Original Article

Intraocular pressure fluctuation after water drinking test in primary angle-closure glaucoma and primary open-angle glaucoma

Yi-Chieh Poon, Mei-Ching Teng, Pei-Wen Lin, Jen-Chia Tsai, Ing-Chou Lai

Context: Only a few studies have assessed intraocular pressure (IOP) changes during the water drinking test (WDT) in patients with primary angle-closure glaucoma (PACG). **Aims:** The aim of this study is to investigate IOP changes during WDT in patients with PACG versus primary open-angle glaucoma (POAG). **Settings and Design:** This was a prospective and single tertiary center study. **Materials and Methods:** PACG and POAG patients (n = 15 each) without prior glaucoma surgery were enrolled and subjected to WDT, wherein they consumed an amount of water proportional to their body weight within 10 min. IOP was measured at baseline and every 15 min for 1 h after water intake. **Statistical Analysis Used:** Intergroup comparisons were performed using Mann–Whitney U-test for continuous variables and Chi-square test for categorical variables. Wilcoxon signed-ranks test was used for comparisons of IOP before and after water intake in the two groups. Regression analysis was used to determine factors associated with IOP fluctuations during WDT. **Results:** IOP changes over 1 h after water intake showed no significant differences between groups. The mean maximum fluctuation from baseline was 3.61 ± 2.49 and 3.79 ± 1.91 mmHg, respectively, in the PACG and POAG groups. The mean peak IOP was 19.17 ± 4.32 and 19.87 ± 3.44 mmHg in the PACG and PAOG groups, respectively. The axial length and anterior chamber depth showed no correlations with IOP fluctuations. **Conclusions:** We found similar IOP fluctuation curves and peak IO*P* values in both PACG and POAG patients subjected to WDT. These findings suggest that WDT is a useful test to induce IOP peaks in both POAG and PACG patients.

Key words: Intraocular pressure, primary angle-closure glaucoma, primary open-angle glaucoma, water drinking test

Access this article online

Website: www.ijo.in

DOI: 10.4103/0301-4738.198851

Quick Response Code:

Elevated intraocular pressure (IOP) is a well-recognized risk factor for the development and progression of glaucoma, and IOP control has been the primary goal of glaucoma treatment.[1-5] However, just like blood pressure, IOP does not remain constant and fluctuates throughout the day, depending on the time,[6-8] body position,[9,10] fluid intake,[7,11] exercise level, and medication intake.[12] Therefore, the range of IOP may be poorly reflected by single measurements obtained during normal clinical hours. In the Advanced Glaucoma Intervention Study,[13] it was found that IOP fluctuation was an independent risk factor for visual field (VF) progression in patients with a low mean IOP. This suggested that inability to detect and control of large IOP fluctuations in glaucoma patients with a seemingly well-controlled mean IOP could lead to further progression in VF defects.

However, 24-h IOP monitoring in patients with glaucoma may be impractical in clinical practice because the process is time consuming for both the patient and the clinician and is highly demanding with regard to professional resources. The water drinking test (WDT) is a provocative test that may be used to detect the range of elevation in IOP that a glaucoma patient may be exposed to. In a pilot study by Kumar *et al*.,[14] it was demonstrated that the peak IOP during WDT correlated well with the peak IOP measured over a 24-h period. The positive correlation between the peak IOP during WDT and the 24-h IOP was also confirmed in a study of a Japanese cohort.[15]

Most published studies on WDT have included patients with primary open-angle glaucoma (POAG), and the IOP changes during WDT in this group of patients are well documented. However, the prevalence of primary angle-closure glaucoma (PACG) is higher in the Asian population than in other populations around the world.[16] Thus far, only a few studies[17-19] have assessed IOP changes during WDT in patients with PACG. Studies by Baskaran *et al*.[20] and Tan *et al*.[21] showed that IOP fluctuations played a role in glaucoma progression and VF defects in PACG patients; therefore, investigation of the IOP response to WDT may be important in this patient population.

Therefore, in this study, we investigated changes in IOP during WDT in PACG patients and compared the findings with those for PAOG patients to gain a better understanding of the effects of this test on different types of glaucoma.

