On Pulse-Wave Propagation in the Ocular Circulation

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PURPOSE. To measure the oscillation phase delay between retinal arterioles and venules in order to analyze pulse wave propagation in the ocular circulation of vasospastic and nonvasospastic subjects and a change thereof during the cold pressor test in another group of healthy subjects.

METHODS. Twenty-four young, healthy women, 12 vasospastic and 12 nonvasospastic, were analyzed. A retinal vessel analyzer was used to obtain 1-minute recordings of the ocular fundus. A phase delay between the arteriole and venule pulsations was assessed at three sites, one (proximal) in the close retinal vicinity of the disc, one (middle) 1 to 2 disc diameters away from the disc, and a third (distal) 3 to 4 disc diameters away from the disc; and, assuming that venules are counterphased to the choroidal circulation, a choroid-to-retina pulse delay was calculated. In addition, the change in these parameters was analyzed during the modified cold-pressor test in 10 healthy subjects (five women, five men).

RESULTS. Pulse oscillations in arterioles led those in venules by 95.0° ± 39.0°, 60.5° ± 57.5°, and 47.5° ± 64.0° in vasospastic subjects, and 76.0° ± 58.0°, 31.5° ± 60.0°, and 2.5° ± 80.5° in nonvasospastic subjects in the proximal, middle, and distal measuring sites, respectively. Calculated choroid-to-retina pulse delays in vasospastic subjects were 0.20 ± 0.10, 0.28 ± 0.14, and 0.30 ± 0.11 seconds and in nonvasospastic subjects 0.25 ± 0.15, 0.35 ± 0.11, and 0.43 ± 0.2 seconds at the proximal, middle, and distal measuring sites, respectively. The difference was significant between vasospastic and nonvasospastic subjects (P = 0.035) and among the measuring sites (P = 0.0023). During exposure to cold, the choroid-to-retina pulse delays changed from 0.31 ± 0.08, 0.40 ± 0.16, and 0.51 ± 0.26 seconds to 0.26 ± 0.12, 0.30 ± 0.10, and 0.33 ± 0.14 seconds at the proximal, middle, and distal measuring sites, respectively (P = 0.024 for the change from baseline to cold exposure, and P = 0.022 for measuring sites).

CONCLUSIONS. Retinal vessels in vasospastic subjects demonstrate an altered pattern of oscillation phase delay between arterioles and venules. Vessels in vasospastic subjects seem to conduct pulse waves faster and are thus stiffer than those in nonvasospastic subjects. The pattern of oscillation demonstrates changes during the cold pressor test in healthy subjects, indicating faster pulse-wave propagation. (Invest Ophthalmol Vis Sci. 2006;47:4019–4025) DOI:10.1167/iovs.06-0168

Structural changes in the retinal vasculature have long been thought to represent important predictors of systemic vascular disease.1 In contrast, arterial stiffness and pulse-wave velocity alterations have been suggested as cardiovascular risk factors.2–5 Arterial stiffness and pulse-wave velocity are increased with age6 and in coronary artery disease,7 myocardial infarction,8 heart failure,9 stroke,10 and hypertension,11 but also in patients with branch retinal vein occlusion.12 Evaluation of vascular pulsations in the eye has been mostly limited to the choroidal circulation.11–14 In the optic nerve head, retinal, and choroidal circulation, using the method described by Petrig and Riva,15 one can capture pulsations of the blood flow in a single location. Venous pulsations in the retina were analyzed in fundus photographs taken at various times during the heart cycle.16,17 An actual pulse-wave propagation from the heart to the ophthalmic artery and choroidal circulation has been estimated at 4.08 m/s in a study of healthy subjects by Michelson et al.18 Retinal vessels are not accessible for direct pulse measurements, and indirect measurements are hindered by the fact that the choroidal circulation constitutes most of the ocular blood flow19 and determines the ocular pulse amplitude.11,20 The retinal vessel analyzer (Retinal Vessel Analyzer [RVA]; IMEDOS GmbH, Weimar, Germany) offers high spatial vessel width resolution21; high reproducibility of measurements22,23; and, with high temporal resolution down to 40 milliseconds,21,24 the possibility of obtaining simultaneous measurements of retinal arterioles and venules. In contrast, biomechanical properties of blood vessels seems to be altered if vasospastic propensity is present.25–27 To investigate a potential yield of new information, in the present study, we analyzed phase delay between retinal arterioles and venules with RVA and calculated pulse delay between the retinal and choroidal circulation in vasospastic and nonvasospastic subjects. In addition, we analyzed changes in these parameters during the modified cold pressor test.

