

Posture Changes and Subfoveal Choroidal Blood Flow

Antonio Longo,¹ Martial H. Geiser,² and Charles E. Riva²

PURPOSE. To evaluate the effect of posture change on subfoveal choroidal blood flow (ChBF) in normal volunteers.

METHODS. The pulsatile, nonpulsatile, and mean ChBF were measured with laser Doppler flowmetry in 11 healthy volunteers with a mean age of 32 ± 13 (SD) years. The posture of the subjects was changed from standing (90°), to supine (-8°), and back to standing, with a mechanically driven table. During the whole experimental procedure, ChBF and heart rate (HR) were continuously recorded. After 30 seconds in standing position, the subjects were tilted to supine during approximately 30 seconds. They remained in this position for approximately 2 minutes, after which they were tilted back to the standing position (recovery), where they remained for another approximately 2 minutes. Systemic brachial artery blood pressure (BP) was measured in the baseline, supine, and recovery positions. This procedure was repeated to measure the intraocular pressure (IOP) at the different postures.

RESULTS. Mean BP did not change significantly throughout the experimental procedure. As the body was tilted from standing to supine, HR decreased by 16% ($P < 0.0004$), IOP increased by 29% ($P < 0.001$), and mean ChBF increased by 11% ($P < 0.01$). The increase in ChBF was primarily due to an increase in the nonpulsatile component of the blood velocity.

CONCLUSIONS. Based on previously reported experimental data that indicate that the ocular perfusion pressure increases less than predicted by purely hydrostatic considerations when the body is tilted from the standing to the supine position, the observed increase in ChBF suggests a passive response of the choroidal circulation to the posture change. (*Invest Ophthalmol Vis Sci.* 2004;45:546-551) DOI:10.1167/iovs.03-0757

The nutrition of the retinal foveal avascular zone depends entirely on blood flow in the subfoveal choroidal vasculature, and more specifically in the choriocapillaris, the innermost layer of the choroid. Therefore, the measurement of blood flow and its regulation in this region is of utmost importance in understanding the pathophysiology of diseases, such as diabetic retinopathy, age-related macular degeneration, and others.

Body inversion represents a simple and convenient method of changing the ocular perfusion pressure. For that reason, it has been widely used as a provocation test for the ocular circulation. Several studies, in which a variety of techniques were used, have described the effect of body position on the orbital, retrobulbar, optic nerve, and retinal circulations.¹⁻¹¹ For the choroidal circulation, studies have been limited to the

assessment of the effect of postural change on the pulsatile component of ChBF, as assessed by the POBF system.^{12,13} To the best of our knowledge, no information is available on the effect of posture change on the nonpulsatile and mean components of choroidal blood flow in humans. In this work, we used continuous, confocal laser Doppler flowmetry (LDF) to assess the effect of posture change on the three components of subfoveal choroidal blood flow (ChBF).

MATERIALS AND METHODS

Subjects

Eleven (seven men, four women) healthy volunteers (age range, 18-61 years; mean, 32 ± 13 [SD]), participated in the study. They had a visual acuity better than 0.8 (emmetropic or < 2 D myopic or hyperopic), clear media, and no history of ocular or systemic pathologies or therapy. The procedures adhered to the tenets of the Declaration of Helsinki. Written informed consent was obtained from all the subjects after the nature and possible consequences of the study were fully explained.

Posture Change and Systemic Blood Pressure

An electrically driven tilting table for radiology (DuoDiagnost; Philips, Eindhoven, The Netherlands) was rotated from 90° to -8° and back to 90° to tilt the subjects from the standing to the supine position and back to the standing position (recovery). The motion from the standing to the supine position and vice versa took approximately 30 seconds. Subjects were connected to the table using straps to prevent them from sliding (Fig. 1). Preliminary trials were first performed to familiarize the subject with the procedure and verify that stable LDF recordings could be obtained during the posture changes.

Brachial artery blood pressure (BP) was measured in standing (baseline), supine, and recovery positions with an automatic sphygmomanometer. Height difference between the eye and the heart was measured for each subject at standing and supine positions. Intraocular pressure (IOP) in these two positions was determined by a handheld tonometer (Tonopen; Mentor, Norwell, MA) during a trial in which no LDF measurements were performed.

