POSTURAL EFFECTS ON SPONTANEOUS RETINAL VENOUS PULSATIONS IN HEALTHY INDIVIDUALS

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Abbreviations: IOP, intraocular pressure; MAP, mean arterial pressure; MOPP, mean ocular perfusion pressure; SVP, spontaneous venous pulsations

ABSTRACT

Purpose: To assess amplitudes of spontaneous retinal venous pulsations (SVP) in three various postures (sitting, supine, and lateral decubitus) in healthy individuals.

Methods: Thirty participants (28±8 years, 25 females) were included in the study. Intraocular pressure (IOP), blood pressure (BP), and SVP's were measured at three different postures using a calibrated Tono-Pen application tonometer, a digital sphygmomanometer, and a custom-built handheld video ophthalmoscope, respectively. SVP amplitudes were extracted from the retinal videos using a custom written MATLAB algorithm. Mean arterial pressure (MAP= (systolic+2diastolic)/3), and mean ocular perfusion pressure (MOPP=(2/3 MAP)-IOP) were also calculated at each posture. A one-way ANOVA was applied to each parameter to determine any significant difference for the various postural changes.

Results: Mean IOP increased ($p<0.0001$) and mean SVP decreased ($p<0.0001$) from sitting to supine. The mean IOP (mmHg) and SVP (MU; measuring units) in sitting, supine and lateral decubitus were 16.2±2, 19.4±4, 19.8±2 mmHg and 5.8±2, 4.5±2, and 4.7±2 MU, respectively. MAP and MOPP also decreased significantly from sitting to supine ($p<0.001$, $p<0.001$), and sitting to lateral decubitus ($p<0.05$, $p<0.01$). There were no significant differences between IOP,SVP , MAP, or MOPP during a postural modification from supine to lateral decubitus.

Conclusions: In this study, we showed a significant reduction in SVP amplitudes and a significant increase in IOP from sitting to supine position in a healthy young cohort. This supports the rationale to further study such phenomenon in ocular conditions such as glaucoma to determine whether relative SVP change, for a similar postural change, can reveal early signs of vascular dysfunction.

INTRODUCTION

A change in body position is known to effect cerebrospinal fluid pressure (CSFp) (Qvarlander et al. 2013), intraocular pressure (IOP) (Weinreb, Cook & Friberg 1984) and mean arterial pressure (MAP) (Olufsen et al. 2005) , all of which contribute to hemodynamic stability at the optic nerve head. A disruption in the interaction between these physiological pressure compartments have been associated with of a number of diseases. The association between IOP and MAP is a major driver of ocular perfusion pressure (OPP), a well-established marker associated with primary open angle glaucoma(Weinreb et al. 2016)(Choi & Kook 2015). The translaminar pressure difference (TLPD), the difference between IOP and CSFp, is affected in idiopathic intracranial pressure and also glaucoma(Berdahl, Yu & Morgan 2012). Taken together, an acute change, such as postural change, in each of these parameters, can affect the OPP and/or TLPD resulting in damage to the optic nerve head and subsequent pathology.

Evidence on TLPD changes associated with body position is limited, mainly due to the fact that direct measurement of CSFp requires complicated invasive methods and the non-invasive methods developed for CSFp estimation lack accuracy(Zhang et al. 2017). Current evidence suggests that assessment of spontaneous retinal venous pulsations (SVP) may be a proxy approach to TLPD evaluation(Golzan et al. 2011, Golzan et al. 2012, Levine & Bebie 2016). SVPs are usually observed in the central retinal veins and appear as visible rhythmic pulses of the vessel wall as the veins cross the optic disc(Jacks & Miller 2003, Jonas 2005, Morgan 2013). They are present in 81% of all eyes, and 90% in healthy controls(Jacks & Miller 2003). Recent studies have reported the amplitude of SVPs is influenced by many factors including IOP, retinal venous pressure (RVP) as well as CSFp(Jonas, Ritch & Panda-Jonas 2015, Jonas & Wang 2013, Lee et al. 2016, Ren et al. 2010). Therefore, assessment of SVPs may provide a useful insight into TLPD changes.

