Influences of Atmospheric Pressure and Temperature on Intraocular Pressure

Sara Van de Veire, Peter Germonpre, Charlotte Renier, Ingeborg Stalmans, and Thierry Zeyen

PURPOSE. To determine whether the atmospheric pressure (ATM) change experienced during diving can induce changes in the intraocular pressure (IOP) of eyes in a normal population.

METHODS. The IOP of 27 healthy volunteers (ages, 23.8 ± 4.9 years; range, 18–44) was measured with a Perkins applanation tonometer by two independent investigators who were masked to the previous measurements. Measurements were taken at baseline (normal ATM, 1 Bar and 24°C), at 28°C and 24°C after the ATM was increased to 2 Bar in a hyperbaric chamber, at baseline again, and finally at the normal ATM of 1 Bar but a temperature of 28°C. Multivariate regression analysis was used to evaluate the results.

RESULTS. The mean IOP decreased significantly from 11.8 mm Hg in the right eye (RE) and 11.7 mm Hg in the left eye (LE) at 1 Bar to 10.7 mm Hg (RE) and 10.3 mm Hg (LE) at 2 Bar (P = 0.024, RE; P = 0.0006, LE). The IOP decrease remained constant during the ATM increase period (40 minutes) and was independent of the temperature change. The temperature increase alone did not significantly influence the IOP.

CONCLUSIONS. An increase of the ATM to 2 Bar (equal to conditions experienced during underwater diving at 10 meters) modestly but significantly decreased the IOP independently of the temperature change. During the period of increased ATM (60 minutes), the IOP decrease remained stable and was independent of blood pressure change or corneal thickness. (Invest Ophthalmol Vis Sci. 2008;49:5392–5396) DOI:10.1167/iovs.07-1578

Glaucoma is the second leading cause of irreversible blindness worldwide and affects approximately 70 million people, of whom 7 million are blind.1,2 Elevated intraocular pressure (IOP) is widely regarded as the most important modifiable risk factor associated with the development and progression of glaucomatous optic neuropathy. Little is known about the effects of external factors such as atmospheric pressure (ATM) and surrounding temperature (T) on the IOP. Rather than looking at changes in IOP, most published reports on the effects of high altitude (low ATM) focus on high-altitude retinal hemorrhage3–5 or on systemic side effects, such as increased blood pressure,6,7 and on cardiac side effects,8 which lead to mountain sickness. However, one study does mention that a higher baseline IOP is a significant risk factor for altitude retinopathy.9

IOP at high altitude has been the subject of controversy for many years. Ninety years ago, Wilmer and Berens10 measured the IOP of 14 aviators in a hypobaric chamber, but no significant changes were found. More recently, the effect of decreased ATM on IOP has been studied by several groups; however, their results are conflicting. Some groups have observed a decrease in IOP,11 some have found either an increase in IOP12,13 or no change in IOP14,15 and some have observed a reduction in IOP that occurs within hours of ascent and subsequently recovers to baseline levels during acclimatization.16,17 The mechanisms of these changes remain unknown, but many different explanations have been suggested. Some investigators speculate that increases in IOP could result from altered intracranial pressure16,17 or corneal thickness.20 Other studies support the idea that lowered IOP may occur with hypobaric hypoxia acclimatization11,16,17 or may be an effect of retinal vasodilatation.21

This controversy may be partially attributable to the different methodologies used in each of these studies. Some investigators examined subjects only several days before departure to and after descent from high altitude; in these cases, the IOP always returned to baseline levels but could have been even lower with prolonged exposure to altitude.22 Furthermore, most studies used a portable device (Perkins or Tonopen) to measure IOP,14,21 but in a few experiments this measurement was taken at low altitude using the weight-dependent and, hence, altitude-dependent Schiotz tonometer.18

Reports on the effect of increased ATM on IOP are rare. Ersanli et al.23 observed that exposure to hyperbaric oxygen therapy at 2.5 Bar significantly reduced IOP, which was indirectly attributed to hypoxic vasoconstriction. Although a matter of controversy, increased IOP has been observed with decreased ATM,15 but it remains to be established whether this same effect occurs during ATM increase without pure oxygen breathing.

The aim of this prospective study was to investigate the effect of ATM and temperature increase on the IOP of healthy eyes. This study was inspired primarily by frequent inquiries from glaucoma patients about the potential deleterious effects of underwater diving, airplane travel, and mountaineering on IOP.