The mean brachial artery pressure was calculated according to the formula³: $BP_m = BP_{dias} + 0.42 \cdot (BP_{sys} - BP_{dias})$, where BP_{dias} and BP_{sys} are the diastolic and systolic brachial artery blood pressures, respectively.

Laser Doppler Flowmetry

The principle of ChBF measurements by LDF and the flowmeter used in this work have been described.¹⁴ The flowmeter was mounted on a mechanical support fixed on the tilting table and placed in front of the eye. This device delivered a laser beam from a laser diode ($\lambda = 785$ nm, $90 \mu\text{W}$ at the cornea) through a confocal optical system. Nominal diameter of the beam at the fundus, when focused, was approximately $12 \mu\text{m}$. The light scattered by the red blood cells and surrounding tissue was collected by six fibers, each having a core diameter of $110 \mu\text{m}$. These fibers were arranged on a $180\text{-}\mu\text{m}$ diameter circle centered on the incident beam. The collected light was guided to an avalanche photodiode, and the output current was amplified before being analyzed by computer (NeXT; Redwood, CA).¹⁵ The LDF photocurrent was fed into a loudspeaker to obtain an audio signal. Its DC, which is proportional to the intensity of the scattered light, was also recorded and displayed.

From the ¹Istituto di Oftalmologia, Università di Catania, Catania, Italy; the ²Institut de Recherche en Ophthalmologie, Sion, Switzerland.

Supported by Swiss National Fund Grant 3163755.00 and Società Oftalmologica Siciliana, Italy.

Submitted for publication July 18, 2003; revised October 7, 2003; accepted October 14, 2003.

Disclosure: A. Longo, None; M.H. Geiser, None; C.E. Riva, None

The publication costs of this article were defrayed in part by page charge payment. This article must therefore be marked "advertisement" in accordance with 18 U.S.C. §1734 solely to indicate this fact.

Corresponding author: Martial H. Geiser, Institut de Recherche en Ophthalmologie, Grd Champsec 64, 1950 Sion, Switzerland; martial.geiser@hevs.ch.



FIGURE 1. The LDF placed in front of the tested subject's eye is mechanically connected to the table. The computer-based data-acquisition system is at the *bottom left* in the photograph.

Subjects were asked to fixate directly on the probing laser beam, which appeared as a weak but clearly visible tiny spot, and to focus it by adjusting the front lens of the flowmeter. Then the operator precisely aligned the device with the eye to obtain maximum loudness of the audio Doppler signal and constant DC during the experiment.

The subfoveal LDF parameters calculated from the analysis of the photocurrent are the velocity, ChBVel (kHz), the volume, ChBVol (arbitrary units [au]) and the flux, ChBF (au), of the red blood cells. These parameters are related to each other through the relationship $\text{ChBF} = k \cdot \text{ChBVel} \cdot \text{ChBVol}$, where k represents an instrumental constant. Assuming no change in hematocrit during the experiments, the changes in the LDF parameters are proportional to the changes in the actual velocity, volume, and flow of blood in the sampled tissue, respectively. Artifacts caused by rapid eye movements and blinks were easily identifiable in the recordings. They were manually removed, as described in a previous publication.¹⁶

By recording the heart pulse using an ear infrared plethysmometer, the average time course of each LDF parameter during the cardiac cycle was obtained by averaging over a period T , taking into account the phase of the heart cycle, as previously described.¹⁷ This average time course for ChBVel, which we denote as ChBVel_T in Figure 2, consists mainly of a nonpulsatile and most often of a much smaller pulsatile component. The magnitude of the nonpulsatile component is the minimum of ChBVel_T , that of the pulsatile component as the mean of ChBVel_T minus this minimum.

Experimental Protocol

After a 5-minute period with the tested subject in a standing position, LDF measurements were performed in the right eye for approximately 30 seconds. Then, while we proceeded with the LDF measurements, the table was tilted to bring the subject into supine position within

approximately 30 seconds. The subject remained in this position for approximately 2 minutes after which the table was tilted back to the vertical position and kept in this position for approximately 2 minutes. During the whole experiment, which was performed in room light, the LDF parameters were continuously recorded. The pupils of the subject were not dilated.

