Altering Body Position Affects Intraocular Pressure and Visual Function

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Intraocular pressure (IOP) can be altered by changing body position. This report describes two experiments evaluating variations in IOP, as well as neural functioning of the retina and visual cortex (as measured by pattern-reversal electroretinogram and visual evoked potential), associated with whole-body, head-down tilt. The subjects, ten per experiment, were visually normal with IOP < 19. In the first experiment, IOP elevations were induced by varying the angle of tilt in discrete steps between +90° (upright) and −90° (inverted). In each position IOP was measured and significant elevations (up to 3X baseline) were noted. These elevations were maintained for 1 min during which simultaneous retinal and cortical biopotentials were measured. In the second experiment, 6° head-down tilt was maintained for 2 hr during which time the IOP and both biopotentials were measured repeatedly. Our findings confirm the effect of body position on IOP, while also revealing that head-down tilt produces significant reductions in neurophysiological function at both the retinal and cortical levels. The neural effect is maximized when 6° head-down tilt is maintained for 20 min. Invest Ophthalmol Vis Sci 29:1492-1497, 1988

Changing body position alters intraocular pressure.1-5 In ocular normotensives intraocular pressure (IOP) increases by 2-4 mm Hg when body position is changed from standing or sitting erect to supine.1,2 IOP elevations, which range up to three times normal, are observed during complete gravity inversion (i.e., whole-body, head-down tilt).3-5 Intermediate angles of head-down tilt produce less pronounced elevations of IOP.2,5 The IOP elevations which accompany these postural variations appear to result from increases in choroidal vascular volume and episcleral venous pressure that are associated with the cephalad fluid shift occurring during head-down tilt due to a gravity-induced, orthostatic venous pressure gradient.5,6 Indeed, similar IOP elevations have been observed in experiments simulating the fluid shifts known to occur in the microgravity environment of space flight.4

Chronic elevations of IOP, such as occur in primary open angle glaucoma, are associated with significant visual impairment, but the visual effects associated with acute IOP elevations are not as well documented. Evidence suggests that acute elevations in IOP do not effect the human flash ERG until IOP approaches or exceeds diastolic ophthalmic artery pressure,7 although one recent report suggests this is only true for photopic conditions.8 The amplitude of the human pattern reversal visually evoked cortical potential (VEP) is significantly reduced when IOP is elevated during gravity inversion.9 This reduction in VEP amplitude during gravity inversion has been interpreted as evidence that acute IOP elevation produces transient neural dysfunction affecting the proximal retina and/or the optic nerve.9 Since the human VEP is an indirect reflection of retinal function, this amplitude reduction could also result from the effects of gravity inversion on cerebral function. Additional study is necessary, therefore, to more precisely localize the neural dysfunction associated with gravity inversion. Furthermore, the relationship between body position, IOP and visual function has not been precisely defined. Both IOP and pressure in the supraorbital vein increase systematically as body position is varied from supine to fully inverted.5,6 The VEP amplitude reductions that are observed during gravity inversion could either parallel the increase in venous pressure or they could result from a gating process not activated until venous pressure exceeds a criterion level.

This report describes two experiments evaluating the variations in IOP, as well as neural functioning of the retina and visual cortex, associated with whole-
body, head-down tilt and expands upon our previously published data.\textsuperscript{10}

**Materials and Methods**

All of the subjects were visually normal, young adults (18 to 36 years of age) from whom informed consent was obtained prior to participation. Inclusion criteria included: visual acuity corrected to 20/30 or better; IOP less than 19 mm Hg and no history of ocular, cardiovascular or neurological disease.

Each subject was positioned on a "Backswing" tilt table which had been modified to hold a video monitor (Nicolet NIC-1004; Nicolet Instrument Co., Madison, WI). The video monitor was used to generate a high-contrast (76%), black-white checkerboard array which subtended 20.6° at the 0.5 m test distance. The visual stimuli were chosen so that both steady-state (60' of arc checks counterphasing at 15 reversal per second) and transient (15' of arc checks counterphasing at 3.6 reversals per second) responses could be examined.