MATERIALS AND METHODS

Our sample population, consisting of 27 healthy, nonsmoking subjects (54 eyes; subject ages, 23.8 ± 4.9 [range, 18–44] years; male/female ratio, 1.25) was randomly divided into two groups. None of the subjects had any ophthalmologic disorder apart from a refractive correction of less than ± 4 D with glasses or contact lenses. Physical pre-examination consisted of standard intake examination for hyperbaric treatments, including plain chest x-ray, tonal audiometry, and resting electrocardiography. Ophthalmologic examination consisted of biomicroscopy, nonindilated fundoscopy, and central corneal thickness (CCT) measurement. All subjects were treated in accordance with the Declaration of Helsinki. They were asked to fill out a general health

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Submitted for publication December 10, 2007; revised June 3 and July 9, 2008; accepted October 8, 2008.

Disclosure: S. Van de Veire, None; P. Germonpre, None; C. Renier, None; I. Stalmans, None; T. Zeyen, None.

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questionnaire and to sign an informed consent form. Ethics committee approval was obtained. The aim of our experiment was to determine whether there is a correlation between ATM and temperature on IOP in a healthy population. To accomplish a reproducible increase in experimental ATM, we used a hyperbaric chamber and stabilized all other parameters, including temperature. We also looked for a correlation between fluctuations of the surrounding temperature and the IOP. For this, we stabilized the ATM and increased the temperature.

We noninvasively measured the IOP in both eyes with applanation tonometry (portable Perkins tonometer; Clement-Clarke International, Harlow, Essex, UK) under topical anesthesia (Unicaine; Bournonville Pharma, Braine-l’Alleud, Belgium) and with locally applied fluorescent strips. The Perkins tonometer evaluates IOP (using a small applanation pin) with a minimal weight mass and has the ability to measure the IOP of a subject in an upright position inside the hyperbaric chamber. IOP was measured in five different settings by two independent investigators. Measurements were alternated for each investigator (investigators A and B, and with alternately monitored groups A and B). A third independent observer registered all the measurements so that the investigators were masked to the previous measurements.

We started measuring IOP at baseline with a normal ATM of 1 Bar and an ambient temperature of 24°C. Pressure exposure was accomplished by placing our volunteers in a clinical multipurpose hyperbaric chamber (Starmed 2800; Haux, Karlsbad, Germany) with microprocessor-controlled pressure and temperature levels. During compression, adiabatic phenomena led to a temperature increase, which, when combined with the air-conditioning system of the chamber, resulted in a surrounding temperature of 28°C once the 2 Bar level was reached. During the pressure ‘plateau,’ the temperature could be modified independently of the pressure. After the first measurements, the chamber was gradually cooled to the baseline temperature of 24°C. Oxygen and humidity levels were kept constant throughout at 21% and 40%, respectively, during exposure, and the noise level never exceeded 85 dB. During the experiment, all subjects remained seated, and no physical exercise was performed.

During compression, some subjects had to perform repeated Valsalva maneuvers to equalize their middle ear pressure to the surrounding pressure. The rate of pressure increase was low (0.1 Bar/min), allowing for easy equalization in all but one subject. All subjects succeeded in equalizing their middle ears using only very light and brief (nonstrenuous) Valsalva maneuvers.

On the day of the experiment, the outside climatologic conditions were sunny, 16°C, and 1016 mBar of pressure. However, the hyperbaric chamber complex was housed in an air-conditioned room with a fixed temperature of 24°C and humidity set at 40%. All the volunteers stayed in these controlled conditions for at least 1 hour before the start of the measurements. A small adjacent room was heated to 28°C with electric heaters.

Two hyperbaric chamber sessions were performed on the same day in the afternoon, and the subjects were randomly assigned to either session. Settings of the hyperbaric chamber at the time of the IOP measurements were as follows:

1. Baseline (normal ATM + normal temperature (T)): measurement of the IOP inside the hyperbaric chamber with a normal ATM of 1 Bar (sea level) and a T of 24°C.
2. ↑ ATM + ↑ T: measurements of the IOP inside the hyperbaric chamber after increasing the ATM to 2 Bar and the T to 28°C (also called ‘the dive’). It took approximately 10 minutes for these conditions to be reached. Five minutes later, the IOP was measured.
3. ↑ ATM (↑ ATM + normal T): measurements of the IOP inside the hyperbaric chamber with a stable ATM of 2 Bar after the T had been lowered to 24°C. It took approximately 20 minutes for the T to be lowered by 4°C. Five minutes later, the IOP was measured.