METHODS

Subjects

Forty healthy nonsmoking women were screened for the study. After approval by the ethics committee, we obtained informed consent from the subjects, in accordance with the guidelines of the Declaration of Helsinki. A notification in the University Eye Clinic of Basel informed potential volunteers (collaborators, students, parents, and friends of patients) of the opportunity to participate in a scientific research project. Subjects were screened for ocular and systemic diseases. A detailed medical and ophthalmic history was recorded, and all subjects completed an ophthalmic examination. Included were individuals with no history of ocular or systemic disease, no history of chronic or current systemic or topical medication, and no history of drug or alcohol abuse. Further inclusion criteria were a normal systolic (100–140 mm Hg) and diastolic (60–90 mm Hg) blood pressure, a best corrected visual acuity 20/25 or better in both eyes, ametropia within −3.0 to +3.0 D of spherical equivalent and less than a 1-D astigmatism in each eye, intraocular pressure (IOP) lower than 20 mm Hg in each eye by (Goldmann) applanation tonometry, and no pathologic findings in slit lamp examination and indirect funduscopy. Subjects were classified as having vasospasm if they related a clear history of frequently cold hands (answering “yes” to the questions: “do you always have cold hands, even during the summer?” and “do other people tell you that you have cold hands?”) and as healthy subjects if they reported no
such history. Vasospastic propensity or the absence of it had to be confirmed by nailfold capillaroscopy, with the examiner unaware of the history of cold hands. Subjects with contradictory findings from nailfold capillaroscopy and history or describing “sometimes having cold hands” were excluded from the present analysis. As hormonal status may influence ocular circulation, subjects with a positive history of contraceptive pill use were recruited. Subjects not taking contraceptive pills had to be in the postovulation phase of the cycle, which was verified by a subsequent phone interview ascertaining that menstrual bleeding had occurred less than 2 weeks after the study examination day.

Nailfold Capillaroscopy

Nailfold capillaroscopy was performed in a room with a constant temperature of approximately 25°C (range, 21–25°C). Before the examination, the hands of the subjects were warmed in a water bath of 40°C. The skin of the nailfold was made transparent by a drop of oil, rendering the capillaries running parallel to the skin surface and the flow of cellular elements visible under a light microscope. A microscope coupled to a television monitor was used, which was coupled to a video recorder and allowed the observed blood flow to be video-taped and analyzed offline. After baseline flow recording, the nailfold area was cooled down to 14°C to 15°C for 60 seconds by rapidly decompressing carbon dioxide (gas stream temperature, −15°C), and the occurrence and duration of blood flow standstill was recorded. A closure of one or more visible capillaries with an average stop time longer than 12 seconds was defined as a vasospastic reaction.

Retinal Vascular Diameter Measurements

Retinal vessel diameter was measured with the RVA. An essential part of the RVA device is the fundus camera (FF450; Carl Zeiss Meditec, GmbH, Jena, Germany), which allows the examination and recordings of the ocular fundus. It incorporates the illumination and the observation optical pathway. After being reflected from the retina, the light is delivered simultaneously through the observation pathway to the observation ocular and to the charge-coupled device (CCD) chip of the video camera. Two half-pictures (every other pixel line) 20 ms apart from each other are formed on the CCD nearly instantaneously and then scanned for 20 ms into the video signal. The standard video signal from the CCD then goes to the RVA control computer and to the SVHS recorder, which enables subsequent offline measurements of the recorded session later on. The measuring principle of the RVA is as follows: Inside the walls of the retinal blood vessels there is a column of red blood cells, separated from the walls by the plasma edge stream. Red blood cells absorb one part of the light. RVA measures the diameter of the column of the red blood cells. The fundus camera was adjusted to the dilated pupil and a clear fundus image with good contrast, and no reflections were visible on the monitor. Temporal resolution was set at 40 ms, which translates to 25 full video frames captured per second. These still pictures, which were taken nearly instantaneously as just described, were analyzed by the RVA software. Such an approach ensures that all vessels and in particular the two selected segments (in this case an arteriole–venule pair) were analyzed instantaneously as just described, were analyzed by the RVA software.