Department of Ophthalmology, Chang Gung University College of Medicine, Kaohsiung Chang Gung Memorial Hospital, Kaohsiung, Taiwan

Correspondence to: Dr. Ing-Chou Lai, Department of Ophthalmology, Kaohsiung Chang Gung Memorial Hospital, No. 123 Dapi Road, Niaosong District, Kaohsiung City 833, Taiwan. E-mail: lai1@cgmh.org.tw

Manuscript received: 17.06.16; Revision accepted: 30.10.16

Cite this article as: Poon YC, Teng MC, Lin PW, Tsai JC, Lai IC. Intraocular pressure fluctuation after water drinking test in primary angle-closure glaucoma and primary open-angle glaucoma. Indian J Ophthalmol 2016;64:919-23.

Materials and Methods

Ethics

This prospective study consecutively recruited patients who visited the glaucoma clinic in a tertiary medical center between November 2013 and June 2014. The study was conducted in accordance with the tenets of the Declaration of Helsinki as revised in 2000. The Hospital's Research Ethics Board approved the study protocols, and informed consent for participation was obtained from all patients before taking the test.

Selection and description of participants

All patients underwent comprehensive ophthalmological evaluations, including measurements of the Snellen visual acuity, automated refraction, gonioscopy, dilated fundus examination of the optic disc using a 90-diopter lens (Volk Optical Inc., Mentor, Ohio, USA), and optic disc imaging with spectral domain optical coherence tomography (Spectralis OCT, Heidelberg Engineering, Heidelberg, Germany). The axial length (AL) and anterior chamber depth (ACD) were measured using the IOLMaster (Carl Zeiss Meditec, Inc., Dublin, CA, USA) before the WDT test.

For both PACG and POAG patients, glaucomatous optic disc changes (vertical elongation of the optic cup, neuroretinal rim thinning, neuroretinal rim notching, and retinal nerve fiber layer defects) and compatible VF defects must be present for a diagnosis of glaucoma. Patients were diagnosed with PACG in the presence of occludable angles or angle synechiae on gonioscopy. All PACG patients had previously received laser iridotomy, and some were receiving topical medications for IOP control, depending on the adequacy of IOP control. Only PACG patients with peripheral anterior synechiae of <90° were included in this study. POAG was diagnosed according to the presence of open angles with a Shaffer grading of >2 on gonioscopy. All POAG patients were receiving one or more topical medications for IOP control. VF evaluations were performed using a Humphrey Field Analyzer (30-2 Program, Carl Zeiss Meditec, Inc., Dublin, CA, USA). VF findings used for analysis were obtained on or within 6 months before the day of WDT.

Patients with secondary glaucoma, including uveitic glaucoma, trauma-related glaucoma, and neovascular glaucoma, were excluded as were patients with a history of filtration surgery and laser trabeculoplasty. Patients with chronic heart failure and chronic renal disease who may not be able to tolerate the rapid intake of excessive fluid were also excluded from this study. When two eyes were eligible, one eye from each patient was randomly selected for analysis.

Water drinking test

For patients who consented to participation, no fluids or meals were permitted for 2 h before WDT, which was performed in the afternoon between 1 and 5 p.m. Each patient was instructed to drink an amount of water that was proportional to the body weight (10 ml/kg) within 10 min. The body weight was measured using an electronic weighing scale. IOP was measured using Tonopen Tonometry (TONO-PEN XL, Reichert Inc., Depew, NY, USA). Tonopen was used for IOP measurements in this study mainly because of its ease of portability which facilitates measurements of IOP while minimizing the need for the patient's postural change and movements in and out of the examination room that would be needed with taking IOP through Goldmann applanation tonometer. IOP measurements were obtained at baseline and every 15 min for 1 h after water intake. An error of <5% in the measured IOP, as indicated on the Tonopen monitor, was considered a valid measurement. The IO*P* value used for analysis was the mean of three consecutive measurements within 2 mmHg or the median of three measurements if the values differed by 3 mmHg or more.

