, 1994) who concluded that regular exercise could be a useful clinical adjunct to the clinical management of ocular hypertensives and glaucoma patients. More recently, Coupland *et al.* (1996) demonstrated that exercise combined with a totally vegetarian diet that excluded all refined oils and sugars, and consisted of low fat high fiber and no cholesterol significantly decreased the heart rate, lowered the systemic BP and the IOP, and increased the facility for

ocular perfusion within 15 days of enrollment in the lifestyle program. The exact mechanism by which the IOP is lowered through exercise is unclear, although decreased choroidal volume (Harris *et al.*, 1992) has been cited as a possible factor leading to a reduction in the steady state IOP.

Remarkably, only a few studies have attempted to elucidate the effects of exercise on blood flow to the eye as a whole, and particularly the neural retina. Most notably, Silver *et al.* (1991) have shown an increased pulsatility of blood flow in the ophthalmic artery by Doppler sonography after exercise. At the retinal level, Brinchmann-Hansen *et al.* (1989) reported that both arteries and veins constricted after an acute exercise-induced increase in the heart rate. Similarly, Lanigan *et al.* (1988) observed a constriction of both the retinal arteries and veins after an increase in systemic BP caused by isometric exercise.

To our knowledge, there have not been any studies that have examined the hemodynamic responsivity of the paramacular retinal vasculature to short periods of strenuous exercise. To fill this void, we examined the effects of a short interval of vigorous exercise on blood flow in the retinal vessels perfusing the macular area by blue field entoptoscopy (Riva & Petrig, 1980), a technique that yields a subjective estimate of the leukocyte flow in the macular capillary bed.

## MATERIALS AND METHODS

### Subjects

Eighteen (twelve men, six women) healthy paid volunteers between 21 and 28 yr of age (Mean age: 23.67, SD: 2.14 yr) participated in this study. All subjects were pre-screened to have 20/20 visual acuity or better in

\*École d'optométrie, Université de Montréal, C.P. 6128, succursale Centre-Ville, Montréal, Québec, Canada H3C 3J7.

†To whom all correspondence should be addressed [Fax: +1-514 343 2382; Email: kergoath@ere.umontreal.ca].

the test eye, as well as normal intraocular and brachial BPs. All subjects were fully informed of the test procedures as well as their right to withdraw from the study at any time and for any reason of their choosing. All aspects of the testing protocol were approved by our institution's ethics committee for human research and were in conformity with the Declaration of Helsinki. Written informed consent was obtained from each subject prior to testing.

#### *Quantification of the macular blood flow*

Blood flow in the macular capillaries was quantified with an Oculix BFS 2000 blue field simulator in reduced ambient lighting. The principle and procedure of blue field entoptoscopy have been described in detail by Riva and Petrig (1980). Briefly, the blue field entoptoscope allows the entoptic visualization of the leukocytes flow in the macular capillaries as the observer views a uniform luminous deep blue background (peak emission, 430 nm, 25 nm width at half-height). The blue light is absorbed by the hemoglobin in erythrocytes, while the leukocytes remain transparent to the blue illumination which consequently impinges on the retinal photoreceptors. As a result, the leukocytes appear as many small luminous spots in the observer's visual field that move in pulsatile fashion at a rate that is synchronous with the cardiac cycle, and moves along random curved paths defined by the inner loops of the retinal capillaries. These moving spots are absent in the avascular foveal zone that is perfused exclusively by the choroid.

In preparation for testing, each subject adjusted the intensity of the blue light illuminating the retina to optimize the visibility of entoptically viewed leukocytes. Once set, this lighting level was kept constant throughout all subsequent testing. All subjects were given a period of training on the entoptoscope so as to attain a high degree of repeatability for leukocyte velocity and density measurements. Subjects who could not achieve a measurement variation of  $< 20\%$  across five consecutive trials were not admitted to the study. Our criterion for acceptance into the study was that recommended by the supplier. All subjects reported easily achieved this level of reliability after proper training.