It is well recognised that changing postures influence IOP, a major component of TLPD. In fact, studies have reported an increase in IOP by up to 6mmHg between seated and supine positions(Tsukahara & Sasaki 1984). The same holds true for CSFp, the other major TLPD component, with a posture change from sitting to supine inducing a rapid change in CSFp levels due to the impacts of the hydrostatic forces acting on the body(Andresen et al. 2015, Poca et al. 2006). The significant increase in CSFp between seated and supine positions is caused by changes in the cerebral blood volume reflecting postural blood pressure waves and brain autoregulation. In pathology such as glaucoma, it has been suggested that sleeping positions may be an important disease mechanism to explore the effects of different postural changes on IOP fluctuations(Malihi & Sit 2012). A recent study by Lazzaro showed that a 20 degree head-up position in glaucoma patients correlates with lower nocturnal IOP compared with supine position(McCulley et al. 2003). As most patients present more glaucomatous pathology in one eye than the other, it may be feasible to suggest that the pathological eye is related to the patient's preferred sleeping position such as lateral decubitus(Malihi & Sit 2012).

Parallel to IOP and CSFp, ocular perfusion is also an important factor in maintaining the homeostatic hemodynamics at the optic nerve head. OPP is the force behind ocular blood flow with low OPP linked to development and progression of glaucoma(Costa et al. 2014, Lee, Yoo & Kim 2013). OPP is influenced by both MAP and IOP. By moving from sitting to supine, IOP has been reported to increase by 6mmHg in healthy individuals(Liu et al. 2003). For a similar change, the MAP increases about 20 to 30 mmHg (Costa et al. 2014). The hemodynamic interactions at the optic nerve head are very complex and while the balance between MAP and IOP, influenced by the auto-regulatory mechanisms of the body, can dictate the OPP delivered to the eye through the arterial system, the venous outflow regulated through the TLPD may ultimately determine the likelihood of an individual for developing optic nerve damage. The current study explores the effects of body position on the amplitude of spontaneous retinal venous pulsatility as a marker of TLPD in healthy individuals to

further explore how changes to various physiological pressure compartments because of postural change may affect the optic nerve head hemodynamics.

METHODOLOGY

Data collection

Thirty healthy participants aged between 18-60 (25 female, 28 ± 8 years) with no history of any eye disease, hypertension or diabetes were included in the study. IOP, blood pressure (BP), and SVPs were measured at sitting, supine, and lateral decubitus positions. IOP was measured using a calibrated Tono-pen applanation tonometer (tonopen). Systolic BP (SBP) and diastolic BP (DBP) was measured using a digital sphygmomanometer. A 10-second recording of the retinal vessels (46^{o} field of view, 30 fps) was captured using a custom-built handheld tablet ophthalmoscope. Mean arterial pressure (MAP) and mean ocular perfusion pressure (MOPP) was calculated using the following formulas;

MAP= DBP+1/3(SBP-DBP)

MOPP=(2/3MAP)-IOP.

This study was performed in accordance with the guidelines of the Tenants of Helsinki and approved by University of Technology Sydneys' human ethics committee. Written consent was obtained from the participants after explanation of the nature and possible consequences of the study.

Experimental Paradigm

A drop of Alcain 0.5% (Alcon, Texas, USA) was instilled for IOP measurements. Following this, one pupil from all patients was dilated using Tropicamide 1% (Alcon, Texas, USA) and three consecutive measurements were taken from each eye in all positions. Two consecutive BP measurements were also recorded in each position. A third measurement was taken if there was a difference of more than 5mmHg between the systolic/diastolic measurements. Participants were given a minimum of 10 minutes rest between posture changes to allow the IOP and BP to stabilize before measurements were

taken. Participants were asked to lay on their back (supine) and then on the side of the dilated eye (lateral decubitus) resting their head on a pillow in preparation for retinal video imaging. A 10 second video recording of SVPs were captured in each position.

SVP Assessment

SVP amplitudes were extracted from retinal videos using a custom written MATLAB (Mathworks, USA) algorithm that has been validated and reported before(Golzan et al. 2014). Briefly, for each participant, a 10 second colour recording (30 fps) of the posterior pole was obtained. The first image in the video sequence was used as reference and the subsequent images were aligned according to the reference image. A template matching slice alignment was used to align all the images in the video. In this technique, the user defines a region of interest and the algorithm will search for the same landmark or similar pattern in subsequent images and re-writes the image into a new position that is aligned with the reference image. Following this, a Contrast Limited Adaptive Histogram Equalization (CLAHE) algorithm was applied to enhance the local contrast of each image(Pizer 1987). The central retinal vein on the optic disc was then manually selected and the inner vessel calibre in each image was measured using a full width at half-maximum algorithm. Finally, the vessel diameter in each image was plotted against the timing of each frame. The difference in peak to trough of the resultant trace was measured and designated as the SVP amplitude. The diameter change measured based on number of pixels is reported herein using measuring units (MU) (Figure 1)

Data Analysis

The consecutive IOP and BP measurements were averaged at each of the three postures. A one-way ANOVA was used to assess the difference in each parameter in all postures. A Tukey post-hoc analysis was applied to determine any significant change within each individual posture change (ie sitting-supine, sitting-lateral, and supine-lateral). We also studied the mean difference across averaged

parameters (ie. IOP, SVPs and MOPP) to determine any inter-postural variability. Graphpad prism (CA,USA) was used for data analysis and visualisation.