The peak IOP was defined as the maximum IOP measured within 1 h after water intake. The maximum IOP fluctuation was defined as the difference between the peak IOP and baseline IOP.

Statistics

All statistical analyses were performed using SPSS software v. 20.0 (IBM Corp., Armonk, NY, USA). Intergroup comparisons were performed using Mann–Whitney U-test for continuous variables and Chi-square test for categorical variables. Wilcoxon signed-ranks test was used for comparisons of IOP before and after water intake in the two groups. Regression analysis was used to determine factors associated with IOP fluctuations during WDT. A $P < 0.05$ was considered statistically significant. All data are expressed as mean ± standard deviations.

Results

A total of 30 eyes from 30 patients (60% women; mean age, 62.0 ± 7.8 years; range, 45–74 years), including 15 with PAOG and 15 with PACG, were enrolled in this study. The mean body weight of patients was 62.1 ± 11.4 kg, which would correspond to a mean ingested water volume of 621.5 ± 114.3 ml/patient. Table 1 summarizes the baseline characteristics of patients in both groups. There were no significant differences in age between the two groups. ACD and AL showed smaller values in the PACG group than in the POAG group, which was as expected. The average IOP, mean deviation (MD) in VF, and circumpapillary retinal nerve fiber layer thickness were comparable between the two groups.

Nine patients (60.0%) in the PACG group and all patients (100%) in the POAG groups were receiving topical medications for IOP control. In the PACG group, seven patients were receiving monotherapy (beta-blocker, $n = 4$; prostaglandin

Table 1: Baseline characteristics of patients in the study population

	PACG (n=15)	POAG (n=15)	P
Age (years)	64.2±8.2	59.9±7.0	0.064
Sex (female:male)	11:4	7:8	0.136
Average IOP of previous three visits (mmHg)	14.6±2.8	14.2±3.7	0.431
Mean deviation (dB)	−3.64±1.87	−3.71±2.72	0.787
CpRNFL thickness (μ)	93.39±13.17	83.89±17.10	0.134
Central corneal thickness (μm)	510.80±26.19	511.93±33.43	0.917
Axial length (mm)	22.78±0.91	24.43±1.47	0.003
Anterior chamber depth (mm)	2.55±0.29	3.21±0.32	<0.001

All values are expressed as mean±SD. PACG: Primary angle-closure glaucoma, CpRNFL: Circumpapillary retinal nerve fiber layer, IOP: Intraocular pressure, POAG: Primary open-angle glaucoma, SD: Standard deviation

analog, *n* = 3), and two were receiving fixed-combination drugs. In the POAG group, 12 patients were receiving monotherapy (prostaglandin analog, *n* = 6; beta-blocker, *n* = 5; alpha-agonist, *n* = 1), and 3 were receiving combination therapy.

Before WDT, IOP (baseline IOP) was 15.55 ± 2.90 and 16.08 ± 2.65 mmHg in the PACG and POAG groups, respectively. IO*P* values for the two groups at each time point are summarized in Table 2 and Fig. 1. There was a significant increase in IOP that persisted until 30 min after water intake in the POAG (15 and 30 min: Both *P* = 0.001; Wilcoxon signed-rank test) group. While for the PACG, IOP elevated significantly until 45 min after the test (15, 30, and 45 min: *P* =0.003, *P* = 0.001, and *P* = 0.013, respectively; Wilcoxon signed-rank test). Subsequently, IOP decreased to near baseline levels at 60 min in the PACG group and 45 min in the POAG group. Overall, comparisons of IOP at each time point showed no significant differences between groups.

In the PACG group, the peak IOP occurred at 15 min in 40% (*n* = 6) of patients, 30 min in 26.7% (*n* = 4) of patients, and 45 min in 33.3% (*n* = 5) of patients. In the POAG group, the peak IOP occurred at 15 min in 60% (*n* = 9) of patients and 30 min in 26.7% (*n* = 4) of patients. The mean peak IOP was 19.17 ± 4.32 mmHg in the PACG group and 19.87 ± 3.44 mmHg in the POAG group (*P* = 0.771).