Training on the blue field apparatus took the form of repeated test sessions under the close coaching of the experimenter. The experimenter detailed what was expected of the subject during blue field testing by demonstrating the effects of adjustments of the computer control knobs that adjusted the density (number of leukocytes per unit area of screen) and velocity (displacement speed of the leukocytes on the screen) of the simulated leukocytes. The subject was then positioned in the Oculix BFS 2000 blue field simulator to view his own leukocytes and asked to compare the density and velocity of entoptically viewed leukocytes to those presented intermittently on a computer monitor past a solenoid displaced mirror. The velocity and density controls on the computer were adjusted as often as required until the subject felt that a match was established

between the physiological and simulated leukocyte dynamics. If the control settings were within the criterion variability level of 20%, the subject was asked to participate in the study. If the subject did not achieve the 20% variability criterion or was not comfortable with his understanding or the performance requirements of the task, he was invited to return for additional training, or could withdraw from the study altogether. Subjects failing to achieve satisfactory performance after the second training session were paid for their time but not accepted into the formal part of the study.

When a match of the dynamic characteristics of the physiological and simulated leukocytes was attained, the settings were automatically stored in the blue field computer system, and then four additional matches were made to derive an averaged measurement of leukocyte density and velocity characteristics. Thus individual and the average of five consecutive leukocyte matching data sets for each subject were made for each test condition, and stored for subsequent analysis and population statistics. To minimize any carry-over effects and learning across trials, the separate knobs controlling the density and the velocity of the simulated leukocytes were readjusted randomly by the experimenter between trials. Since the principal objective of the study was to compare the leukocyte flow characteristics before and after exercise under identical viewing conditions within individual subjects, corrections for the distance of the monitor from the subject and the subject's ocular refraction to derive absolute values for leukocyte velocity and density were not deemed necessary.

For both resting and post-exercise blood flow measurements, subjects wore any habitual glasses or contact lenses to view the simulated leukocytes displayed on a computer monitor some 50 cm away from the ocular of the BFS 2000 system. An equivalent sphere compensating lens was positioned before the eye to compensate optically for the observation distance due to the loss of accommodative ability caused by the topical cycloplegic agent whenever clear visibility of the simulated leukocytes on the monitor was not achievable because of the residual refractive status of the eye. As part of a concurrent study examining the effects of intense exercise on retinal function via the flash electroretinogram, the pupil of the test eye was fully dilated and accommodation paralyzed temporarily with one drop of 1% cyclopentolate HCl (Cyclogyl). This eye was used to make macular blood flow measurements reported in the present study.

A miniature blood flow sensor from the BFS 2000 system was clipped to the earlobe to relay the subject's blood flow pulsatility to the computer so that the computer simulated leukocytes had the same pulsatility characteristics as the biological leukocytes.

#### *Exercise on a stationary bicycle*

A computerized stationary bicycle (Cat-Eye, Model EC-1500 ergociser, Physio E.R.P. Ltd, Laval, Quebec, Canada) was used to standardize the physical exertion

during the test session. An integrated microprocessor displayed the moment-to-moment heartbeat, and allowed for a pre-selected heartbeat rate to be achieved or workload to be monitored and applied.

At the beginning of each test session the height of the ergociser handles and seat were adjusted to the subject's stature and maximized his/her comfort throughout testing. Pedal straps on the bike were also tightened to suit each subject's feet and ensure an un-interrupted and constant load during each exercise session. A clip-on blood flow sensor attached to the earlobe time averaged the heart rate and allowed an accurate match between the work demand and output.

Each subject was required to exercise at a rate that would elevate the heart rate to the criterion of 140 beats/min throughout 20 min of exercise. When the targeted heart rate was achieved by each subject, typically within a minute or so of pedaling, the automated control of the cadence of pedaling and/or the resistance of the pedals sustained the heartrate at 140 beats/min. For our subjects, the applied physical load increased the resting heartrate by an average of  $\times 1.78$ . Measurements of macular blood flow were taken at rest, and immediately after the 20 min exercise session, while the subject was seated on the ergociser. For the measurement of retinal macular blood flow after exercise, subjects stopped pedaling during all adjustments of leukocyte density and velocity. After the first post-exercise blue field blood flow measurement, and each of the next three measurement sessions, subjects were required to pedal until the target heart rate of 140 beats/min was achieved, and then the blue field measurements were repeated.

#### *Quantification of the IOP, brachial blood pressure and ocular perfusion pressure*

The IOP was derived for the right eye with the gas tonometer forming part of a Langham Ocular blood flow system whose IOP printout typically represented the average of over 100 instantaneous IOP measurements over a 5 sec period. To obtain the IOP measurements, the cornea was first anaesthetized with 1–2 drops of topical proparacaine HCl 0.5% (Alcaine®). At least two IOP records were made, and averaged to derive IOP values representative of the baseline and test conditions.