Experimental Procedures

After evaluation of inclusion criteria and the nailfold capillaroscopy, an RVA measurement was scheduled. The investigator performing RVA measurement was masked to the history and nailfold capillaroscopy results. Retinal vessel diameters were measured in one randomly chosen eye in the morning, after the subject had fasted for 12 hours overnight. Participants were instructed to abstain from a large meal, alcohol consumption (including alcohol-containing products and drugs), and physical exercise for 24 hours before the measurements. On the day of the experiments, the subjects were seated for 30 minutes in the laboratory, and local tropicamide was applied in one eye three times every 5 minutes for pupil dilation. Blood pressure was measured every 10 minutes for at least 30 minutes. After stabilization of blood pressure, retinal vessel diameter was assessed. The fellow eye was covered to improve fixation during imaging with the fundus camera. The image of the retina was adjusted on the screen of the real-time monitor, and a recording was taken of the inferior temporal segment.

Calculation of a Choroid–Retina Pulse Delay

An assumption was made that venule pulsations are precisely counter-phased to the choroidal circulation—the retinal venous trough mirrors the choroidal peak—and that a pulse wave of choroidal circulation leads the retinal circulation. From a given phase shift in degrees of the cycle and a corresponding frequency, we calculated the choroid-to-retina pulse delay. Two examples are shown in Figure 2. Cycle duration in seconds is defined by the frequency (1/frequency in Hz), and the delay in seconds can be calculated for each pair. A choroid-to-retina pulse-wave delay in seconds was calculated (preceding venous trough-to-arterial peak, as shown in Fig. 2) and also was analyzed with a two-way analysis of variance, with vasospastic propensity (difference between the two groups) as one factor and difference between three sites of measurement as the other.

Calculation of Pulsation Phase Delay between Retinal Arterioles and Venules

Three paired arteriole–venule measurements were analyzed offline: one (proximal) in the close retinal vicinity of the disc, one (middle) 1 to 2 disc diameters away from the disc, and a third (distal) 3 to 4 disc diameters away from the disc. Ultimately there was a 1- to 1.5-disc-diameter distance between each of the proximal, middle, and distal measuring sites along an arteriole and venule, respectively, as shown in Figure 1. The whole 1-minute recording except for the first 10 seconds, which were discarded, entered the analysis. The chosen segment length was 500 UM (corresponding to 500 μm in an emmetropic eye), with 40 vessel width measurements along this segment length (40 × 12.5 UM = 500 UM). A relatively short segment length (equivalent of 500 μm) was chosen because RVA recording is sensitive to eye movements. The shorter the segment, the fewer poorly recorded frames and missing data. In our sample, there was less than 5% of data missing. The 1250 video frames (50 seconds × 25 frames per second) with 40 vessel width measurements in each produced a time–space table with 1250 × 40 individual data points per pixel for each vessel. Each of 1250 frames was averaged, and a data series with 1250 mean vessel diameters during 50 seconds for each vessel obtained. A cross-spectrum bivariate Fourier analysis (Statistica, ver. 5.1; StatSoft Inc., Tulsa, OK) was performed on paired data series for arteriole and venule in three measuring locations. The technique measures an extent to which each frequency component of one series leads the other. The frequency peak in the range between 0.75 and 2 Hz, corresponding to the pulse rates between 45 and 120, was readily discernible in the cross-amplitude spectrum (bivariate analogue for the power spectrum in single Fourier analysis), and the lead or lag of arteriolar peak to the corresponding venule peak was obtained in degrees of the cycle. This parameter was analyzed with a two-way analysis of variance, with vasospastic propensity (difference between the two groups) as one factor and the difference between three sites of measurement as the other.
Pulse-Wave Propagation along the Entire Vascular Tree

To test a hypothesis that a pulse wave is propagated along the retinal arterial tree and the capillaries and back through the venules, an analysis of peak-to-peak phase delay was performed along the venule, between one measuring site distal to the ONH and one proximal to the ONH.