The baseline of the LDF parameters were defined as the average values of these parameters obtained during the approximately 30 seconds of measurement time in the starting (standing) position. The averaged data obtained during the last minute in supine position were defined as supine LDF parameters and those obtained after 40 seconds of recovery time were averaged to obtain the recovery LDF parameters.

Statistics

Mean changes in the LDF parameters were assessed for significance using the Student's paired t -test. $P \leq 0.05$ was considered significant.

RESULTS

Systemic BP, IOP, and HR

Table 1 shows the group average BP_{dias} , BP_{sys} , BP_m , IOP, and HR at baseline (standing) and supine positions. Between these two positions, BP_{dias} decreased significantly by 13% (paired t -test, $P = 0.016$), whereas BP_{sys} and BP_m did not change significantly. IOP increased significantly by 4 mm Hg (29%, $P = 0.001$) and HR decreased significantly by 16% ($P = 0.0004$).

LDF Parameters

Figure 3 shows a representative recording of the LDF parameters recorded as the subject was tilted from standing to supine and back to standing. A change in ChBVel and ChBF during tilting was clearly observed, whereas there was no apparent change in ChBVol.

For each LDF parameter, the mean values during baseline (Bl), supine (Sp), and recovery (Rc) were determined (Fig. 4) by averaging each parameter over the respective periods indicated by black horizontal bars lines on the time scale of Figure 3. In one subject, the data during Rc were measured during the first 20 seconds in this position, because no data could be obtained afterward.

Table II shows the group average changes for the nonpulsatile, pulsatile and mean components of the LDF parameters between baseline and supine and baseline and recovery. These changes were expressed in % of the baseline values.

Both group average of the magnitude of the mean and nonpulsatile components of ChBVel_T (Table 2) increased significantly (8%) between the baseline and supine positions. For ChBF_T these increases were respectively 12% and 11%. There was no significant change in all components of ChBVol_T . None of the pulsatile, nonpulsatile, and mean components of ChBVel_T and ChBF_T at recovery were significantly different from baseline. In contrast, the nonpulsatile and mean components of ChBVol_T at recovery were significantly larger than at baseline (9% and 7%).

DISCUSSION

Near-infrared LDF, with either a modified fundus camera¹⁸ or a confocal optical arrangement,¹⁵ has demonstrated its capability to quantify the response of subfoveal ChBF to various physiological stimuli. Thus, studies have assessed the effect on this flow of acute increases¹⁹ in the IOP, increases in systemic blood pressure caused by static²⁰⁻²² dynamic exercises,²³ and light-dark transitions.²⁴ The compactness and low weight of the confocal LDF flowmeter made it possible to connect this device to the tilting table and to place it directly in front of the