Brachial BPs were taken objectively with an electronic sphygmomanometer (Spacelabs®), with the subject seated in the ergociser and the arm resting comfortably on the bicycle handle. Two consecutive measurements of BP were averaged to derive BPs representative of the baseline and test conditions.

The mean brachial BP ( $p_{\text{mean brachial}}$ ) was calculated according to the following formula originally advanced by Weigelin and Lobstein (1963) for the time-averaged pressure in a vessel with pulsatile flow:  $p_{\text{mean brachial}} = p_{\text{diastolic}} + (p_{\text{systolic}} - p_{\text{diastolic}}) \times 0.42$ , where 0.42 is a time average constant for the systolic and diastolic phases of the cardiac cycle. For healthy individuals, the mean pressure in the central retinal artery (CRA) can be predicted from  $p_{\text{mean brachial}}$  using the

formula:  $p_{\text{mean CRA}} = 0.73 (p_{\text{mean brachial}} - \text{IOP}) + \text{IOP}$ , where 0.73 is a correction factor that takes into account the distance between the heart and the eye (Lovasik *et al.*, 1994). The ocular perfusion pressure (OPP) was calculated from the formula:  $\text{OPP} = p_{\text{mean CRA}} - \text{IOP}$ .

#### *Statistical analyses*

Paired *t*-tests, as well as analyses of variance (ANOVA) with an alpha level of 0.05 were used to determine whether any observed trends in data sets across test conditions and subjects achieved statistical significance.

## RESULTS

All IOP values for our subjects were within clinical normal limits. The range of IOPs for the baseline resting condition was 14.0–21.4 mmHg and 11.3–20.7 mmHg after exercise. The group average decrease of 1.46 mmHg after exercise was statistically significant ( $P = 0.02$ ).

The range of brachial BPs for the baseline resting vs exercise conditions were also within clinical norms, 108/70–137.5/90.5 mmHg, and 123.5/77.0–189.5/94.5 mmHg for the resting and post-exercise conditions, respectively. The group-averaged BP increased significantly ( $P \leq 0.0007$ ) after exercise (124.6/78.69–152.72/96.11 mmHg). The group-averaged increase in BP after exercise was 28.11/17.42 mmHg.

The calculated OPP across subjects ranged between 51.93 and 67.72 mmHg. The group averaged increase of 17.8 mmHg in the OPP after exercise achieved statistical significance ( $P = 0.0001$ ).

The change in leukocyte *velocity* after exercise fell into two categories. About 61% (11) of subjects showed an increase in velocity while about 39% (7) subjects showed reduced leukocyte velocity. These data are illustrated graphically in Fig. 1(A) where individual data have been ordered from the smallest to the largest *differences* found between the resting and post-exercise conditions.

An evaluation of the leukocyte *density* data reveals a more uniform response to exercise than that seen for the velocity data. Some 83% (15 out of 18) of the subjects showed an increase in leukocyte density while only 17% (three out of 18) of the subjects showed a decrease in leukocyte density. These data are illustrated graphically in Fig. 1(B) where this data also has been ordered from smallest to largest changes in density.

A comparison of the group averaged ( $n = 18$ ) leukocyte velocities before (mean:  $1.03 \pm \text{SEM } 0.05$ ) and after (mean:  $1.13 \pm \text{SEM } 0.08$ ) exercise indicated a tendency for a slight increase in leukocyte velocity after exercise, but this change did not achieve a statistically significant level ( $P > 0.05$ ). The group averaged ( $n = 18$ ) leukocyte density before and after exercise indicated that the leukocyte density increased significantly from about 106 to 150 units after exercise ( $P = 0.0063$ ).

In order to obtain an overview of global changes in macular blood flow with exercise, the leukocyte velocity and density measurements were combined to derive a so called "flow index" so as to allow a comparison of