Change of Pulse Propagation in the Ocular Circulation during the Cold Exposure

To validate the data, we performed an additional experiment in 10 healthy subjects: 5 women and 5 men. We did not differentiate between subjects based on their vasospastic propensity, but instead analyzed the change of retinal vascular parameters during a modified cold pressor test. One-minute recordings of temporal inferior quadrant in one randomly selected eye per subject were taken with the RVA (same as detailed earlier), at baseline and after the ipsilateral lower arm had been immersed in the 10°C water for 5 minutes. Systemic cardiovascular parameters were recorded in the two phases of the experiment. Phase delay between arterioles and venules, and calculated choroid-to-retina pulse delay were analyzed in a two-way ANOVA model, with the change of parameter between baseline and cold exposure as one factor, and the difference between proximal, middle, and distal measuring sites as the other factor. As validity parameters, change of arteriolar and venule diameter at three measuring sites was analyzed in the same two-way ANOVA model, and blood pressure and pulse rate change were analyzed with a paired t-test.

The cold pressor test increases the aortic pulse-wave velocity and causes vasoconstriction in the retinal arterioles. An increase in blood pressure, but also an increased release of endothelin-1 during the cold pressor test, may mediate the vasoconstriction. In contrast, in the face of an acute increase of blood pressure, retinal venules show no change or a modest dilation. Choroidal circulatory response to an acute blood pressure increase is a vasoconstriction, probably mediated through endothelin-1. In the present model, we hypothesized that the exposure to cold would lead to vasoconstriction and thus to an acceleration of the pulse-wave propagation in the ocular circulation.

RESULTS

Twenty-four healthy nonsmoking women were analyzed in the study: 12 with a positive history of cold hands and feet and a positive nailfold capillaroscopy result, and 12 with a negative
Figure 2. A, arteriole peak, V, venule peak, C, venule trough, corresponding to the assumed peak of the choroidal circulation. Case 1 shows a situation in which the arteriole peak led 90° in relation to the venule peak. In this case, the retinal arteriolar peak A lagged the previous venous trough by 90°, which corresponds to the assumed peak of choroidal circulation. C. Case 2 shows a situation in which the arteriole peak lagged the venule peak by 90°. The previous venous trough, corresponding to the assumed peak of the choroidal circulation C, would in this case lead 270° in relation to the retinal arteriolar peak A.

In the similar two-way ANOVA model, calculated choroid-to-retina pulse-wave delay in seconds, reported in Table 2 and

### Table 1. Phase Shift between Retinal Arterioles and Venules at Measuring Locations in Relation to the Optic Disc

<table>
<thead>
<tr>
<th>Measurement Site</th>
<th>Vasospastic Subjects</th>
<th>Nonvasospastic Subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Proximal</td>
<td>95.0 ± 39.0</td>
<td>76.0 ± 58.0</td>
</tr>
<tr>
<td>Middle</td>
<td>60.5 ± 57.5</td>
<td>31.5 ± 60.0</td>
</tr>
<tr>
<td>Distal</td>
<td>47.5 ± 64.0</td>
<td>2.5 ± 80.5</td>
</tr>
</tbody>
</table>

Data from the three measurement sites are expressed in mean degrees of a cycle ± SD. A positive value indicates that the arteriole peak leads and the venule peak lags.

In contrast to qualitative assessment of retinal vessels with ophthalmoscopy, which has a poor reproducibility, the RVA provides highly reproducible measurements. The RVA provides highly reproducible measurements. Temporal resolution is 40 ms, spatial vessel width resolution is high, and simultaneous paired measurements of arterioles and venules are possible. An alternative method of assessing pulsatile behavior of the ocular vessels would be color Doppler

### Table 2. Calculated Choroid-to-Retina Pulse Delay

<table>
<thead>
<tr>
<th>Measurement Site</th>
<th>Vasospastic Subjects</th>
<th>Nonvasospastic Subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Proximal</td>
<td>0.20 ± 0.10</td>
<td>0.25 ± 0.15</td>
</tr>
<tr>
<td>Middle</td>
<td>0.28 ± 0.14</td>
<td>0.35 ± 0.11</td>
</tr>
<tr>
<td>Distal</td>
<td>0.30 ± 0.11</td>
<td>0.43 ± 0.20</td>
</tr>
</tbody>
</table>

Data are mean seconds ± SD.
we recruited only female subjects in the first part of the study. When we attempted optimal separation between the vasospastic and nonvasospastic group, only subjects who had a clear positive history of cold hands confirmed by a positive nailfold capillaroscopy result were included in the vasospastic group and vice versa. In contrast, groups were balanced in age, blood pressure and IOP, and contraceptive pill history and menstrual cycle, and standardized vessel segments were measured in both groups. The group in the second experiment had a balanced gender distribution.