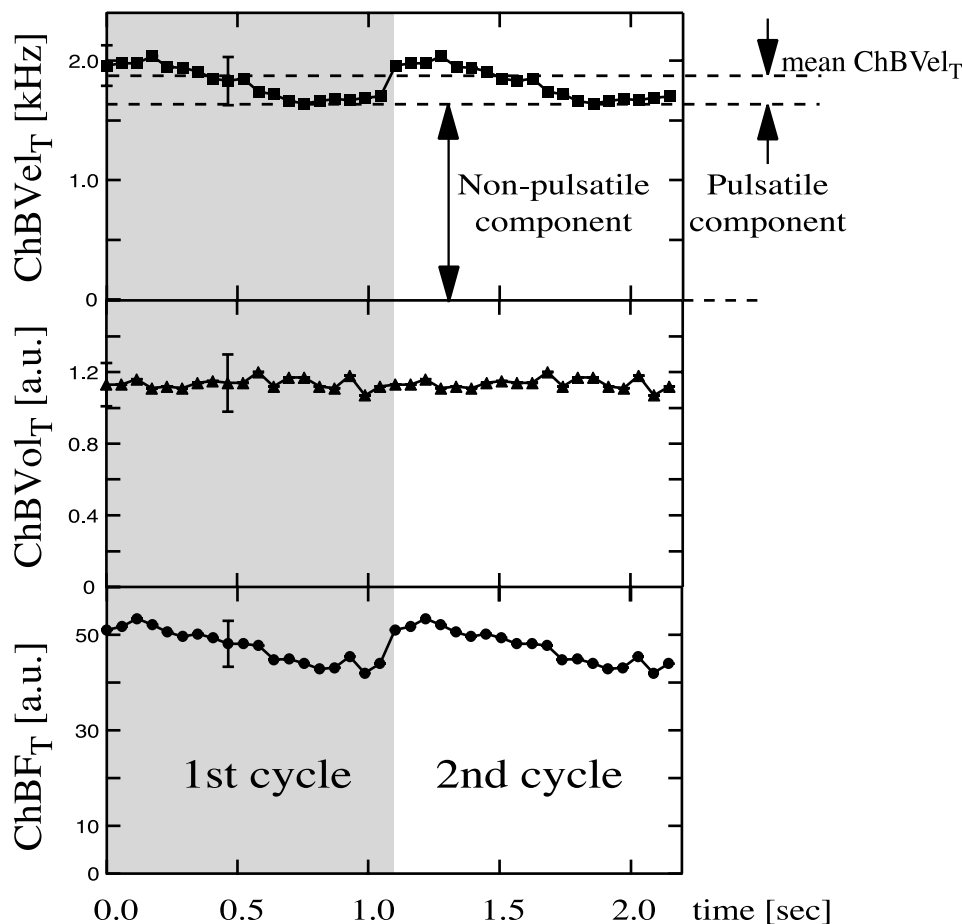


FIGURE 2. LDF measurements during a period (T) were averaged over the heart cycle. This average is shown during two cycles for the three LDF parameters. In this case, T was 30 seconds. The pulsatile and nonpulsatile components are shown for ChBVel_T. The pulsatile component of ChBVol_T was not apparent.

subject's eye, allowing measurements to be made continuously during tilting.

Tilting the body from standing to supine changed the systemic parameters in accordance with previously reported findings.^{3,9} Quantitatively, the decrease in BP_{diast} of 7.4% is similar to the 6.6% found by Evans et al.¹¹ and the 4.4% reported by Sayegh and Weigelin.³ Confirming previous findings, we found no change in BP_{syst}. The 15.8% decrease in heart rate in our study (82–69 bpm) is practically identical with that found by James and Smith¹³ (70–59 bpm) but larger than the 10.3% and 7.9% reported by Savin et al.¹⁰ (77–69 bpm) and Evans et al.¹¹ (76–70 bpm), respectively. The differences between our study and the data of others are, most probably, only of statistical nature.

Regarding the IOP, Kothe⁹ has summarized the changes in IOP occurring between upright and supine obtained by previous investigators. These changes ranged from 0.5 to 4.4 mm Hg. Thus, the average 4-mm Hg increase observed in our subjects lies within this range.

TABLE 1. Group Averaged Brachial Artery BP, IOP, and HR at the Different Postures

Postures	BP _{syst} (mm Hg)	BP _{diast} (mm Hg)	BP _m (mm Hg)	IOP (mm Hg)	HR (b/min)
Baseline	116 ± 15	81 ± 7	96 ± 10	13 ± 1	82 ± 13
Supine	124 ± 13	75 ± 6*	95 ± 8	17 ± 2†	69 ± 10‡
Recovery	117 ± 15	81 ± 6	96 ± 9	—	80 ± 11

* $P < 0.0016$.

† $P < 0.001$.

‡ $P < 0.0004$.

According to Friberg et al.²⁵ this increase in IOP between standing and supine postures appears to be closely related to increased venous pressure in the orbit and possibly to increased choroidal blood volume. This increase in volume may not translate into an increase in ChBVol, because it probably occurs in the large vessels rather than in the choriocapillaris—the region sampled by LDF.

Our results demonstrate a significant increase in the blood volume at recovery. The reason for this increase is not clear. Longer measurements during recovery may help determine the duration of this effect and whether it is due to some spatial redistribution of blood after the decrease in IOP on the return to the standing position.

The tilting of the body from standing to supine increased ChBF by an average of 11%. This increase was mainly due to a statistically significant 8% change in the velocity. This flow increase was entirely reflected in the increase of the nonpulsatile component of the flow. For each LDF parameter, the change in the pulsatile component was not significant, most probably because this component is much smaller than the nonpulsatile component, resulting in a greater variability of its measurement. The reason for this is that the LDF signal originates predominantly from the choriocapillaris,¹⁸ where pulsatility is markedly attenuated compared with that in the choroidal arteries.