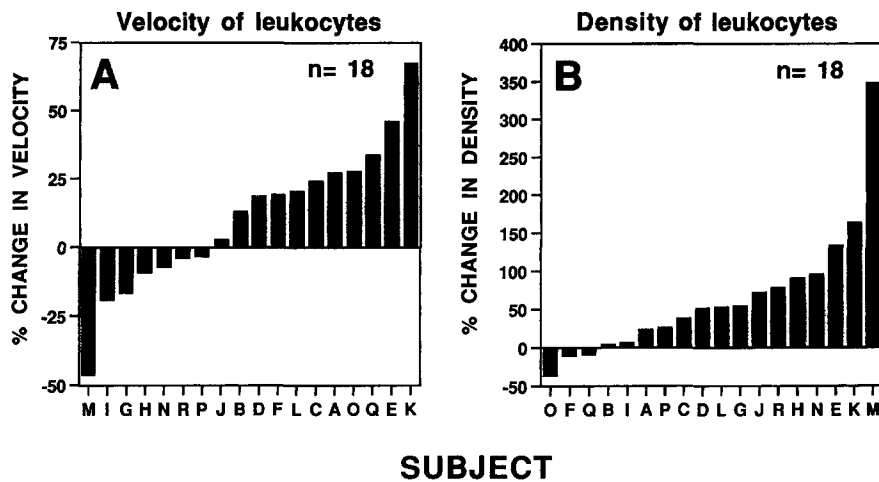


FIGURE 1. Summary of the effects of exercise on macular blood flow as indexed by the percentage change in (A) leukocyte velocity and (B) leukocyte density with respect to resting values. The velocity and density data for individuals have been ordered independently to show that exercise had differing effects on both the direction and degree of change in the test parameters across subjects. Data bars below the zero value on the ordinate identify subjects whose leukocyte velocities and densities were below baseline values after exercise, while those above the zero line represent those subjects in whom these parameters were increased by exercise.

hemodynamic responses across subjects. The flow index was expressed as the number of leukocytes flowing through the retinal macular capillaries per unit time, that is, (leukocyte density)  $\times$  (leukocyte velocity). Figure 2 compares this index of blood flow in the macula during exercise and resting conditions for our test group. The linear regression line through the data points indicated a slight increase in the flow index after exercise regardless of the degree of blood flow found for baseline conditions across our cohort of subjects. This observation suggests the existence of a mechanism in all subjects that regulates and maintains the amount of blood flowing through the macular capillaries nearly constant throughout rest and when there is a significant elevation in both the systemic BP and OPP as occurs during rigorous exercise.

### DISCUSSION

Our findings related to the effects of exercise on IOP and systemic BP parallel previous findings on these variables (Marcus *et al.*, 1970; Myers, 1974; Passo *et al.*, 1987; Ashkenazi *et al.*, 1992; Lamb, 1984). In addition, however, our study has demonstrated that short periods of intense exercise predominantly altered the *density* of leukocytes in the macular capillaries. Most previous studies assessing macular blood flow by the blue field technique have mainly quantified leukocyte *velocity* rather than leukocyte density (Riva *et al.*, 1981; Petrig *et al.*, 1985; Robinson *et al.*, 1985a,b; Sponsel *et al.*, 1990). This latter focus on leukocyte velocity has likely been based on the assumption that a change in velocity is equivalent to a change in volumetric blood flow since the paramacular capillaries have cross-sectional diameters of some 7–10  $\mu\text{m}$  which just permits the passage of leukocytes, and the capillary walls do not have muscle layers to modify their cross-sectional diameter (Sinclair *et al.*, 1989).

In the present study, it is interesting to see that the group averaged leukocyte velocity remained essentially unchanged in the presence of a significant increase in the group averaged leukocyte density. This can be interpreted simply as a tighter packing of the leukocytes within the same length of capillaries. The increased density could also be related to an increase in the number of leukocytes in the general circulation after exercise, either through recruitment mechanisms (Harris *et al.*, 1994), through an increase in the number of precapillary sphincters that are opened as a result of altered blood chemistry during exercise (Vander *et al.*, 1989), or less likely, through a rapid increase in the production of new blood cells. In this regard, a comparison of blood chemistry analyses for one of our subjects before and after biking indicated a significant increase in the number of red and white blood cells. The mechanism by which the density of blood cells would be increased through exercise could not be defined by the design of the present study.

