Vascular and Neural Changes During Body Inversion: Preliminary Findings

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Abstract

This report examines the influence of gravity on physiological fluid pressures within the body. The interest in the influence of gravity on ocular structures and function is topical not only because of the current fashion of body inversion devices as forms of exercise, but also the ocular consequence of a cephalic redistribution of blood with its subsequent effect on IOP during the micro-gravity environment of spaceflight. For the ophthalmic practitioner, the effect of body orientation on intraocular pressure and on the blood supply to the eye is of practical importance.

In this paper the influence of precisely controlled body positions on the intraocular pressure, central retinal artery pressure, and systemic blood pressure is presented. Deficits in visual neural function resulting from body inversion are documented. Clinical implications of these findings are discussed.

Introduction

In recent years, considerable interest has developed in the area of body posture and its effects on human ocular structures and visual function. This interest is a result of the popularity of gravity inversion devices advocated as a form of exercise and/or therapy, as well as its applicability to aeronautical and space research. Body inversion procedures have also been used in conjunction with techniques such as suction ophthalmodynamometry to identify whether the absolute level of intraocular pressure (IOP) or retinal vas-

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cular perfusion pressure (RVPP) is the more important factor in the etiology of glaucomatous optic nerve damage

In the inverted (head down) position, the blood volume is displaced to the headward parts of the body resulting in several readily observable changes in the face and neck region. Several researchers (Klatz et al, 1983, 1985; LeMarr et al, 1984) have reported a significant rise in systemic systolic and diastolic blood pressure immediately on inversion with a return to normal immediately on resuming the upright position.

Correlated with an increase in systemic blood pressure is a significant rise in systolic and diastolic ophthalmic artery pressure (more commonly referred to as central retinal artery pressure or CRA pressure), as measured by clinical ophthalmodynamometry (Friberg et al, 1984; Friberg & Weinreb, 1985; Goldman et al, 1985; Plocher, 1985).

Intraocular pressure is likewise significantly increased to approximately twice baseline values on inversion (LeMarr

et al, 1984; Mansour et al, 1984; Weinreb et al, 1984; Friberg & Sanborn, 1985; Draeger & Hanke, 1986). The increase in IOP occurs almost immediately and remains elevated during the period of inversion (Friberg & Weinreb, 1985). IOP returns to its pre-inversion level within 15 seconds (Weinreb et al, 1984) to several minutes (LeMarr et al, 1984) on resuming the upright position. The increase in IOP with inversion is believed to be due to increased episcleral venous pressure (Klatz et al, 1983) as well as orbital congestion and increased ocular blood volume (Smith & Lewis, 1985).

The structural integrity of ocular tissues on inversion has been examined by various investigators. Friberg et al (1984) failed to find any retinal hemorrhages, cotton wool spots, or leakage of fluorescein dye within 30 minutes after inversion in normal volunteers. Plocher (1985) found no observable change in either ocular blood flow or appearance of retinal vasculature by retinal photography. However, using fluorescein angiography,

Table I

Body Orientation (degrees)	IOP (mm Hg)	Systolic CRA Pressure (mm Hg)	Diastolic CRA Pressure (mm Hg)	RVPP (mm Hg)
0	20.60 ± 1.60	88.30 ± 3.42	56.60 ± 1.98	49.22 ± 3.48
20	20.10 ± 1.39			
40	21.20 ± 1.04	91.30 ± 3.33	62.20 ± 1.94	53.18 ± 2.61
60	22.70 ± 1.38			
80	24.00 ± 1.51	100.50 ± 2.51	66.50 ± 2.58	56.87 ± 2.93
100	27.20 ± 1.85			
120	30.90 ± 1.17	124.20 ± 3.30	87.40 ± 3.60	71.92 ± 3.29
140	35.80 ± 1.14			
160	39.60 ± 1.02			
180	42.30 ± 1.47	147.20 ± 3.84	114.50 ± 3.03	85.80 ± 3.25

* Values represent group averaged mean +/− 1 standard error of the mean (SEM)

Friberg & Weinreb (1985), noted vaso-constriction of retinal arterioles on inversion. They attributed the constriction of arterioles to a manifestation of retinal vascular autoregulation.

Other reported findings associated with inversion include dilatation of conjunctival vessels (Friberg et al, 1984; Friberg & Weinreb, 1985), periorbital petechiae (Plocher, 1982) subconjunctival hemorrhages, orbital congestion, conjunctival hyperemia and epiphora (Friberg & Weinreb, 1985).