Pulsatile ocular blood flow, as measured by POBF, decreases from the seated to the supine position.^{12,13,26} Based on data compiled by Kothe,⁹ we calculated a mean decrease of pulsatile ocular blood flow of 19% ± 5% from the standing or sitting to the supine posture. We presume, as mentioned before, that the pulsatile component of ChBF in our measurement was too low (11.2%) to reveal such an effect. By comparison, the POBF

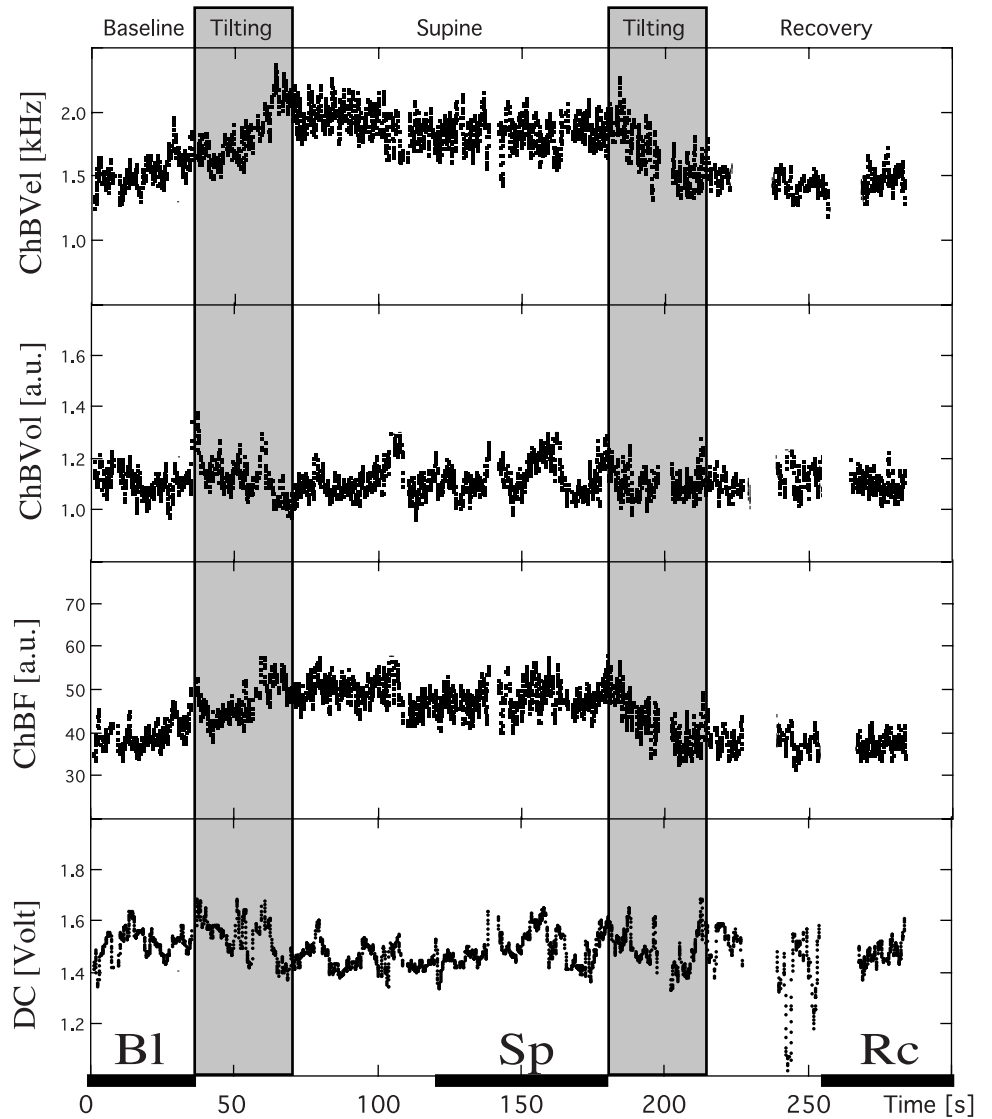


FIGURE 3. Representative recordings of the LDF parameters and the DC obtained from the subfoveal choroidal region in one subject. Horizontal bars on the time scale show the time span (T) during which the LDF parameters were averaged for further analysis. The data were displayed with a running average over four successive measurements, each separated by a time interval of 0.058 second.

technique is based on the pulsatility in the large vessels which is markedly higher than in the choriocapillaris. Thus, based on the value of the resistance index (RI; [(peak systolic velocity - peak diastolic velocity)/peak systolic velocity]) of 0.68 that was found by Kaiser et al.²⁷ in the short and long posterior ciliary arteries, we estimated that the pulsatile component of the velocity in these vessels is approximately 55% of the mean velocity.