RESULTS

Data for all 30 participants were analysed to measure the mean SVP amplitude, IOP, MAP and MOPP for all participants in sitting, supine, and lateral decubitus. Table 1 summarises the mean value for each parameter.

Mean SVP amplitude and IOP difference

We observed a 22% reduction in the SVP amplitude from seated to supine position. In contrast, we observed an 18% increase in IOP for a similar postural change. The rate of change for SVP amplitude and IOP from seated to lateral decubitus were -18% and 14%, respectively. The changes between SVP amplitude and IOP from supine to lateral decubitus were minute (4% and 3%), suggesting trivial changes in between the two postures (Figure 2).

Mean MOPP difference

To further evaluate postural effects on systemic blood pressure and subsequent mean ocular perfusion pressure (MOPP), we studied the mean difference in MOPP in the various postures. Similar to SVP amplitudes and IOP, the greatest difference in MOPP was observed in a postural change from seated to supine (-14%). Mean MOPP difference from seated to lateral decubitus and supine to lateral decubitus were -8% and 6.9% (Figure 3).

DISCUSSION

In this study, we showed a significant reduction in the amplitude of SVPs and MOPP and a significant increase in mean IOP for a postural change from sitting to supine in healthy individuals. All current evidence in the literature have measured and assessed SVPs in the seated position and this is the first study, to our knowledge, to present data on the effects of body position on SVPs. Up to 98% of normal individuals have been reported with visible SVPs (Morgan et al. 2004) , however, objective digital methods have been able to visualise SVPs in 100% of normal and glaucoma patients (Golzan et al. 2015). While the origin of SVPs is highly debated in the literature, current evidence suggests that the interaction between IOP, within the globe, and the CSFp within the optic nerve sheath outside the globe, is the main driver of SVP in normal physiological conditions(Chang & Singh 2010, Golzan, Avolio & Graham 2012, Levine & Bebie 2016). An increase in IOP levels or decrease in CSFp levels will lead to higher SVP amplitudes. In a previous study (Golzan et al. 2011), we demonstrated that pharmacological lowering of IOP results in reduced SVPs in the sitting position (assuming constant CSFp), further supporting the hypothesis that SVPs are mainly driven by the difference between IOP and CSFp, also known as the TLPD. In the current study, we observed a reduction in SVP amplitudes from sitting to supine. As the mean IOP also increased for the same postural change, the plausible explanation for a drop in SVP amplitude is a greater increase in CSFp levels from sitting to supine leading to an overall reduction in TLPD (IOP-CSFp) and subsequent loss of SVPs. While we did not measure CSFp to confirm whether such hypothesis is true, results from animal and clinical studies support such notion. A study on rats showed that a change in body position from vertical to horizontal produced a significant increase in CSFp levels leading to reduced TLPD levels(Skrzypecki & Ufnal 2017). Similar studies in rabbits(Klarica et al. 2016) and cats(Kuzman et al. 2012) also show an increase in CSFp from vertical to horizontal postural change (with minimal IOP change) leading to decreased TLPD.

The effects of body position on TLPD in human participants have been poorly studied as both IOP and CSFp are known to be affected by body position(Linder, Trick & Wolf 1988, Magnaes 1976) and therefore any attempt to assess TLPD in clinical studies needs to simultaneously measure both parameters at any given body position. The lack of an accurate approach in non-invasive estimation of CSFp in various body positions has made such attempts cumbersome. Eklund et al have measured CSFp invasively at different postures and have showed a reduction in TLPD from sitting (19.8 ± 4.6 mmHg) to supine (12.3 ± 2.2 mmHg) in healthy participants (Eklund et al. 2016). The authors report an increase of 18% in IOP levels from sitting to supine. Our results are also consistent with this as we also observed an IOP increase of 18% from sitting to supine. Furthermore, results from this study demonstrate that a postural change influences the CSFp more than the IOP, leading to an approximately 37% TLPD reduction. Our observation of approximately 22% SVP loss from seated to supine mirrors the TLPD reduction reported by Eklund et al.