The maximum change in IOP during WDT ranged from 1.0 to 7.0 mmHg for the entire study group. The mean maximum IOP fluctuation from baseline was 3.61 ± 2.49 mmHg (23.0%) in the PACG group and 3.79 ± 1.91 mmHg (23.9%) in the POAG group, with no significant differences between groups (*P* = 0.740).

Regression analysis revealed no correlation of body weight, severity of glaucoma, baseline IOP, AL, and ACD with IOP fluctuations in the present study population.

Discussion

IOP is known to fluctuate depending on the time of the day and the individual's activity levels. These fluctuations have been identified as factors that can lead to disease progression in patients with both open-angle glaucoma[13,22-25] and ACG.[20,21] Even though a consensus on the definitive role of IOP fluctuations in glaucoma progression has not been reached,[26-28] such fluctuations are nevertheless an important factor for consideration, particularly in patients presenting with progressive VF defects despite an apparently stable IOP during follow-up visits.[7,13]

WDT was introduced in the early to mid-1900s as a diagnostic test[29] for patients with suspected glaucoma. The findings were considered positive when IOP increases above 6 mmHg after the water intake. The test later decreased in popularity because it was unable to distinguish between patients with and without glaucoma. In other words, its sensitivity as a diagnostic test was relatively low.[30] However, it has again gained popularity in recent years and is currently considered a useful provocative test for the detection of IOP fluctuations and peak diurnal IOP variations in patients with glaucoma.[31,32] Studies have demonstrated a good correlation between the peak IOP during WDT and the peak diurnal IOP;[14,15] therefore, WDT, which requires approximately an hour to complete, may be a more convenient alternative to 24-h measurements of IOP. Although WDT is a historical test with low diagnostic sensitivity for glaucoma,[30] its value in clinical practice today lies not in its glaucoma diagnostic value but in it being a provocative test that can allow physicians to detect the fluctuations in IOP that a glaucoma patient may be exposed to outside of routine clinic visits during normal office hours.

Increased IOP fluctuations during WDT have been shown to be associated with the severity of glaucoma. In a study by Susanna *et al*.,[33] which included patients with asymmetrical VF defects, eyes with the worse MD value presented with greater peak IO*P* values and fluctuations during WDT compared with the healthier eyes. Therefore, eyes with greater IOP fluctuations during WDT may be prone to greater VF damage.

The mechanism underlying the increase in IOP after water intake is currently hypothetical. In fact, several mechanisms have been postulated. Some have suggested that the IOP increase is associated with the influx of fluid that causes an increase in choroidal perfusion and volume.[34] In a more recent study involving swept-source optical coherence tomography, Mansouri *et al*.[35] found an increase of 5.7% in the peripapillary choroidal thickness and 4.3% in the macular choroidal thickness after water intake by healthy participants. However, several

Table 2: Intraocular pressure response after water drinking test in primary angle-closure glaucoma patients and primary open-angle glaucoma patients

	PACG (*n*=15)	POAG (*n*=15)	*P*
Baseline IOP (mmHg)	15.55±2.90	16.08±2.65	0.950
15 min IOP (mmHg)	17.83±4.46	18.64±3.24	0.787
30 min IOP (mmHg)	18.36±4.26	18.67±3.22	0.950
45 min IOP (mmHg)	17.57±3.74	17.29±3.68	0.743
60 min IOP (mmHg)	15.65±3.32	17.22±3.69	0.289
Maximum IOP fluctuation from baseline (mmHg)	3.61±2.49	3.79±1.91	0.755
Percentage IOP fluctuation	23.0±15.0	23.9±12.7	0.740

All values are expressed as mean±SD. PACG: Primary angle-closure glaucoma, IOP: Intraocular pressure, POAG: Primary open-angle glaucoma, SD: Standard deviation