James and Smith¹³ have assumed that the decrease in pulsatile ocular blood flow is associated with an increase of the nonpulsatile part. Our results confirm this increase in nonpulsatile flow, which is primarily due to an increase in velocity. An increase in velocity was also observed in the ophthalmic artery by Doppler ultrasonography.¹²

Mean ocular perfusion pressure is defined as $OPP_m = OABP_m - IOP$, where $OABP_m$ is the mean ophthalmic artery

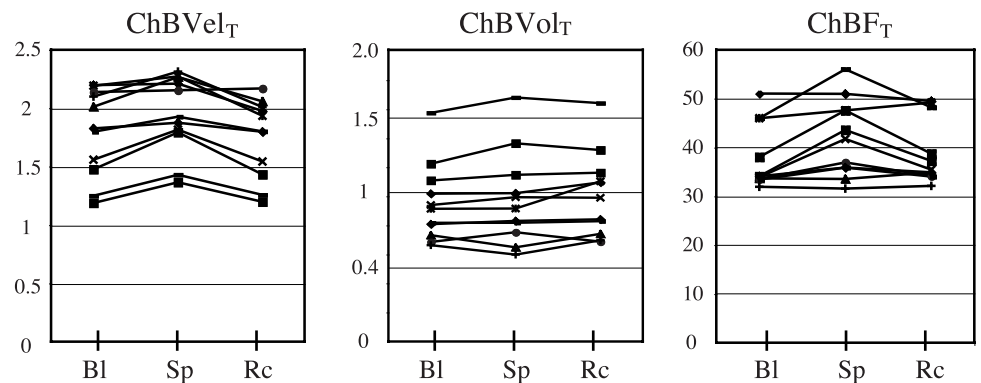


FIGURE 4. Mean value of $ChBVel_T$ (kilohertz), $ChBVol_T$, and $ChBF_T$ (both in arbitrary units; defined in Fig. 2) for each subject during baseline (Bl), supine (Sp), and recovery (Rc) positions (see Fig. 3).

TABLE 2. Percent Changes in the Magnitude of the Pulsatile, Mean and Nonpulsatile Components of ChBVel_T, ChBVol_T, and ChBF_T between Baseline and Supine and Baseline and Recovery

	Supine Versus Baseline			Recovery Versus Baseline		
	Nonpulsatile	Mean	Pulsatile	Nonpulsatile	Mean	Pulsatile
ChBVel _T	8 ± 8*	8 ± 6†	7 ± 32	-3 ± 7	-3 ± 6	5 ± 22
ChBVol _T	4 ± 10	3 ± 7	-11 ± 34	9 ± 34‡	7 ± 10‡	-16 ± 32
ChBF _T	12 ± 11†	11 ± 11†	-15 ± 40	3 ± 6	4 ± 7	8 ± 28

* $P < 0.03$.

† $P < 0.01$.

‡ $P < 0.05$.

pressure. In this study, OABP_m was not measured. Therefore, to assess OPP_m in standing and supine postures, we used values of OABP_m based on previous studies, involving postural changes, but where ophthalmodynamometric measurements were performed in healthy volunteers under these conditions. From the data of two studies of Sayegh and Weigelin^{3,28} performed on a total of 254 normal volunteers, we derived a relationship between OABP_m and BP_m. In upright position OABP_m = 0.74 · BP_m, and in supine position OABP_m = 0.84 · BP_m. Applying these values to our BP_m and IOP data, we obtain OPP_m = 57 mm Hg in the standing and 63.6 mm Hg in the supine position—that is, an increase in OPP_m of 11.6% between these two postures. This value is very similar to the 11% change observed in our study and suggests some passive response of ChBF to the increase in OPP_m.