Changes to MOPP in various body positions have also been studied previously, with contradictory results reported mainly due to the different equations used to assess MOPP. Kara et al used the commonly used equation to evaluate MOPP (i.e. 2/3 MAP-IOP) (Barbosa-Breda et al. 2018) and found a significantly lower MOPP in supine compared with sitting (39.71 ± 6.96 vs 46.41 ± 5.54 mmHg)(Kara et al. 2013). However, when using the equations below, proposed by Bill (Bill 1978) for each of the postures, Tae-Eun Lee et al (Lee, Yoo & Kim 2013)reports a significantly higher MOPP in supine compared with sitting (54.4±4.3 vs 45.6±5.0 mmHg)

(1)Sitting position: OPP = 95/140 × MAP – IOP

(2)Sleeping position: OPP = 115/130 × MAP – IOP

Our findings were consistent with the former study using the commonly used equation with significantly lower MOPP in supine compared to sitting (39.1±7.8 vs 45.6±6.4 mmHg, p=0.005). Interestingly, when we applied the equations developed by Bill, we observed an increase in MOPP

from sitting to supine (47.4±6.5 vs 59.3±9.8 mmHg, $p<0.0001$). A follow-up study by Liu (Liu et al. 2003) showed that MOPP was higher in the nocturnal period (supine) compared with diurnal period (sitting). While it's difficult to confirm which method represents the most accurate MOPP in the various postures, it appears the equations developed by Bill and validated by others may be suitable in studies where certain body positions are maintained for a prolonged period of time (e.g. sleep) and thus the physiological measures such as IOP and MAP have adjusted accordingly. In an acute setting, such as the current study, where the measurements were taken only after 10 minutes rest time, the more commonly accepted equation may represent the actual MOPP levels. Nonetheless, further studies are required to confirm such theory.

Our findings may have implications in neurological and ocular pathology such as idiopathic intracranial hypertension and glaucoma. (Balaratnasingam et al. 2007, Choudhari, Raman & George 2009, McKee & Ahad 2004, Wong & White 2013) (Golzan et al. 2015, Morgan et al. 2004). More specifically, SVP frequency has been reported to be higher in glaucoma suspects compared to primary open angle glaucoma (86.5% vs 53.3%) (Seo et al. 2012) , SVP amplitudes have been linked to glaucoma severity (Golzan et al. 2015)and that lower SVP presence has been linked to increased CSFp(Choudhari, Raman & George 2009). The IOP and CSFp interaction (and subsequent TLPD) observed in these diseases (Jóhannesson, Eklund & Lindén 2018) exposes the lamina cribrosa and the optic nerve head to large pressure gradients. Results from animal studies suggest that when CSFp falls below the surrounding intraorbital pressure in sitting position, a normal physiological response is the occlusion of the optic nerve sheath to protect the axons from the low CSFp levels (Morgan et al. 1998). A dysfunction of this mechanism has been hypothesised as a major contributing factor to optic nerve head pathology seen in glaucoma (Jóhannesson, Eklund & Lindén 2018, Morgan, Yu & Balaratnasingam 2008). As a result, baseline SVP together with its relative change due to a postural change, may be a useful indicator of TLPD change and subsequent pathology.

Our study has a few limitations. First, we only studied posture-induced SVP changes in healthy participants. Hence, whether a similar change can be observed in other pathologies particularly glaucoma, remains to be elucidated. Second, the majority of our participants were females. However, we do not expect at the age range of our cohort, gender may have influenced our results. Third, we did not measure CSFp between postural changes. Therefore, it is difficult to confirm whether the changes we see in SVPs (as a proxy for TLPD) in various postures is predominantly IOP or CSFp driven.

In conclusion, we provide the first in-sight into dynamic vascular changes at the optic nerve head in various postures. Our results showed that SVP amplitude is significantly reduced from sitting to supine. This suggests that both factors driving the SVP (i.e. IOP and CSFp) play a significant role in maintaining the homeostatic hemodynamic conditions at the ONH. Further studies are required not only to confirm whether a similar change can be observed in glaucoma patients, but also to investigate whether sleep duration (in supine position) is associated with glaucomatous pathology.

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Figure Captions

Figure 1. Two magnified frames from a retinal venous pulse video at 0.54 seconds apart. *Left picture)* arrows indicate max venous collapse (trough) and max venous dilation (peak), *right graph)* SVP trace (note corresponding peak and trough).

Figure 2. Change in SVP (left) and IOP (right) between postures. A significant change in SVP and IOP from sitting to supine and lateral decubitus (LD) was observed.

Figure 3. Change in MOPP between postures. A significant change from sitting to supine and lateral decubitus (LD) was observed.