Changes in the functional aspects of vision in body inversion have received relatively little attention in the past. Visual acuity (Friberg et al, 1984; Friberg & Sanborn, 1985) and visual fields (Weinreb et al, 1984; Sanborn & Friberg, 1985) have been reported to be unchanged after inversion despite the significant rise in IOP. Optic nerve function, assessed by the amplitude of pattern reversal visual evoked potentials (VEPs), has been reported to be significantly impaired when compared with pre-inversion values (Friberg & Sanborn, 1985). Curiously, a similar elevation in IOP caused by suction ophthalmodynamometry did not cause as great a degradation in the VEP amplitude (Friberg & Sanborn, 1985; Srebro et al, 1985). A variety of body orientations with associated effects on IOP also occur during the micro-gravity environment associated with spaceflight. In a recent study, the contrast sensitivity function was evaluated before, during and after spaceflight (Ginsburg & Vanderploeg, 1986). While statistically significant changes in sensitivity were found, these were sufficiently small so as not to affect visual performance. However, these changes were not related to changes in retinal vascular perfusion and therefore provide directions for future research needs. Currently, in our lab (unpublished data) we are investigating the correlation between altered RVPP and contrast sensitivity. Preliminary findings have linked altered RVPP with significant changes in the contrast sensitivity function, particularly in the middle spatial frequencies (3 to 6 c/deg).

In as much as functional deficits occur prior to clinically observable changes in ocular structures, an examination of alterations in neural function is likely to yield useful information on causal mechanisms in experiments designed to examine various disease processes. Objective monitoring of retinal function by non-invasive neurophysiological testing, electroretino-

Table II

Parameter Measured	Method of Raising IOP	
	Inversion	Ophthalmo-dynamometry
Intraocular Pressure	38.13 ± 2.02 (mm Hg)	38.60 ± 1.87 (mm Hg)
Change in scotopic blue flash ERG b-wave amplitude	− 10.47 ± 1.88 (%)	− 31.79 ± 8.23 (%)
Change in photopic red flash ERG b-wave amplitude	− 7.67 ± 3.69 (%)	− 4.80 ± 4.93 (%)
Change in scotopic blue flash ERG b-wave implicit time	+ 2.25 ± 2.21 (%)	+ 3.23 ± 1.93 (%)
Change in photopic red flash ERG b-wave implicit time	− 0.21 ± 1.17 (%)	− 3.35 ± 2.25 (%)

Values represent the group averaged percent (%) change from baseline +/− 1 SEM

% change = (experimental — baseline)/baseline X 100

graphy (ERGs), provides an ideal method for evaluating the consequences of abnormal fluid pressure relationships in the eye. When combined with objective measures of optic nerve function by VEPs, ERGs may provide new insight into the etiology of glaucomatous nerve damage.

In this report we present the results of some preliminary studies into the neural reaction at the retina to altered IOP and RVPP. The objectives of our pilot studies were: (1) to develop an apparatus to manipulate systemic blood pressure, IOP, and RVPP by non-invasive procedures; (2) to document precisely the nature of fluid pressure changes in the body by controlled body inversion procedures; (3) to determine the effect of measured changes in IOP and RVPP on neural function at the retinal level; and (4) to outline the clinical implications of the measured ocular and neural reactions to altered RVPP.

Methods and Procedures

All experimental procedures in this study complied with the guidelines of Human Experimentation Ethics and were approved by the University of Waterloo, Human Experimentation Committee.

Outline of Testing

The aims of the initial phase of experimentation were to evaluate the special apparatus designed to allow precise body orientation and to establish the relationships among IOP, CRA pressure, and systemic BP across various angular positions of the cephalo-caudal axis of the body. This was done for a group of 10 paid volunteers between the ages of 21 and 29 years who had good systemic and ocular health and normal IOPs. For this group, IOPs were measured at 20 degree intervals between 0 and 180 degree body positions; CRA pressure in 40 degree intervals, and systemic BP at 0, 100, and 180 degrees.

The second phase of experimentation involved comparing the effects of IOP raised by (1) body inversion and (2) ophthalmodynamometry, on the scotopic and photopic flash ERGs in a second group of ten qualified subjects in the same age group as the first. For this phase of our study, the IOP was manipulated to create an increase of about 120% by both procedures. This allowed a comparison of the effects of similar IOP levels but dissimilar RVPPs, on retinal function as monitored by the flash ERGs. During

body inversion, the RVPP is increased simultaneously but more rapidly than the IOP. In ophthalmodynamometric scleral indentation the IOP is increased but the RVPP is reduced.