Bill²⁹ has theoretically assessed the expected OPP_m in the standing and supine positions in normal subjects. From his analysis, OPP_m = BP_m - ΔP_r - ΔP_h - IOP. ΔP_r is the loss of pressure (assumed by Bill to be 5 cm H₂O, i.e., approximately 4 mm Hg), because of the flow resistance of the vessels between the heart and the eye. ΔP_h is the static pressure difference of a water column with a height equal to the distance between the heart and the eye (assumed by Bill to be 40 cm, i.e., 29 mm Hg). The BP_m and IOP data in Bill's study, when converted were 103 and 13 mm Hg, respectively, in upright posture and 96 and 14.8 mm Hg, respectively in the supine position (eyes 10 cm above the level of the heart). These values result in a change in OPP_m between upright and supine from 57 to 70 mm Hg (i.e., an increase of 23%).

Applying this analysis to our data and taking into account that in our test, in the supine position, the eyes were approximately 3 cm below the heart (which reduces the OABP_m by approximately 4 mm Hg, instead of the 7 mm Hg in Bill's analysis), we found that OPP_m = 59 mm Hg in the standing and 79 mm Hg in the supine position—that is, an increase of approximately 34%.

If Bill's model were representative of the events occurring in the body, the change in ChBF of only 12% in the face of a 34% increase in OPP_m would inevitably lead to the conclusion that an active mechanism is operating to increase choroidal vascular resistance to maintain ChBF in the supine posture close to that in standing position. The ophthalmodynamometric data of Sayegh and Weigelin,³ however, suggest that some compensatory mechanism is already acting between the heart and the eye to buffer most of the increase in the blood pressure induced by the tilting from upright to supine. This could occur in the ophthalmic artery or even at the level of the internal and common carotid arteries.¹⁰

In conclusion, our results show that mean subfoveal ChBF increases significantly (by an average of 11%) when the body is tilted from upright to supine position. This increase, which is primarily due to the nonpulsatile component of this flow, results from an increase in blood velocity. Because experimental data indicate that the ocular perfusion pressure increases by

a similar percentage, which is less than expected based on purely hydrostatic considerations (Bill's model), the response of subfoveal ChBF can be explained by the passive response of the choroidal vascular system to postural change.

Acknowledgments

The authors thank Dominique Fournier, radiologist, for the opportunity to use the tilting table.

References

- Anderson DR, Grant WM. The influence of position on intraocular pressure. *Invest Ophthalmol Vis Sci.* 1973;12:204-212.
- Kriegelstein GK, Waller WK, Leydhecker W. The vascular basis of the positional influence of the intraocular pressure. *Albrecht Von Graefes Arch Klin Exp Ophthalmol.* 1978;206:99-106.
- Sayegh FN, Weigelin E. Functional ophthalmodynamometry: comparison between brachial and ophthalmic blood pressure in sitting and supine position. *Angiology.* 1983;34:176-182.
- Hague S, Hill DW. Postural changes in perfusion pressure and retinal arteriolar calibre. *Br J Ophthalmol.* 1988;72:253-257.
- Baer RM, Hill DW. Retinal vessel responses to passive tilting. *Eye.* 1990;4:751-756.
- Mader TH, Taylor GR, Hunter N, Caputo M, Meehan RT. Intraocular pressure, retinal vascular, and visual acuity changes during 48 hours of 10 degrees head-down tilt. *Aviat Space Environ Med.* 1990;61:810-813.
- Baxter GM, Williamson T H, McKillop G, Dutton GN. Color Doppler ultrasound of orbital and optic nerve blood flow: effects of posture and timolol 0.5%. *Invest Ophthalmol Vis Sci.* 1992;33:604-610.
- Lovasik JV, Kothe AC, Kergoat H. Comparison of noninvasive methods to derive the mean central retinal artery pressure in man. *Optom Vis Sci.* 1993;70:1005-1011.
- Kothe AC. The effect of posture on intraocular pressure and pulsatile ocular blood flow in normal and glaucomatous eyes. *Surv Ophthalmol.* 1994;38(suppl):191-197.
- Savin E, Bailliart O, Checoury A, Bonnin P, Grossin C, Martineaud JP. Influence of posture on middle cerebral artery mean flow velocity in humans. *Eur J Appl Physiol Occup Physiol.* 1995;71:161-165.
- Evans DW, Harris A, Garrett M, Chung HS, Kagemann L. Glaucoma patients demonstrate faulty autoregulation of ocular blood flow during posture change. *Br J Ophthalmol.* 1999;83:809-813.
- Trew DR, Smith SE. Postural studies in pulsatile ocular blood flow: I Ocular hypertension and normotension. *Br J Ophthalmol.* 1991;75:66-70.
- James CB, Smith SE. Pulsatile ocular blood flow in patients with low tension glaucoma. *Br J Ophthalmol.* 1991;75:466-470.
- Geiser MH, Diermann U, Riva CE. Compact laser Doppler choroidal flowmeter. *J Biomed Opt.* 1999;4:459-464.
- Petrig BL, Riva CE. New continuous real-time analysis system for laser Doppler flowmetry and velocimetry in the ocular fundus using a digital signal processor. *Vision Science and Its Applications. Technical Digest Series.* Vol. 2. Santa Fe, NM: Optical Society of America; 1994:238-241.