Overall, the cycle inside the hyperbaric chamber took approximately 60 minutes. All subjects underwent the same procedure in the same order. During the experiment, the investigators were also inside the hyperbaric chamber to measure the IOP. Before every measurement, we waited 5 minutes to allow for acclimatization.

In addition to IOP, we measured CCT with a handheld device (Pachmate DGH15; DGH Technology, Exton, UK), and we measured systemic blood pressure before and after the dive at the baseline settings 1 and 4. Five subjects routinely wore soft contact lenses for myopia, but these were removed the evening before the examination. Statistical analysis was performed to determine the statistical significance of the observed changes in IOP in response to the change in pressure, temperature, or both. A multivariate normal model with the covariance matrix modeled by the Kronecker product of a 2 × 2 covariance matrix for side and a 5 × 5 covariance matrix for repetition (five settings) was used to model the 10 repeated IOP measurements per subject. Based on this model, the interaction between repetition and side was significant (P = 0.026). Therefore, the analysis was performed separately for each side (using a multivariate normal model with an unstructured 5 × 5 covariance matrix for the five repeated measures of IOP per eye). Analyses were performed using statistical software (PROC MIXED procedure, SAS version 9.1; SAS Institute Inc., Cary, NC). The confidence interval was set at 95%.

RESULTS

Increased ATM in the hyperbaric chamber lowered the IOP significantly, regardless of the temperature. The IOP-lowering effect was more pronounced in the left eyes than in the right eyes, probably because of random fluctuation (Figs. 1, 2). For
one subject, no measurements were available at the third and fourth settings because of ear clearance problems; as a result, the subject was prematurely removed from the hyperbaric chamber. One subject was substantially (20 or more years) older than the others; his results were 4 mm Hg higher than observed in the other subjects’ IOP measurements, but the measurements followed the same pattern during the five settings. One subject had outlying data points for the third and fourth settings; IOP increases of 3 mm Hg for the right eye (RE) and 2 mm Hg for the left eye (LE) were observed.

Under normal temperature conditions, we found that increased ATM significantly lowered IOP (RE, 11.74–10.72 mm Hg \(P = 0.026\); LE, 11.69–10.52 mm Hg \(P = 0.005\)).

Regardless of temperature, when we took into account the overall effect size of the ATM increase on the IOP, the IOP-lowering effect was still significant (RE, \(P = 0.024\); LE, \(P = 0.0006\); Table 1 and Fig. 3). However, the correlation between IOP and temperature was not significant in both eyes (RE, \(P = 0.18\); LE, \(P = 0.24\)). Therefore, we can conclude that the effect of ATM was independent of temperature.

To determine whether temperature influences IOP, we altered the temperature under both ATM settings. We observed a small, but not significant, IOP-lowering effect in both eyes, where RE IOP measurements ranged from 11.74 mm Hg to 10.93 mm Hg \(P = 0.22\) and LE IOP measurements ranged from 11.69 mm Hg to 11.2 mm Hg \(P = 0.52\); Table 1).

We found that the IOP was still reduced after the complete hyperbaric cycle (60 minutes), where the average RE IOP measurement was 11 mm Hg after cycle compared with 11.74 mm Hg before cycle \(P = 0.13\), and the average LE IOP measurement was 11.2 mm Hg after cycle compared with 11.69 mm Hg before cycle \(P = 0.18\); however, these differences were not significant. Corneal thickness was measured as an independent variable before and after the hyperbaric cycle examination, and we found that it did not have a significant influence on IOP values \(P = 0.997\). CCT values for contact lens users were also not significantly increased compared with values for those who did not wear contact lenses \(P = 0.37\).

Systemic blood pressure was measured before and after hyperbaric exposure; there was no significant difference between the two readings. It has been previously shown that systemic blood pressure is not modified during mild hyperbaric exposure\(^{24}\), however, systemic blood pressure measurements during the entire experimental protocol were not performed.