For the body inversion procedure, the maximum increase in IOP was achieved by rotating the body axis into the 180 degree position (total body inversion). The IOPs achieved by this procedure were matched for each subject by controlled ophthalmodynamometric compression of the globe while the subject was seated in an upright position with his head immobilized within a painless head-restraint device. Diastolic and systolic CRA pressures were measured in each of the test conditions described above in order to allow calculation of the RVPPs.
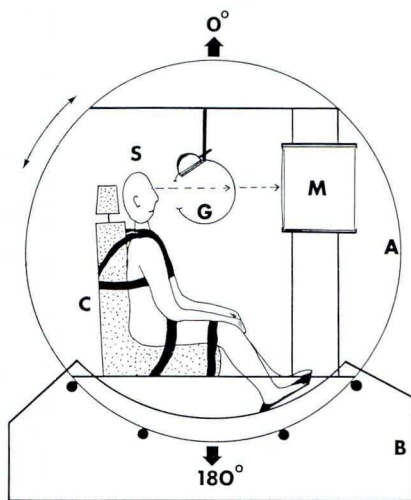
Figure 1

Schematic of the body inversion apparatus used to raise the IOP. It was composed of a circular steel frame (A) resting on roller bearings within a non-rotating base (B). During inversion the subject (S) was secured to a padded chair (C) attached to the circular frame. A strobe attached to a diffusing sphere (G) was used to present flashes for ERGs in ganzfeld mode. Pattern stimuli could be presented on a high resolution monitor (M) when G was rotated out of the line of sight. The solid arrows indicate the position of the subject's head in the upright (0 degree) and inverted (180 degree) positions.

Body Inversion Apparatus

The apparatus to control body orientation and thereby the IOP and RVPP is shown in Fig. 1. It consisted of a robust circular metal frame that contained a padded seat

and head rest with supporting belts as well as apparatus to provide controlled visual stimulation. This latter apparatus consisted of a ganzfeld unit for the diffuse flash needed to elicit the ERG and a quality monitor for electronic generation of reversing checkerboard patterns used
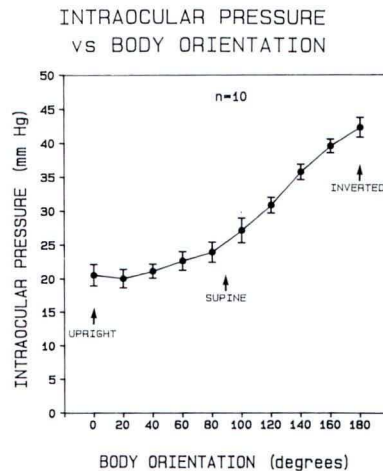

Figure 2

Change in IOP with body orientation. In this and subsequent figures the zero degree position corresponds to an upright seated position while the 180 degree orientation denotes the completely inverted (head down) seated position. For this group of ten subjects, the IOP increased in a curvilinear manner from the upright through the supine and into the inverted position. The solid bars through data points for this and subsequent figures represent $+/- 1$ SEM (standard error of the mean).

in visually evoked cortical potential studies. The entire frame with stimulators sat on roller bearings and could be rotated by a winch in a continuous fashion into any angle between 0 and 180 degrees. The 0 degree position corresponded to a seated upright position for the subject while the 180 degree corresponded to complete body inversion.

IOP and CRA Pressure Measurements

For convenience, all measurements were taken for the right eye. The pupil was dilated with one drop 1% cyclopentolate hydrochloride (1% Cyclogyl). IOP was determined with a calibrated gas driven tonometer (Digilab pneumatonometer) following administration of a topical anaesthetic, 0.5% proparacaine hydroch-

loride (Ophthaine). A minimum of three IOP measurements were made in each body position following a one minute stabilization period at each new position.

Ophthalmodynamometry for measuring CRA pressures was performed by two investigators using a spring ophthalmodynamometer (Luneau, France). One experimenter placed the circular footplate (diameter 7.3 mm) of the dynamometer 2 to 3 mm nasal to the corneo-scleral limbus while the subject looked to his right. The plunger was advanced perpendicular to the sclera until the second investigator using an ophthalmoscope, reported a pulsation of the central retinal artery at the optic nerve head. Three measurements were made of this diastolic pressure. The sclera was then indented further until the pulsation ceased and the central retinal artery collapsed. This was recorded as the systolic pressure and a minimum of two such measurements were made.