16. Movaffaghy A, Chamot SR, Petrig BL, Riva CE. Blood flow in the human optic nerve head during isometric exercise. *Exp Eye Res.* 1998;67:561-568.
17. Petrig BL, Riva CE. Laser Doppler flowmetry in the optic nerve head: principle and technique. In: Pillunat LE, Harris A, Anderson DR, Greve EL, eds. *Current Concepts on Ocular Blood Flow in Glaucoma*. The Hague, The Netherlands: Kugler; 1999:171-182.
18. Riva CE, Cranstoun SD, Grunwald JE, Petrig BL. Choroidal blood flow in the foveal region of the human ocular fundus. *Invest Ophthalmol Vis Sci.* 1994;35:4273-4281.
19. Riva CE, Titze P, Hero M, Petrig BL. Effect of acute decreases of perfusion pressure on choroidal blood flow in humans. *Invest Ophthalmol Vis Sci.* 1997;38:1752-1760.
20. Riva CE, Titze P, Hero M, Movaffaghy A, Petrig BL. Choroidal blood flow during isometric exercises. *Invest Ophthalmol Vis Sci.* 1997;38:2338-2343.
21. Gugleta K, Orgül S, Hasler PW, Picornell T, Gherghel D, Flammer J. Choroidal vascular reaction to hand-grip stress in subjects with vasospasm and its relevance in glaucoma. *Invest Ophthalmol Vis Sci.* 2003;44:1573-1580.
22. Luksch A, Polska E, Imhof A, et al. Role of NO in choroidal blood flow regulation during isometric exercise in healthy humans. *Invest Ophthalmol Vis Sci.* 2003;44:734-739.
23. Lovasik JV, Kergoat H, Riva CE, Petrig BL, Geiser M. Choroidal blood flow during exercise-induced changes in the ocular perfusion pressure. *Invest Ophthalmol Vis Sci.* 2003;44:2126-2132.
24. Longo A, Geiser MH, Riva CE. Subfoveal choroidal blood flow in response to light-dark exposure. *Invest Ophthalmol Vis Sci.* 2000;41:2678-2683.
25. Friberg TR, Sanborn G, Weinreb RN. Intraocular and episcleral venous pressure increase during inverted posture. *Am J Ophthalmol.* 1987;103:523-526.
26. Lovasik JV, Kergoat H. Gravity-induced homeostatic reactions in the macular and choroidal vasculature of the human eye. *Aviat Space Environ Med.* 1994;65:1010-1014.
27. Kaiser HJ, Schotzau A, Flammer J. Blood-flow velocities in the extraocular vessels in normal volunteers. *Am J Ophthalmol.* 1996;122:364-370.
28. Sayegh FN, Weigelin E. Functional ophthalmodynamometry: comparison between dynamometry findings of healthy subjects in sitting and supine positions. *Ophthalmologica.* 1983;187:196-201.
29. Bill A. Physiological aspects of the circulation in the optic nerve. In: Heilmann K, Richardson KT, eds. *Glaucoma Conceptions of a Disease Pathogenesis, Diagnosis Therapy*. Stuttgart: Georg Thieme Publishers; 1978:97-103.