### DISCUSSION

In the present study, an increase in ATM resulted in a significant and sustained decrease in IOP, both at increased and at baseline temperature. A second experiment was performed consecutively in which the temperature was kept constant. This condition did not lead to a significant change in IOP. Therefore, the observed decrease in

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**Table 1. Effect of ATM and T on the IOP in Healthy Persons**

<table>
<thead>
<tr>
<th></th>
<th>Right Eye</th>
<th></th>
<th>Left Eye</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Estimate (mm Hg)</td>
<td>SE</td>
<td>(P)</td>
<td>Estimate (mm Hg)</td>
</tr>
<tr>
<td>Effect ATM at nl T</td>
<td>-1.02</td>
<td>0.45</td>
<td>0.026</td>
<td>-1.26</td>
</tr>
<tr>
<td>Effect ATM at (\uparrow) T</td>
<td>-0.22</td>
<td>0.34</td>
<td>0.53</td>
<td>-1.06</td>
</tr>
<tr>
<td>Overall effect ATM</td>
<td>-0.62</td>
<td>0.26</td>
<td>0.024</td>
<td>-1.01</td>
</tr>
<tr>
<td>Effect T at nl ATM</td>
<td>-0.74</td>
<td>0.48</td>
<td>0.13</td>
<td>-0.50</td>
</tr>
<tr>
<td>Effect T at (\uparrow) ATM</td>
<td>0.06</td>
<td>0.30</td>
<td>0.84</td>
<td>0.21</td>
</tr>
<tr>
<td>Overall effect T</td>
<td>-0.34</td>
<td>0.27</td>
<td>0.22</td>
<td>-0.14</td>
</tr>
<tr>
<td>Interaction T and ATM</td>
<td>-0.80</td>
<td>0.59</td>
<td>0.18</td>
<td>-0.71</td>
</tr>
<tr>
<td>Difference settings 1 vs. 4</td>
<td>-0.69</td>
<td>0.43</td>
<td>0.13</td>
<td>-0.52</td>
</tr>
</tbody>
</table>

The overall effect sizes of the ATM and T on the IOP are stated in italic. Significant values are stated in bold; significant intervals were set at \(P < 0.05\).
IOP was presumably caused by the elevated ATM rather than by the concurrent change in temperature in the hyperbaric chamber.

To the best of our knowledge, this is the first report documenting the effect of elevated ATM on IOP in a healthy population without supplemental oxygen breathing. Ernsani et al.\textsuperscript{25} also studied the effect of hyperbaric oxygen on the IOP, but their study required patients to breathe hyperbaric oxygen (100\% \textsubscript{O\textsubscript{2}} at 2.5 Bar) while in the hyperbaric chamber.

In our study, the ATM increase led to a significant and sustained IOP decrease. After the complete hyperbaric cycle (60 minutes), the IOP was still reduced, so there was no sign that the eyes had adapted to the ATM. This might have been because of the prolonged effect of the ATM; it is not known how long external pressure changes can influence IOP. It should be noted that although the differences in IOP were small, they remained significant during the entire investigation. It is likely that longer recovery time after hyperbaric exposure would have normalized the IOP back to a baseline level. This is a shortcoming of our methodology that may be addressed in future studies with the introduction of an additional time-course of IOP measurements into the cycle. Additionally, it is possible that IOP differences may be larger in elderly persons, as suggested by the increased IOP measurements observed in the older subject in our sample population.\textsuperscript{25}

The mechanisms underlying the IOP decrease after the ATM increase are unclear. We hypothesize that different mechanisms could produce the IOP-lowering effect after ATM increase.

One possible cause is that the subjects were breathing higher oxygen levels at 2 Bar than at sea level. The percentage of oxygen in the breathing air was kept constant at 21\% during the experiment; therefore, at increased ATM, all subjects breathed a gas with partial oxygen pressure of 0.42 Bar (according to Dalton's law, 0.21 \times 2 Bar, corresponding to 42\% oxygen breathing at sea level). It has been shown that 75\%\textsuperscript{26} but not 25\%;\textsuperscript{27} oxygen breathing causes moderate hyperventilation and consequent respiratory alkalosis. Although we did not measure Pa\textsubscript{CO\textsubscript{2}} in our subjects, this minimal hyperventilation might have contributed to the observed IOP decrease.\textsuperscript{13,28}

To address issues of oxygen and CO\textsubscript{2} partial pressure, follow-up studies should adjust the concentration of the breathing gases to keep the partial pressure constant.