Ophthalmodynamometer scale readings were converted into mm Hg units using the Bedavanija conversion scales which take into account the resting intraocular pressure. Mean time-averaged CRA pressures (P_m) were derived from the formula, $P_m = P_{diastolic} + (P_{systolic} -$
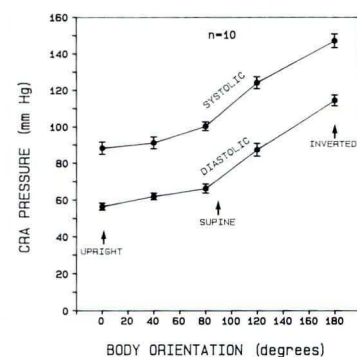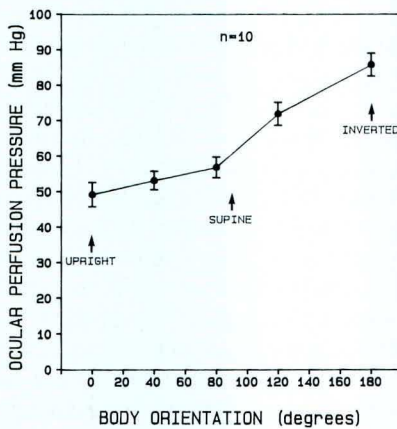

Figure 3

Group averaged values for ten subjects showing the increase in the diastolic and systolic central retinal artery pressure with body orientation as measured by ophthalmodynamometry. As was the case with the IOP (Fig. 2), the CRA pressure increased in a manner that approximated a curvilinear function. Changes in diastolic and systolic pressure paralleled each other throughout the entire range of body positions. This congruity of data provides indirect validation of the experimenters' method of measuring CRA pressure values.

VASCULAR PERFUSION PRESSURE
vs BODY ORIENTATION

Figure 4

Group averaged retinal vascular perfusion pressure data for the ten subjects whose IOP and CRA values are shown in Figs. 2 and 3. While the IOP more than doubled with complete body inversion, the calculated perfusion pressure increased by about 70%. This slight divergence of IOP and RVPP data with inversion may be due to (1) a limitation on the amount of vascular infiltration possible with inversion, (2) an accelerated rise in the IOP due to individual differences in globe expandability (scleral rigidity), or (3) a constant error in the subjective determination of the onset and cessation of the central retinal artery pulsation with ophthalmodynamometric indentation of the eyeball.

P diastolic) × 0.42 (Weigelin & Lobstein, 1963). Retinal vascular perfusion pressure was calculated as:

$$RVPP = P m - IOP$$

Systemic BP

Diastolic and systolic pressure readings were recorded from the brachial artery with the arm elevated to eye level. Measurements were made with an electronic auto-inflation, digital readout blood pressure unit (Taylor, Model A-200). Three determinations of systolic and diastolic pressure were made for each body position.

Neurophysiological Testing

Both scotopic and photopic flash ERGs were recorded when IOP was increased by body inversion and globe compression by ophthalmodynamometry. In both procedures the pupil was dilated with one

drop 1% cyclopentolate hydrochloride. During body inversion procedures, ERGs were obtained after one minute pressure stabilization for body orientations of 0 and 180 degrees. Scotopic ERGs were recorded after 30 minutes of dark adaptation in response to high intensity blue flashes (Grass PS22 photostimulator, X16) under ganzfeld presentation. The blue flashes insured a rod isolated ERG. Following a 2 minute period of light adaptation to a white ganzfeld background (73 lux) ERGs were recorded to scotopically matched red flashes. The red flash insured a cone dominated ERG. A DTL-type fibre electrode placed in the lower conjunctival fornix of the right eye served as the active electrode. A commercially prepared pre-gelled Ag/AgCl electrode placed about 1.0 cm lateral to the temporal canthus acted as the reference electrode, and a similar electrode on the wrist served as the electrical ground. Each trial consisted of an average of 15 flashes delivered at a rate of 0.3 Hz. Responses were