We assumed that the venous pulsations are a consequence of IOP pulsations, and these in turn are the result of choroidal pulsations. Indeed, even if more complex explanations of the origin of proximal venule pulsations involving cerebrospinal fluid pressure held true, synchronization with IOP and thus with the choroidal circulation was still acknowledged. Venous diameter decreases in early systole, increasing thereafter to a maximum level in early diastole and then declines toward end diastole, whereas arterial diameter peaks in mid to late systole. Central retinal vein blood flow velocity is synchronized with the ocular pulse amplitude and IOP. An alternative explanation for our initial finding of centrifugally decreasing peak-to-peak venous-to-arterial delay is that the pulse wave actually propagates along the retinal arterial tree, the capillaries, and back through the venules, producing in fact centrifugal increase in peak-to-peak venous-to-arterial delay. Although this seems unlikely, because different length of capillaries tend to neutralize the pulse by ways of interference, to investigate, we tested a phase delay between proximal and distal venules. No relevant phase delay was found, thus indicating that venous pulsations are synchronous and paced by the same source, probably the choroidal circulation, as just discussed.

Another assumption that we made was that the pulse wave of the choroidal circulation leads that of retinal circulation. Although an angiography captures an actual movement of the volume of blood, whereas in this study a parameter of interest was the pressure-volume pulse wave, it is noteworthy that in healthy subjects during angiography dye-filling of the choroid precedes that in the retina. Of note, we also analyzed the alternative assumption, that retinal pulse wave leads that of

### Table 3. Results of the Cold Exposure Experiment

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Baseline</th>
<th>During the Cold Exposure</th>
<th>Statistics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phase shift between arterioles and venules (units as in Table 1)</td>
<td></td>
<td></td>
<td>Two-way ANOVA:</td>
</tr>
<tr>
<td>Proximal</td>
<td>57.8 ± 26.7</td>
<td>78.6 ± 39.8</td>
<td>Factor 1: $P = 0.016$</td>
</tr>
<tr>
<td>Middle</td>
<td>22.4 ± 59.4</td>
<td>60.6 ± 28.5</td>
<td>Factor 2: $P = 0.014$</td>
</tr>
<tr>
<td>Distal</td>
<td>-19.5 ± 95.1</td>
<td>49.7 ± 45.5</td>
<td>Interaction: $P = 0.22$</td>
</tr>
<tr>
<td>Calculated choroid-to-retina pulse delay (units as in Table 2)</td>
<td></td>
<td></td>
<td>Two-way ANOVA:</td>
</tr>
<tr>
<td>Proximal</td>
<td>0.31 ± 0.08</td>
<td>0.26 ± 0.12</td>
<td>Factor 1: $P = 0.024$</td>
</tr>
<tr>
<td>Middle</td>
<td>0.40 ± 0.16</td>
<td>0.30 ± 0.10</td>
<td>Factor 2: $P = 0.022$</td>
</tr>
<tr>
<td>Distal</td>
<td>0.51 ± 0.26</td>
<td>0.33 ± 0.14</td>
<td>Interaction: $P = 0.23$</td>
</tr>
<tr>
<td>Diameter of arterioles (units equivalent to μm in an emmetropic eye)</td>
<td></td>
<td></td>
<td>Two-way ANOVA:</td>
</tr>
<tr>
<td>Proximal</td>
<td>115.3 ± 12.7</td>
<td>108.3 ± 15.7</td>
<td>Factor 1: $P = 0.022$</td>
</tr>
<tr>
<td>Middle</td>
<td>108.5 ± 15.5</td>
<td>105.5 ± 16.6</td>
<td>Factor 2: $P = 0.007$</td>
</tr>
<tr>
<td>Distal</td>
<td>100.6 ± 13.3</td>
<td>98.0 ± 14.5</td>
<td>Interaction: $P = 0.27$</td>
</tr>
<tr>
<td>Diameter of venules (units equivalent to μm in an emmetropic eye)</td>
<td></td>
<td></td>
<td>Two-way ANOVA:</td>
</tr>
<tr>
<td>Proximal</td>
<td>147.9 ± 14.5</td>
<td>145.8 ± 11.3</td>
<td>Factor 1: $P = 0.47$</td>
</tr>
<tr>
<td>Middle</td>
<td>144.7 ± 18.8</td>
<td>145.9 ± 18.9</td>
<td>Factor 2: $P = 0.0009$</td>
</tr>
<tr>
<td>Distal</td>
<td>123.1 ± 21.9</td>
<td>132.1 ± 28.9</td>
<td>Interaction: $P = 0.39$</td>
</tr>
<tr>
<td>Systolic blood pressure (mmHg)</td>
<td>124.5 ± 15.2</td>
<td>147.5 ± 27.7</td>
<td>Paired t-test: $P = 0.005$</td>
</tr>
<tr>
<td>Diastolic blood pressure (mmHg)</td>
<td>74.5 ± 8.4</td>
<td>87.5 ± 12.4</td>
<td>Paired t-test: $P = 0.0003$</td>
</tr>
<tr>
<td>Pulse rate (bpm)</td>
<td>69.6 ± 11.0</td>
<td>70.3 ± 11.7</td>
<td>Paired t-test: $P = 0.76$</td>
</tr>
</tbody>
</table>