The five consecutive entoptic measurements of blood flow for a subject each required a finite time for appropriate adjustments of leukocyte density and velocity to be made. The group average leukocyte density and leukocyte velocity measurement times between the first and fifth test sessions was  $4.44 \pm 0.4$  min (SEM). After the first blue field measurement series taken immediately at the end of the 20 min exercise period, subjects were required to rapidly pedal and each time return the heartbeat to the criterion 140 beats/min. An analysis of the blue field blood flow estimates over the five measurement sessions for our cohort of subjects did not reveal any trend for change in our index of blood flow, (leukocyte density)  $\times$  (leukocyte velocity), (ANOVA,  $P = 0.9346$ ). A further comparison of the group averaged blue field values immediately after exercise and the last measurement set after exercise did not differ (paired *t*-

test,  $P = 0.7648$ ). The results of these analyses were interpreted as proof that there were no systematic effects of repeated measurements of blood flow over time; and that the biking spurts between blood flow measurement sessions to restabilize the heart beat at 140 beats/min achieved similar physiological states throughout the entire blood flow measurement interval.

A recent study by Petrig *et al.* (1993) reported that the density of perceived leukocytes increased as intensity of the blue background light used to visualize the leukocytes was increased. Our present results are not likely to have been influenced by this variable in blue field entoptoscopy since each subject had been asked to adjust the background luminance so as to optimize the visibility of his own leukocytes, and the pupils were maximal and nonreactive to light or accommodation due to the application of a cycloplegic eye drop. Once determined, this intensity setting was kept constant throughout all phases of the experiment. In addition, all measurements of blood flow were carried out on the right eye of each subject. These test eyes were previously light-adapted to a uniform, constant background light within a Ganzfeld bowl in preparation for photopic ERG recordings that formed the basis of a companion study.

Vascular autoregulation within the eye is defined as the ability to maintain a constant flow of blood, in the presence of altered perfusion pressure (Bill, 1975). Our results showing that leukocyte velocity was not uniformly altered by acute exercise may be indicative of a form of autoregulation for the macular capillaries that relies not only upon blood velocity but also blood cell density. Some support for this hypothesis comes from the data manipulation presented graphically in Fig. 2. When "leukocyte velocity  $\times$  density" during exercise was compared to "leukocyte velocity  $\times$  density" for resting conditions, a linear relationship was seen. The positive slope of the regression line indicated that a subject's basal index of blood flow in the macula was closely related to the blood flow measured during exercise which overall was slightly higher after exercise. Thus, blood flow as reflected by the "flow index" was maintained at a near constant level during considerable physiological stress, suggesting regulated blood flow either in the macular capillaries *per se*, or the arterioles giving rise to the capillary network. Some subjects responded to the physiological challenge by an increased leukocyte velocity combined with a slight decrease in leukocyte density, while others demonstrated the reverse response to achieve constancy of leukocyte flow in the macular area throughout exercise. The ability to maintain blood flow to the macula relatively constant perhaps is not surprising since the larger retinal vessels from which the macular capillaries are derived have previously been shown to have autoregulatory capacity during physiological stress applied locally through scleral suction (Petrig *et al.*, 1985; Riva & Loeb, 1977; Riva *et al.*, 1981). However, the observation that the macular blood flow increased only slightly after a relatively large generalized increased metabolic load through exercise leads one to

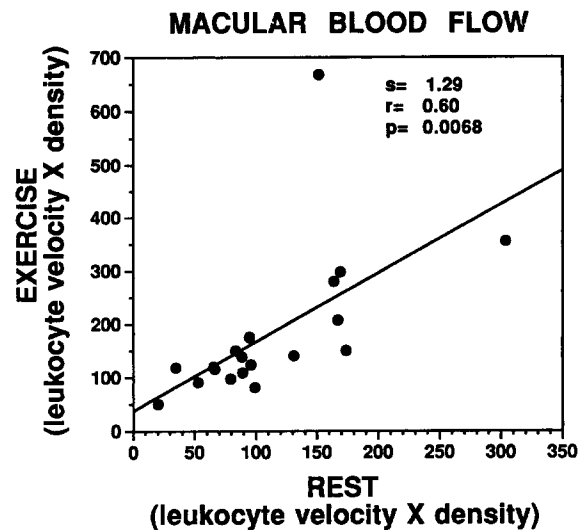


FIGURE 2. Comparison of the macular blood flow at rest and immediately after exercise for our test group ( $n = 18$ ) of subjects. Blood flow is quantified here as the product of (leukocyte velocity)  $\times$  (leukocyte density). In the graph, "s" represents the slope of the linear regression line through the data points; "r" the Pearson correlation coefficient, and "p" the alpha level of significance for the slope of the regression line. The slope of the linear regression line through the data points (+1.29) indicates a slight increase in flow after exercise over the wide range of basal flow values found in our subjects. Only one subject deviated from otherwise tightly clustered data points.

speculate that a relatively constant blood flow such as seen in the present study is the physiological objective of the capillaries feeding the macular area even when the stress nearly doubles the heart rate. However, since no measurements of central vision were made in the course of this study, it is uncertain whether the predominant increase in leukocyte density coupled with the pre-existing blood supply in the choroid was adequate to maintain central visual function at its normal level of sensitivity.