SYSTEMIC B.P. vs BODY ORIENTATION
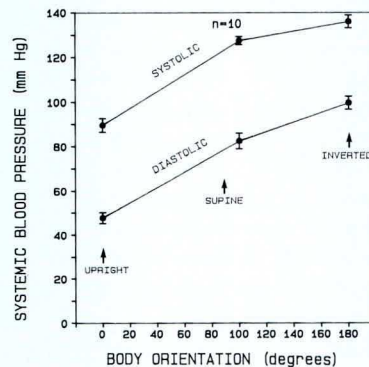
Figure 5

Group averaged trend for ten subjects showing the increase in both diastolic and systolic systemic blood pressure with the degree of body inversion. Note that while the pressure difference between diastole and systole (about 40 mm Hg) is within normal limits, the absolute pressure values for both are far below the normally measured systemic blood pressures. This occurred because all pressure measurements were made with each subject's arm held alongside his head so that the brachial artery was at eyeball level. This technique allowed a better estimate of the systemic blood pressure entering the eyeball. With the arm in its normal resting position, sphygmomanometry measurements reflect the blood pressures at the level of the heart which are some 40 mm Hg higher.

electronically processed by filters providing a bandpass between 1 and 250 Hz, amplified on a clinical averager (Nicolet CA1000), and stored on floppy diskettes for subsequent retrieval and analysis.

In a separate session, prior to ERG testing during IOP elevation by ophthalmodynamometry, the cornea was anaesthetized by one to two drops of topically applied 0.5% proparacaine hydrochloride (Ophthaine). Thereafter, the dark adapted subject, with the ERG fibre electrode already within the inferior fornix, was seated upright with his head fixed within a head and chin support mechanism. The ophthalmodynamometer, used to raise IOP by scleral indentation, was secured within a rotatable x-y-z micromanipulator which allowed precise positioning of the footplate at right angles 3 to 5 mm temporal to the corneoscleral limbus of the right eye. The placement of the probe was done under Kodak safelight illumination to preserve dark adaptation. Subsequent scotopic and photopic ERG testing was identical to that carried out in the inversion procedures. ERG analysis consisted of measuring the amplitude and implicit time of the b-wave component of each ERG.

Results

Individual IOPs increased slowly by 4 to 5 mm Hg on body rotation from the upright to a supine position. Further rotation caused a much quicker rise in IOP, ending on complete inversion with an IOP value approximately twice that measured for the baseline (upright) condition. The group averaged data (n = 10) for the IOP-body orientation function is shown in Fig. 2.

Systolic and diastolic CRA pressures increased in a fashion similar to that seen for IOPs with body tilt from 0 to 180 degrees. The group-averaged data (n = 10) for the CRA pressure-body orientation function is shown in Fig. 3. The calculated RVPP also increased in a similar two stage manner with inversion (Fig. 4). The systemic systolic and diastolic blood pressure measured with the arm at eyeball level (see Methods) increased by about 30 mm Hg on body rotation from the upright to supine position and then increased a further 10 to 20 mm Hg on complete inversion. (Averaged systolic and diastolic blood pressure values are about 30 mm Hg higher when measured with the arm held alongside the torso with the brachial artery in line with

the heart.) The group averaged (n = 10) changes in systemic systolic and diastolic blood pressures with body inversion are shown in Fig. 5. Averaged values for the IOP, CRA pressure, and RVPP obtained in the first phase of the study for each of the tested body positions are given in Table 1.
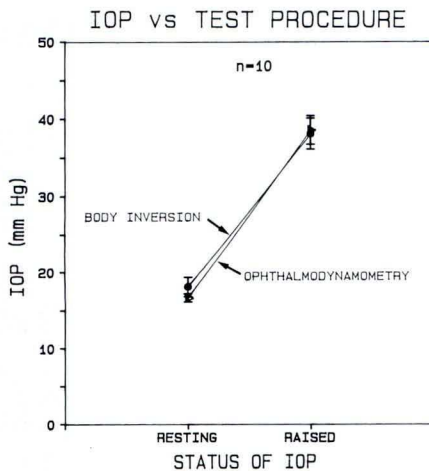
IOP vs TEST PROCEDURE

Figure 6

Group averaged data for a second group of ten subjects showing the congruence of resting and test levels of IOP as raised by body inversion and ophthalmo-dynamometric compression of the globe.