Monocular (OS) pattern-reversal electroretinograms (prERGs) and pattern-reversal VEPs (prVEPs) were recorded simultaneously, amplified (80k), bandpass filtered (1-30 Hz, -3dB points) and averaged over a 200 msec epoch. A minimum of 150 averages was included in each response. The prERGs were recorded with a DTL-microfiber electrode placed in the inferior cul-de-sac and referenced to a Ag-AgCl electrode positioned on the skin adjacent to the lateral canthus. The prVEPs were recorded with a Ag-AgCl electrode positioned 1-2 cm superior to the inion in the mid-sagittal plane and referenced to similar electrode placed near the mastoid. Electrode impedance was monitored in all testing positions to ensure a constant value. IOP was recorded using an applanation technique (Perkins; Clement Clarke, Inc., Reynoldsburg, OH) from the contralateral eye (OD), which was otherwise occluded. The differences in IOP between the fellow eyes of individual subjects was less than or equal to 2 mm Hg.

Two separate experiments, each involving ten subjects, were conducted. In both experiments baseline IOP, prERG and prVEP measurements were made with the subject in an upright position (ie, +90°). In the first experiment IOP, prERG and prVEP measurements were made with the subject in an upright position (ie, +90°). In the first experiment IOP, prERG and prVEP measurements also were made with the subjects supine (ie, 0°), fully inverted (ie, -90°) and at three intermediate angles of whole-body, head-down tilt (-6°, -30° and -60°). The order in which the subjects were placed in each of the six positions (+90°, 0°, -6°, -30°, -60°, -90°) was randomly assigned. Each measurement was completed within 1 min of

![Fig. 1. Typical prERG and prVEP waveforms for the steady-state and the transient response conditions. Amplitudes were measured between the points indicated by arrows. Implicit times were measured from stimulus onset (ie, start of the trace) to the second arrow.](image)

the time the subject was placed in position. Immediately following each measurement the subjects were returned to an upright position for a period of at least 2 min before proceeding to the next test position. This sequence was performed twice for each of the two visual stimuli.

In the second experiment baseline measurements were made initially and then the participants were placed in the -6° position. PrERGs and prVEPs were measured at 2.5, 12.5, 22.5, 32.5, 47.5, 62.5, 92.5 and 122.5 min following the baseline determination, while IOP was measured at 2.5, 32.5, 62.5 and 122.5 min post-baseline. In a control experiment, prERGs and prVEPs were recorded from four of the same participants who were maintained in the upright position for 2 hr.

The prERG and prVEP amplitudes, defined as the difference in voltage between the initial negative and major positive components of each response (Fig. 1), were determined from each waveform. The latency (transient response) or phase (steady-state response) of each response was also measured. The data then were analyzed statistically using a one-way, repeated measures analysis of variance.

**Results**

Neither the latency nor phase characteristics of the biopotentials varied systematically in either experiment. Therefore, the remainder of this report will focus on the amplitude data as well as IOP.

In the first experiment significant elevations in IOP were observed as a function of whole-body, head-
Fig. 2. Intraocular (IOP) pressure plotted as a function of the sine of the angle of whole-body, head-down tilt. Error bars represent the standard error of the mean. n = 10.

These IOP elevations occurred within one minute of when body position was altered and systematically varied as a function of the angle of tilt (P < 0.00001). Complete gravity inversion produced a 3-fold increase in IOP, while intermediate angles of head-down tilt produced smaller IOP elevations. The relationship between body position and IOP is fit by a sinusoidal function of tilt angle when the head is below the level of the heart, as would be expected based upon the pressure gradient in the venous blood column.

In all positions of head-down tilt, and for both types of visual stimuli, the average prERG and prVEP amplitudes decreased. The largest reduction in amplitude was observed in the prERGs (up to 20%), whereas the prVEPs were less effected (at most a 14% reduction). However, there was considerable intersubject variability and only the steady-state prERG exhibited a statistically significant decrease as a function of tilt angle (P < 0.006) (Fig. 3). For this case, the reduction in amplitude paralleled the tilt angle. However, we were unable to precisely define the relationship between body position and either
prERG or prVEP amplitude because of the intersubject variability.

In the second experiment, 6° whole-body, head-down tilt produced an immediate (within 1 min) elevation in IOP of approximately 3.5 mm Hg ($P < 0.0008$). This increase persisted during the first hour, but then decreased by about 1 mm Hg during the second hour (Fig. 4).

Both the steady-state and transient prERGs were significantly reduced in amplitude ($P < 0.005$ and $P < 0.0007$, respectively), but only the transient prVEP was reduced significantly ($P < 0.0007$) during this experiment (Fig. 5). The largest amplitude reductions for the transient responses occurred at 20 min and again at 90 min, while the steady-state prERG showed its greatest reduction only at 20 min. During prolonged head-down tilt the observed amplitude reductions were consistently larger for the transient response condition (as much as 45%) as compared to the steady-state response condition (less than 33%).