All parameters are presented as the mean ± SD. In the two-way ANOVA factor 1 is the change of parameter between baseline and cold exposure and factor 2 is the difference between the proximal, middle, and distal measuring sites. Blood pressure and pulse rate changes were analyzed with a paired t-test.
Data analysis showed more rigid vessels in response to cold stimulus.

Calculated pulse delays between the choroidal and retinal circulation translate to pulse-wave velocity in the magnitude of centimeters per second, which is concordant with the literature regarding microcirculation.\(^1\) Pulse-wave velocity in the macrocirculation is expressed in meters per second. For example, Michelson et al.\(^2\) found the calculated pulse-wave velocity between the heart and ophthalmic artery to be 4.08 m/s; in the microcirculatory department, however, the iridal blood-velocity pulse curve started 0.24 seconds after the heart beat (the R-peak in electrocardiogram) and took another 0.23 seconds to reach its maximum.

Caution is warranted in interpreting the present results. Indeed, RVA measures the diameter of the red blood cell column in a vessel. It is not clear how accurately dynamic changes of the blood cell column reflect the wall vasomotion. Yet, a primary parameter of interest in the present study was the pulse phase and not an amplitude or a shape of vessel wall excursion during the pulse cycle.

We assumed that retinal venule pulsations are precisely counterphased to the pulsations of the choroidal circulation, which may not be true, and there could be a certain delay in the chain of transmission between the choroidal pulse, IOP peak, and compression of venules. However, this does not explain either the observed difference between vasospastic and nonvasospastic subjects or the changes during the exposure to cold stimulus.

In the present study, we did not measure ocular pulse amplitude. If it had been significantly different between the two groups, this could have affected the retinal pulsatile behavior differently. There are indications, however, that the ocular pulse amplitude is unaltered in vasospastic subjects.\(^3\) In contrast, there are indications that biomechanical vessel properties of persons with a tendency toward vasospasm are altered\(^4\)–\(^6\); hence, there is a rationale for studying retinal arterial stiffness in otherwise healthy vasospastic subjects.

The present study is a preliminary report on new potential use of the RVA. Additional validation of the method is prudent, such as in studies with a vasospastic (hyperoxia) or vasodilatory challenge (hypercapnia, calcium channel blockers), before an application in the clinical setting can be envisaged. We used venules as a time reference. Using a similar approach, one could quantify the pulse propagation between two points in the retinal arterial tree itself, and obtain a pulse-wave velocity in centimeters per second. For this, however, contrary to the present study, a correction for magnification factor would be necessary. Relevant parameters in our study are expressed either in degrees or in seconds and require no such correction. Analysis of diameter of arterioles and venules in the second experiment was used to indicate whether a local response to cold stimulus was present. Furthermore, the refractive power of the eyes probably remained stable during the exposure to cold, and thus the change in vessel diameters was of interest, rather than the baseline values.

In conclusion, the RVA can be used to investigate oscillation phase delay between retinal arterioles and venules. An altered pattern of arteiole-venule pulse delay was detected in vasospastic subjects, indicating stiffer vessels than in the control nonvasospastic group. Exposure to cold in healthy subjects seems to induce faster pulse-wave propagation in the ocular circulation.

References