Additionally, it has been reported that hyperoxygenation induced by breathing 100\% oxygen induces retinal vasoconstriction, which may be responsible for a slight decrease of IOP, independently of changes in arterial CO\textsubscript{2} tension.\textsuperscript{29-31}

This has been confirmed by measurements of IOP in patients undergoing hyperbaric oxygenation (Fi\textsubscript{O\textsubscript{2}} = 2.5).\textsuperscript{25} However, the effect of moderate (Fi\textsubscript{O\textsubscript{2}} = 0.42) hyperoxygenation on ocular pressure has not been studied. From the literature we know that breathing air at 2 ATM causes only minimal arterial PCO\textsubscript{2} changes and cerebral vasoconstriction, or approximately one-third the effect of 100\% oxygen breathing at 1 Bar.\textsuperscript{32} It is possible that a similar effect contributed to the observed decrease in IOP in our study\textsuperscript{33} because the degree of IOP decrease was proportionally less than what was observed by Lukesch.\textsuperscript{29}

Another possibility is that, at higher ATM, the increased density of the breathing gas may modify respiratory dynamics by increasing the breathing resistance; this might, in theory, increase blood pressure by increasing the workload of the inspiratory and expiratory muscles. However, this effect is absent in resting conditions at 2 Bar. All subjects remained seated, and no physical exercise was performed because exercise is known to provoke a decrease in episcleral pressure.\textsuperscript{34} Nonstrenuous Valsalva maneuvers were performed to equalize middle ear pressure during the pressure increase (compression phase). All subjects avoided strenuous Valsalva because this could have been a confounding factor and could have led to an increase in IOP instead of a decrease.\textsuperscript{35,36} It is possible that these Valsalva maneuvers could have increased IOP in a small and transient way, but then the consequent IOP decrease would have even been lower.

Because of practical reasons, we only measured arterial blood pressure before and immediately after exposure and found no significant difference, consistent with the findings of Thomson et al.\textsuperscript{24} Although it is possible that some psychological stress could have caused a temporary increase in blood pressure, this seems unlikely in the hyperbaric chamber used for the experiments because it was spacious, comfortable, and confidence-inspiring. In fact, this hyperbaric chamber is specifically designed for treatments of a wide variety of patients, and all stress-provoking aspects of hyperbaric technology are concealed.

Although it is unlikely, an external tonographic effect by the ATM (e.g., massage after trabeculectomy) could have caused the IOP-lowering effect in our study. Pascal's law states that external pressure is transmitted integrally to any fluid-filled space, such as the eyeball. Similarly, we do not think this effect was caused by external pressures on the eyeball (e.g., Schiotz tonometry) because the tonometer we used was not influenced by external pressure.

We found that IOP decreases as ATM increases to conditions similar to those experienced when diving underwater to approximately 10 meters. Theoretically, the observed IOP decrease in our healthy sample population should be helpful to glaucomatous eyes; however, real underwater diving requires not only the physical exercise and pressure increase itself but also the use of goggles or a diving mask. Because our study did not account for all these variables, we could not immediately conclude that this activity is safe for such patients. Indeed, another recent report indicates that wearing swimming goggles significantly increases IOP at sea level conditions.\textsuperscript{37,38} This is probably related to an external pressure on the orbit that should not be present when wearing a real scuba diving mask because the rubber or silicon skirt of this type of mask rests on the facial bones around the orbit and not on the eyeball itself.

\section*{Conclusion}

ATM was inversely correlated to IOP in our healthy sample population. Our results show a small, but significant and sustained, decrease of IOP in a hyperbaric chamber at 2 Bar (equivalent to the pressure at 10 meters under water). The IOP decrease remained present throughout the entire experiment (60 minutes) of increased ATM. This decrease was of minor physiological significance in these subjects, whose IOP values were within the normal range. We can presume that hyperbaric exposure is not harmful for glaucoma patients. If there is any effect, induced by either a slight hyperventilation, hyperoxygenation or any other mechanism, it results in an IOP decrease.

This study was designed to observe the effect of ATM changes in a healthy population. We plan to determine whether the IOP changes observed in a healthy population after increased ATM carry over to glaucomatous eyes by conducting a prospective follow-up study with a higher ATM change (up to 4 Bar) on healthy persons and on glaucoma patients. Furthermore, to verify as precisely as possible the mechanisms responsible for any observed change in IOP, subgroups will be studied in whom the partial pressures of oxygen will be kept constant during exposure; longer periods of equilibration between the experimental steps will be included.
References