For the second phase of testing where the effects of IOP raised by body inversion and ophthalmodynamometry on flash ERGs were examined, the group averaged IOP values for inversion and ophthalmodynamometry were 38.13 +/− 2.02 mm Hg and 38.60 +/− 1.87 mm Hg, respectively (Fig. 6). Typical ERGs obtained for baseline and each test procedure are shown in Fig. 7. Elevating the resting IOP by either body inversion or ophthalmodynamometry caused the amplitude of the b-wave to decrease in both scotopic and photopic conditions. However, the magnitude of the reduction in the b-wave amplitude varied with the method of increasing the IOP and the state of adaptation of the retina. Under scotopic conditions, the size of the reduction was much greater by ophthalmodynamometry (about 32%) than body inversion (about 10%). Under photopic conditions the reduction in the b-wave amplitude was nearly equal for ophthalmodynamometry (about 5%) and body inversion (about 8%). This difference, shown in Fig. 8, appeared more related to the RVPP than absolute level of IOP. For example, the

RVPP given within brackets in Fig. 8 indicate that the RVPP was about 2.7 times greater in body inversion than during ophthalmodynamometry, in the presence of nearly identical raised levels of IOP. Thus the greater RVPP appeared to hold the loss in b-wave amplitude to a modest level. In photopic testing conditions, the differences in RVPP was associated with a small reduction in amplitude of the ERG b-wave and this decrease was very similar for both methods used to raise the IOP.

The implicit time of the ERG b-wave was altered only slightly (less than 3.5%) under scotopic or photopic conditions by either method of IOP elevation (Fig. 9). Thus the implicit time of the b-wave was much less susceptible to pressure effects than the b-wave amplitude. Changes in the b-wave amplitudes and implicit times with respect to baseline value are summarized in Table II according to test condition.

Discussion

Our findings are in agreement with those of previous researchers, who also showed

that IOP is altered with body tilt (Krieglstein et al, 1978; Draeger & Hanke, 1986) and is significantly increased in the head-down position (Klatz et al, 1983; Srebro et al, 1985). Concomitant is a non-linear increase in central retinal artery pressure. In earlier studies Smith & Cogan (1959) had reported that the ophthalmic artery pressure changes little between the upright and supine position while others (Klatz et al, 1983) showed that it rises signifi-cantly on inversion not unlike the changes in CRA pressure shown in Fig. 3.

Many changes in body orientation also occur in manned spaceflight. Draeger et al (1986) reported that the IOP after launch increased 20 to 25%. These changes may have been caused exclu-sively by changes in body orientation and the associated vascular changes. Alterna-tively they may have been as yet uniden-tified residual pressure effects subsequent to large G-forces affecting the astronauts during the launch. Dynamic changes in IOP during launching procedures have not been fully documented and deserve investigation in order to determine the
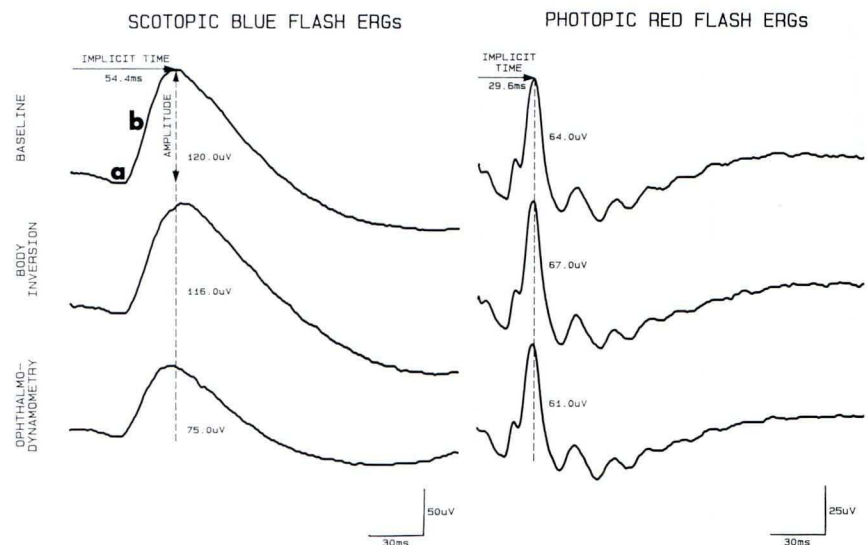
Figure 7

Digitally averaged scotopic (rod isolated) and photopic (cone dominated) flash ERGs showing the morphology of the a-wave and b-wave components, the para-metric measures made in the study, and the typical changes in individual ERGs from baseline in body inversion and ophthalmodynamometry procedures. Note that while the implicit time of the scotopic b-wave varies only slightly across test conditions, its amplitude shows a

progressively larger drop with body inversion and ophthalmodynamometry. In comparison, the photopic ERGs show little change in both amplitude and b-wave implicit time across test conditions. The numbers within each wave represent the trough-to-peak amplitudes of the b-wave. Sample times for the baseline b-wave implicit times are also presented to emphasize the time difference between rod and cone responses to light.

exact effect of probable large increments in IOP on visual function during that critical phase of spaceflight.