In all instances there was a decrease in amplitude by the time of the first measurement (2.5 min). On the
other hand, during the control experiment, in which the subjects remained upright throughout the 2 hr test period, the prERG and prVEP amplitudes remained at or above baseline levels.

The results confirm the systematic IOP elevation that occurs when head position is lowered. We also have observed a reduction in neural functioning, evident both in the retina and in the visual cortex, that varies with body position. Our findings indicate that there is an acute reduction in visual neurophysiologic functioning associated with acute elevations of IOP.

Discussion

There is considerable evidence that altering body position effects intraocular pressure. Intraocular pressure is 2–4 mm Hg higher in a supine as compared to a sitting position and up to 3-fold increases in IOP have been observed during complete gravity inversion. In addition, Tarkkanen and Leikola observed IOP elevations for intermediate positions of whole-body, head-down tilt (1.6-fold at –30° and 2.4-fold at –75°). Our data, along with the recent results of Draeger and Hanke, suggest that IOP increases as body position is altered from upright (+90°) through intermediate angles of head-down tilt to complete gravity inversion (–90°). This interpretation is consistent with each of the earlier studies of the influence of postural changes on IOP. Our data also support the notion that during relatively brief (ie, 1 min or less) changes in body position IOP varies as a sinusoidal function of the angle of head-down tilt, as would be expected from a consideration of the hydrostatic properties of the venous blood column. This increase in IOP probably is the result of a gravity-induced increase in the filling of the choroidal vessels and the episcleral venous pressure. As was suggested by Draeger and Hanke, however, some other factors (eg, autoregulation) must be involved when head-down tilt is sustained, since IOP continues to change while body position remains constant.

Significant reductions in both prERG and prVEP amplitude were also noted during head-down tilt. When whole-body, head-down tilt was brief (ie, 1 min or less), the amplitude reductions ranged up to 20% for the prERG and up to 14% for the prVEP. When head-down tilt was prolonged, these amplitude reductions increased, reaching a maximum after approximately 20 min. Using a similar technique, Friborg and Sanborn found a 17% reduction in the transient prVEP after 3 to 4 min of gravity inversion. This value is in agreement with the 11% reduction in the amplitude of the transient prVEP that we noted after 1 min of gravity inversion. Therefore, we conclude that brief changes in body position can produce significant reductions in the amplitude of the prERG, while a slightly longer period of –6° tilt (20 min) also can produce significant reductions in both the prERG and the prVEP. These acute reductions in prERG and prVEP amplitude are evident with intermediate angles of whole-body, head-down tilt as well as during complete gravity inversion.

prERG and prVEP amplitude reductions which far exceed the magnitude of change observed during 1 min of gravity inversion were noted after 20 min of –6° head-down tilt. This striking result was unexpected. When head-down tilt (experiment 2) is continued for more than 20 min the magnitude of the prERG and prVEP amplitude reduction either remains constant or gradually decreases. The time course of this change appears to parallel the variation in IOP that is maximal after 15 to 30 min of head-down tilt and is followed by a period in which IOP decreases slightly and then remains constant. However, the degree of intersubject variability in prERG and prVEP amplitude reductions precludes determination of the precise relationship between amplitude changes and IOP elevations associated with postural change.

While the current results demonstrate that visual evoked retinal and cortical potentials are altered by varying body position, the specific neural processes underlying these changes remain ill-defined. Whole-body, head-down tilt must effect some neural elements in the retina since significant prERG alterations were found. The prVEP amplitude reduction then could be an indirect, and possibly distorted, reflection of the retinal dysfunction. The observed prERG amplitude reductions may result from altered bioelectrical activity in the proximal retina and/or optic nerve since there is some evidence that the prERG represents ganglion cell function. This interpretation is supported by the evidence that acute elevations of IOP do not influence the photopic flash ERG (which is generated in the distal retina) until IOP approaches or exceeds diastolic ophthalmic artery pressure. However, the neural generators of the human prERG are still the subject of some controversy. In the second experiment the amplitude reductions of both the retinal and the cortical responses consistently were greater for the transient conditions than for the steady-state conditions. This might indicate that some neural channels in the retina (ie, sustained and transient) are differentially affected by postural changes.

Key words: intraocular pressure, pattern electroretinogram, visual evoked potential, retinal function, gravity inversion
References