The present experimental design involved raising the heart rate to a criterion 140 beats/min in each test subject to determine the effect of increased systemic perfusion on blood flow in the perimacular retinal capillaries subsequent to biking, a conventional format of exercise. However, unquantified individual differences in cardiovascular efficiency might have translated into differences in the absolute workloads across subjects and differences in the OPP across subjects. Such differences could conceivably result in differences in the measured macular blood flow. An analysis of our data, looking at percent change in velocity, density, and (leukocyte velocity)  $\times$  (leukocyte density) as a function of percent change in OPP showed that there was no significant correlation between the level of change in the OPP and the degree of change in these blood flow indices; all  $P > 0.22$  for linear regression lines correlating density, velocity, and (leukocyte velocity)  $\times$  (leukocyte density) with OPP. This suggests that even if subjects differed in the effort required to achieve the pre-set criterion of 140 beats/min, all subjects showed relatively little change in the macular blood flow. Therefore in our subject population,

autoregulation appeared to be operative and maintained blood flow relatively constant in spite of different levels of OPP. This latter observation is consistent with the results of an earlier study where the present authors showed macular blood flow to be independent of the degree of change in OPP during body inversion procedures (Lovasik & Kergoat, 1994).

In summary, our study has shown that acute exercise is associated with a near constancy of blood flow within the macular area effected through a modulation of leukocyte velocity and density. The measured changes in leukocyte velocity and density were interpreted as part of a hemodynamic autoregulatory mechanism within the macular area in response to physiological stress associated with exercise. Our results also indicate that future studies of ocular hemodynamics using a blue field entoptoscope should quantify changes in both leukocyte velocity and density for a more comprehensive understanding of the macular vessel response to physiological stress. Additional information on macular retinal blood flow dynamics may be gleaned using more rapid and advanced objective techniques such as laser Doppler flowmetry.