For our subjects, although IOP increased by an average of 21.7 mm Hg on inversion, the mean time-averaged CRA pressure increased by 56.46 mm Hg resulting in a net increase in RVPP as shown in Fig. 4. This finding is in agreement with that reported by Friberg & Weinreb (1985), although Plocher (1985) stated that there was little net change in perfusion pressure with inversion. The reason for these reported differences is unclear.

The observation that the ERG b-wave was reduced by only 10% with a doubling of IOP by body inversion and reduced by about 32% when the IOP was doubled by ophthalmodynamometry suggests a primary role for vascular perfusion in the preservation of neural function. The great increase in IOP in itself, albeit a short-lived one, was insufficient to cause altered function of the outer two-thirds of the neural retina, the site of origin of the flash ERG. Increased vascular perfusion was apparently able to hold neural activity to within 10% of baseline levels. A similar vascular preservation of neural function mechanism may be operative in patients with raised IOP levels but no measurable changes in the optic nerve head appearance or function. The ERG b-wave implicit times and overall waveform were unaltered under all test conditions. This was not surprising since clinically significant delays in ERGs are usually associated with retinal degeneration and acute ischemic conditions (Brunette, 1982; Brunette et al, 1983).

That the electrical function of the retina is dependent upon its oxygen supply is well established (Flower & Patz, 1971; Alder & Constable, 1981; Grehn & Prost, 1983). The relationship between the ERG and IOP has been examined by several studies (Ward, 1967; Uenoyama et al, 1968a, 1968b; Sipperley et al, 1973; Benedikt et al, 1974; Bartl, 1978) but most of these have raised the IOP to very high levels, well above mean CRA perfusion. Not surprisingly, these studies have indicated a decrease in the amplitude of the ERG in acute ischemia. The present study however, indicates that the ERG is altered by much lower IOP levels. Furthermore, the scotopic b-wave amplitude may be more vulnerable to an increase in IOP (with a concomitant decrease in perfusion pressure) than its photopic counterpart. Differential vulnerability of the scotopic

ERG b-WAVE AMPLITUDE vs IOP
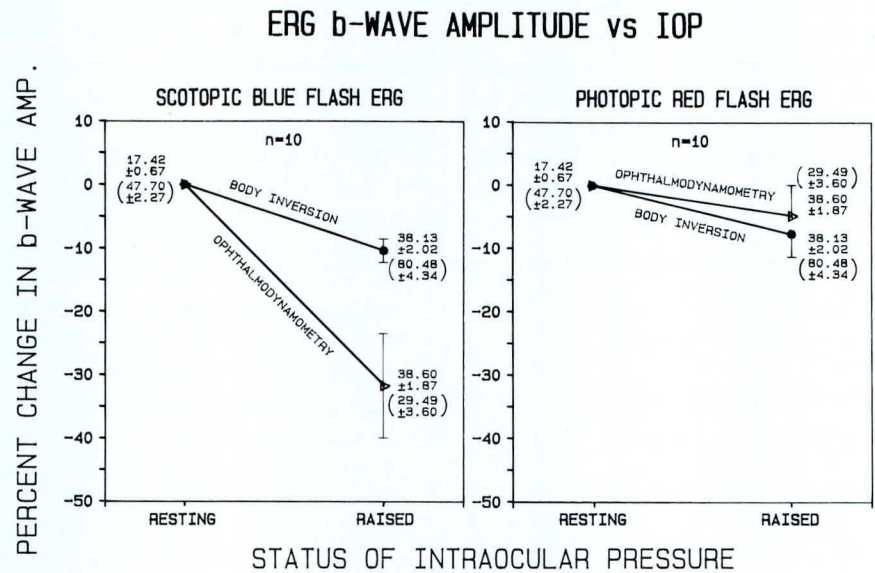
Figure 8

Group averaged data for ten subjects showing the reduction in scotopic and photopic ERG b-wave amplitudes when the resting IOP (17.42 +/− 0.67 mm Hg) level was raised by body inversion (38.13 +/− 2.02 mm Hg) and ophthalmodynamometry (38.60 +/− 1.87 mm Hg). The numbers within brackets represent the vascular perfusion pressure values for baseline and test conditions. The vascular perfusion pressures associated with each method of increasing the IOP differed significantly from the resting RVPP (P < 0.001) as well as from