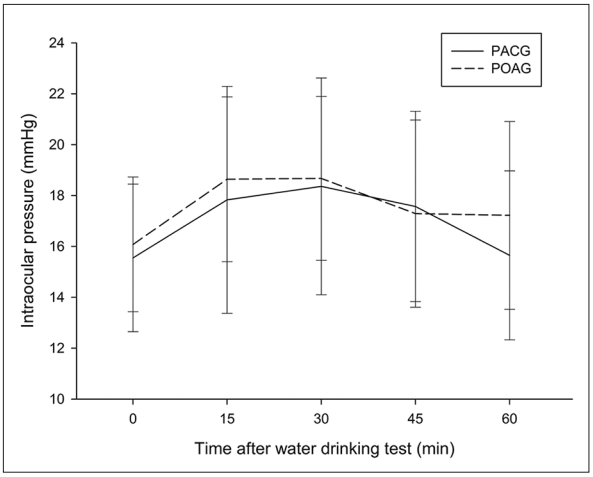

Figure 1: Intraocular pressure curve for intraocular pressure response to water drinking test in primary angle-closure glaucoma and primary open-angle glaucoma patients

authors have been unable to determine a definite correlation between the increase in choroidal thickness and the increase in IOP.[17,18,35] Moreover, it has long been suggested that an IOP increase may be related to changes in the blood osmolality after water intake. In a recent study, Nongpiur *et al*.[18] demonstrated that a significant decrease in serum osmolality occurred after water intake during WDT, and this was significantly correlated with changes in IOP. Finally, water intake has also been shown to be associated with an increase in the blood pressure and peripheral vascular resistance.[36] Changes in hemodynamics may be associated with an increased episcleral venous pressure, which leads to a decreased outflow facility.

To better understand the IOP response to WDT and provide further insights into the possible mechanisms involved, it may be important to evaluate the responses of different types of glaucoma to this test. In the present study, we performed WDT in age-matched and glaucoma severity-matched POAG and PACG patients and evaluated their IOP responses.

We found that the two groups displayed very similar IOP curves after water intake, despite differences in ocular anatomy and biometrics. The mean maximum IOP fluctuation was 3.61 ± 2.49 mmHg in the PACG group and 3.79 ± 1.91 mmHg in the POAG group. Only one other study by Arora *et al*.[17] has made a direct comparison between these two types of glaucoma. In that study, after ingestion of 1 L of water, eyes with ACG demonstrated greater IOP fluctuations compared with those with open-angle glaucoma (6.00 vs. 4.25 mmHg, respectively, $P = 0.004$). However, considering the high proportion of patients without medications (40%) in the open-angle group in the previous study,[17] we speculate that a large proportion of their participants were glaucoma suspects. Therefore, the differences in the study populations can explain the differences in findings between the two studies.

In a recent study by Waisbourd *et al*.,[19] the authors performed WDT before and after laser iridotomy for patients with suspected PACG and found no differences between the peak IOP values before and after iridotomy. Even in the present study, we found no differences in peak IOP values during WDT between POAG and PACG patients. This lack of difference in the IOP response to WDT between POAG and PACG patients, who exhibit different angle anatomies, suggests that choroidal expansion-related angle narrowing does not play a major role in IOP increases during WDT. Regression analysis in the present study also showed that biometric parameters, including AL and ACD, were not associated with IOP changes. Accordingly, we believe that factors that similarly affect both types of glaucoma, such as changes in osmolality, blood pressure, and hemodynamics, are more likely to play a greater role in IOP fluctuations during WDT. Because blood pressure and osmolality were not assessed in the present study, future studies should include systemic evaluations during assessments of the IOP response to WDT in patients with different types of glaucoma.

The major limitations of this study include the small sample size in both groups, the lack of a healthy control group without glaucoma, the lack of direct assessments of systemic factors, and assessment of biometric change in the choroid and ACD. It would be important to include the assessment of systemic factors, measurement of changes in structural and biometric parameters before and after WDT, and including a healthy control group in a future study that explores and investigates specifically on the mechanisms of WDT. However, to our knowledge, the present study is one of the few to evaluate the IOP response to WDT in PACG patients and the first to make direct comparisons between PAOG and PACG patients with glaucomatous VF defects.