#### REFERENCES

- Ashkenazi, I., Melamed, S. & Blumenthal, M. (1992). The effect of continuous strenuous exercise on intraocular pressure. *Investigative Ophthalmology and Visual Science*, *33*, 2874–2877.
- Bill, A. (1975). Ocular circulation. In Moses, R. A. (Ed.), *Adler's Physiology of the eye. Clinical application* (6th edn, pp. 210–231). St. Louis: Mosby.
- Brinchmann-Hansen, O., Myhre, K. & Sandvik, L. (1989). Retinal vessel responses to exercise and hypoxia before and after high altitude acclimatisation. *Eye*, *3*, 768–776.
- Coupland, S. G., Mellgren, S. G. M., Crane, M., Lovasik, J. V. & Gimbel, H. V. (1996). Effect of lifestyle change on intraocular pressure and ocular blood flow. *Investigative Ophthalmology and Visual Science*, *37 Suppl.*, S36.
- Harris, A., Arend, O., Kopecky, K., Caldemeyer, K., Wolf, S., Sponsel, W. & Martin, B. (1994). Physiological perturbation of ocular and cerebral blood flow as measured by scanning laser ophthalmoscopy and color Doppler imaging. *Survey of Ophthalmology*, *38 Suppl.*, S81–S86.
- Harris, A., Malinovsky, V. E., Cantor, L. B., Henderson, P. A. & Martin, B. J. (1992). Isocapnia blocks exercise-induced reductions in ocular tension. *Investigative Ophthalmology and Visual Science*, *33*, 2229–2232.
- Jones, R. K. & Wilcott, I. T. (1977). Topographic impairment of night vision related to exercise. *American Journal of Ophthalmology*, *84*, 868–871.
- Koskela, P. U. (1988). Jogging and Contrast sensitivity. *Acta Ophthalmologica*, *66*, 725–727.
- Lamb, D. R. (1984). *Physiology of exercise: Responses and adaptations* (2nd edn). New York: Macmillan.
- Lamb, J. F., Ingram, C. G., Johnston, I. A. & Pitman, R. M. (1990). *Manuel de physiologie*. Paris: Masson.
- Lanigan, L. P., Clark, C. V. & Hill, D. W. (1988). Retinal circulation responses to systemic autonomic nerve stimulation. *Eye*, *2*, 412–417.
- Lovasik, J. V. & Kergoat, H. (1994). Gravity-induced homeostatic reactions in the macular and choroidal vasculature of the human eye. *Aviation, Space and Environmental Medicine*, *65*, 1010–1014.
- Lovasik, J. V., Kothe, A. C. & Kergoat, H. (1994). A comparison of non-invasive methods to derive the mean central retinal artery pressure in man. *Optometry and Vision Science*, *70*, 1005–1011.
- Marcus, D. F., Krupin, T., Podos, S. M. & Becker, B. (1970). The effect of exercise on intraocular pressure: Human beings. *Investigative Ophthalmology*, *9*, 749–752.
- Myers, K. J. (1974). The effect of aerobic exercise on intraocular pressure. *Investigative Ophthalmology*, *13*, 74–77.
- Passo, M. S., Goldberg, L., Elliot, D. L. & VanBuskirk, E. M. (1987). Exercise conditioning and intraocular pressure. *American Journal of Ophthalmology*, *103*, 754–757.
- Passo, M. S., Goldberg, L., Elliot, D. L. & VanBuskirk, M. V. (1989). Regular exercise lowers intraocular pressure in ocular hypertensive subjects. *Investigative Ophthalmology and Visual Science*, *30 Suppl.*, 28.
- Passo, M. S., Hunt, S. C., Elliot, L. & Golberg, L. (1994). Regular exercise lowers intraocular pressure in glaucoma patients. *Investigative Ophthalmology and Visual Science*, *35 Suppl.*, 1254.
- Petrig, B. L., Kamoun, L. & Riva, C. E. (1993). Effect of light intensity on macular blood flow parameters measured by blue field simulation. *Noninvasive Assessment of the Visual System*, *3*, 236–239.
- Petrig, B., Werner, E. B. & Riva, C. E. (1985). Response of macular capillary blood flow to changes in intraocular pressure as measured by the blue field simulation technique. In Heijl, A. & Greve, E. L. (Eds) *Proceedings of the 6th Visual Field Symposium* (pp. 447–451). Dordrecht: Junk.
- Riva, C. E. & Loebl, M. (1977). Autoregulation of blood flow in the capillaries of the human macula. *Investigative Ophthalmology and Visual Science*, *16*, 568–571.
- Riva, C. E. & Petrig, B. (1980). Blue field entoptic phenomenon and blood velocity in the retinal capillaries. *Journal of the Optical Society of America*, *70*, 1234–1238.
- Riva, C. E., Sinclair, S. H. & Grunwald, J. E. (1981). Autoregulation of retinal circulation in response to decrease of perfusion pressure. *Investigative Ophthalmology and Visual Science*, *21*, 34–38.
- Silver, D. M., Farrell, R. A., Langham, M. E., Michelson, G. & Schilder, P. (1991). Ocular blood flow in response to acute exercise. *Investigative Ophthalmology and Visual Science*, *32 Suppl.*, 864.
- Sinclair, S. H., Azar-Cavanagh, M., Soper, K. A., Tuma, R. F. & Mayrovitz, H. N. (1989). Investigation of the source of the blue field entoptic phenomenon. *Investigative Ophthalmology and Visual Science*, *30*, 668–673.
- Sponsel, W. E., DePaul, K. L. & Kaufmann, P. L. (1990). Correlation of visual function and retinal leukocyte velocity in glaucoma. *American Journal of Ophthalmology*, *109*, 49–54.
- Vander, A. J., Sherman, J. H., Luciano, D. S. & Gontier, J. R. (1989). *Physiologie humaine* (2nd edn). Montréal: McGraw-Hill.
- Weigelin, E. & Lobstein, A. (1963). *Ophthalmodynamometry*. New York: Hafner.

---

*Acknowledgements*—This work was funded in part by research awards from the Canadian Optometric Education Trust Fund to P. Forcier and H. Kergoat, Fonds pour la formation de chercheurs et l'aide à la recherche (FCAR) to H. Kergoat, and the Natural Sciences and Engineering Research Council of Canada, and Bausch and Lomb InVision grant to H. Kergoat (OGP0121750) and J. V. Lovasik (OGP0116910). We gratefully acknowledge the technical expertise of N. Lalonde and M. Gloin, and thank all of our subjects for their participation in the study.