Conclusions

We found that the POAG and PACG patients demonstrated similar IOP fluctuation curves and peak IOP values during WDT and that there was no correlation between AL and ACD to IOP changes. Induced IOP peaks after WDT can be significantly higher than baseline values; therefore, it is important to be able detect these IOP fluctuations, particularly in patients with glaucoma progression. The findings of our study suggest that WDT is a useful provocative test for the detection of IOP peaks in both POAG and PACG patients, particularly when a 24-h IOP monitoring facility is not available to detect such IOP changes.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. The effectiveness of intraocular pressure reduction in the treatment of normal-tension glaucoma. Collaborative normal-tension glaucoma study group. Am J Ophthalmol 1998;126:498-505.
2. The advanced glaucoma intervention study (AGIS): 7. The relationship between control of intraocular pressure and visual field deterioration. The AGIS Investigators. Am J Ophthalmol 2000;130:429-40.
3. Leske MC, Heijl A, Hussein M, Bengtsson B, Hyman L, Komaroff E; Early Manifest Glaucoma Trial Group. Factors for glaucoma progression and the effect of treatment: The early manifest glaucoma trial. Arch Ophthalmol 2003;121:48-56.
4. Leske MC, Heijl A, Hyman L, Bengtsson B, Dong L, Yang Z; EMGT Group. Predictors of long-term progression in the early manifest glaucoma trial. Ophthalmology 2007;114:1965-72.
5. Sultan MB, Mansberger SL, Lee PP. Understanding the importance of IOP variables in glaucoma: A systematic review. Surv Ophthalmol 2009;54:643-62.
6. Wilensky JT. The role of diurnal pressure measurements in the management of open angle glaucoma. Curr Opin Ophthalmol 2004;15:90-2.
7. Malerbi FK, Hatanaka M, Vessani RM, Susanna R Jr. Intraocular pressure variability in patients who reached target intraocular pressure. Br J Ophthalmol 2005;89:540-2.
8. Fogagnolo P, Orzalesi N, Ferreras A, Rossetti L. The circadian curve of intraocular pressure: Can we estimate its characteristics during office hours? Invest Ophthalmol Vis Sci 2009;50:2209-15.
9. Gautam N, Kaur S, Kaushik S, Raj S, Pandav SS. Postural and diurnal fluctuations in intraocular pressure across the spectrum of glaucoma. Br J Ophthalmol 2016;100:537-41.
10. Prata TS, De Moraes CG, Kanadani FN, Ritch R, Paranhos A Jr. Posture-induced intraocular pressure changes: Considerations regarding body position in glaucoma patients. Surv Ophthalmol 2010;55:445-53.
11. Kerr NM, Danesh-Meyer HV. Understanding the mechanism of the water drinking test: The role of fluid challenge volume in patients with medically controlled primary open angle glaucoma. Clin Exp Ophthalmol 2010;38:4-9.

12. Sit AJ, Asrani S. Effects of medications and surgery on intraocular pressure fluctuation. Surv Ophthalmol 2008;53 Suppl 1:S45-55.
13. Caprioli J, Coleman AL. Intraocular pressure fluctuation a risk factor for visual field progression at low intraocular pressures in the advanced glaucoma intervention study. Ophthalmology 2008;115:1123-9.e3.
14. Kumar RS, de Guzman MH, Ong PY, Goldberg I. Does peak intraocular pressure measured by water drinking test reflect peak circadian levels? A pilot study. Clin Exp Ophthalmol 2008;36:312-5.
15. Sakata R, Aihara M, Murata H, Saito H, Iwase A, Yasuda N, *et al.* Intraocular pressure change over a habitual 24-hour period after changing posture or drinking water and related factors in normal tension glaucoma. Invest Ophthalmol Vis Sci 2013;54:5313-20.
16. Tham YC, Li X, Wong TY, Quigley HA, Aung T, Cheng CY. Global prevalence of glaucoma and projections of glaucoma burden through 2040: A systematic review and meta-analysis. Ophthalmology 2014;121:2081-90.
17. Arora KS, Jefferys JL, Maul EA, Quigley HA. Choroidal thickness change after water drinking is greater in angle closure than in open angle eyes. Invest Ophthalmol Vis Sci 2012;53:6393-402.
18. Nongpiur ME, Foo VH, de Leon JM, Baskaran M, Tun TA, Husain R, *et al.* Evaluation of choroidal thickness, intraocular pressure, and serum osmolality after the water drinking test in eyes with primary angle closure. Invest Ophthalmol Vis Sci 2015;56:2135-43.
19. Waisbourd M, Savant SV, Sun Y, Martinez P, Myers JS. Water-drinking test in primary angle-closure suspect before and after laser peripheral iridotomy. Clin Exp Ophthalmol 2016;44:89-94.
20. Baskaran M, Kumar RS, Govindasamy CV, Htoon HM, Wong CY, Perera SA, *et al.* Diurnal intraocular pressure fluctuation and associated risk factors in eyes with angle closure. Ophthalmology 2009;116:2300-4.
21. Tan S, Yu M, Baig N, Chan PP, Tang FY, Tham CC. Circadian intraocular pressure fluctuation and disease progression in primary angle closure glaucoma. Invest Ophthalmol Vis Sci 2015;56:4994-5005.
22. Tajunisah I, Reddy SC, Fathilah J. Diurnal variation of intraocular pressure in suspected glaucoma patients and their outcome. Graefes Arch Clin Exp Ophthalmol 2007;245:1851-7.
23. Rao HL, Addepalli UK, Jonnadula GB, Kumbar T, Senthil S, Garudadri CS. Relationship between intraocular pressure and rate of visual field progression in treated glaucoma. J Glaucoma 2013;22:719-24.
24. Fogagnolo P, Orzalesi N, Centofanti M, Oddone F, Manni G, Rossetti L. Short- and long-term phasing of intraocular pressure in stable and progressive glaucoma. Ophthalmologica 2013;230:87-92.
25. Asrani S, Zeimer R, Wilensky J, Gieser D, Vitale S, Lindenmuth K. Large diurnal fluctuations in intraocular pressure are an independent risk factor in patients with glaucoma. J Glaucoma 2000;9:134-42.
26. Bengtsson B, Leske MC, Hyman L, Heijl A; Early Manifest Glaucoma Trial Group. Fluctuation of intraocular pressure and glaucoma progression in the early manifest glaucoma trial. Ophthalmology 2007;114:205-9.
27. Medeiros FA, Weinreb RN, Zangwill LM, Alencar LM, Sample PA, Vasile C, *et al.* Long-term intraocular pressure fluctuations and risk of conversion from ocular hypertension to glaucoma. Ophthalmology 2008;115:934-40.
28. Leidl MC, Choi CJ, Syed ZA, Melki SA. Intraocular pressure fluctuation and glaucoma progression: What do we know? Br J Ophthalmol 2014;98:1315-9.
29. Leydhecker W. The water-drinking test. Br J Ophthalmol 1950;34:457-79.
30. Roth JA. Inadequate diagnostic value of the water-drinking test. Br J Ophthalmol 1974;58:55-61.
31. Danesh-Meyer HV. The water-drinking test: The elegance of simplicity. Clin Exp Ophthalmol 2008;36:301-3.
32. Goldberg I, Clement CI. The water drinking test. Am J Ophthalmol 2010;150:447-9.
33. Susanna R Jr., Hatanaka M, Vessani RM, Pinheiro A, Morita C. Correlation of asymmetric glaucomatous visual field damage and water-drinking test response. Invest Ophthalmol Vis Sci 2006;47:641-4.
34. De Moraes CG, Reis AS, Cavalcante AF, Sano ME, Susanna R Jr. Choroidal expansion during the water drinking test. Graefes Arch Clin Exp Ophthalmol 2009;247:385-9.
35. Mansouri K, Medeiros FA, Marchase N, Tatham AJ, Auerbach D, Weinreb RN. Assessment of choroidal thickness and volume during the water drinking test by swept-source optical coherence tomography. Ophthalmology 2013;120:2508-16.
36. Lu CC, Diedrich A, Tung CS, Paranjape SY, Harris PA, Byrne DW, *et al.* Water ingestion as prophylaxis against syncope. Circulation 2003;108:2660-5.