each other (P < 0.001) by the paired t-test. Note the larger decrease in the scotopic ERG b-wave when an increase in IOP was accompanied by a decrease in perfusion pressure, as was the case in ophthalmodynamometry. For each test condition the associated change in b-wave amplitude was significantly different from baseline values (P < 0.005) and from each other (P < 0.02). Photopic ERGs showed measurable but insignificant (P > 0.05) reductions in amplitude when IOP was elevated by either ophthalmodynamometry or body inversion.

and photopic ERGs to IOP levels has been reported by Uenoyama et al (1969) for the cat. It is interesting to speculate on the cause of the apparently enhanced sensitivity of the scotopic ERGs (rod function) to raised IOP levels. The rod photoreceptors are absent from the macular area and increase in density outwards from the fovea, reach a peak density about 15 degrees away from the fovea, and then decrease again towards the periphery. The decrease in the scotopic ERG seen with increased IOP may reflect a functional impairment across the entire rod population. More likely, however, it represents a signal dropout from rods in discrete retinal areas, perhaps those found in retinal locations subserved by vessels most easily collapsed by raised IOP. The large decrease in scotopic ERG amplitude with increased IOP may be the electrophysiological counterpart of the psychophysical observation that the nasal visual field is the first to dim, followed by inferior and temporal fields when the

IOP is raised transiently to sufficiently high levels in a normotensive eye. The observation of a pressure dependent sequence of segmental losses of the visual field was first reported by Jaeger et al, 1964. Two anatomical considerations may account for the present electrophysiological observation and the psychophysical findings of Jaeger et al. First, there is about 50% more retina temporal to the optic nerve head than nasally (Jaeger et al, 1964; Reed & Drance, 1972). Thus arterioles headed for the most distal temporal retina must traverse a greater distance and nourish a larger retinal area than arterioles headed for nasal areas. The larger travel distance for temporal arterioles may mean smaller vessels supplying less blood at the far temporal periphery. If this is the case, factors affecting retinal vascular flow may be manifest as functional deficits first at the nasal visual field followed by losses in sections of the visual field corresponding to retinal areas with an inferior blood

supply. Second, Duke-Elder & Wybar (1961) reported that the general structure of retinal arteries changes with retinal location. Retinal vessels become much thinner towards the periphery because the muscle coat of the medial layer gradually attenuates toward the finest branches of the vessels. This loss of smooth muscle cells may mean decreased mechanical stability in the presence of transient elevations of IOP.

The preservation of macular fields in elevated levels of IOP may parallel the present findings that the photopic ERGs are relatively unaffected when the baseline IOP is doubled. This may reflect differential sensitivity of the dual retinal vascular supply to pressure effects, with the choroidal blood supply being structurally more impervious to raised IOP levels. Alternately, autoregulation of choroidal blood flow may be hypothesized to preserve the photopic ERG. However, even though autoregulation of macular blood flow in relation to perfusion pressure has been shown using blue field entoptic phenomenon (Riva & Loebl, 1977; Riva & Petrig, 1980; Riva et al, 1981; Fallon et al, 1985), it is difficult to imagine an autoregulatory process which could operate over a range of IOPs that extended beyond double the baseline IOP and thereby preserve the photopic ERG.

The results of the present study suggest that loss of retinal responsivity may be more a reflection of vascular perfusion rather than absolute IOP levels. This tentative conclusion parallels the reports of largely preserved optic nerve function in elevated IOP levels if adequate vascular perfusion is maintained (Grehn & Prost, 1983; Friberg & Sanborn, 1985; Srebro et al, 1985). There are several clinical implications resulting from this investigation. The most important of these pertains to the management of low tension glaucoma patients, i.e. those presenting with symptoms characteristic of glaucomatous episodes yet showing IOP values within an acceptable range. Having demonstrated that retinal neurons continue to function relatively unimpaired even if the IOP is more than doubled, when the RVPP is sufficiently increased, it is clear that defective vascular perfusion can result in neural damage when IOPs are in the normal range. Thus the ophthalmic practitioner must be alert to the possibility of encroaching glaucoma even when IOPs are low, visual fields are normal, and the optic nerve heads appear to have normal structural features. Ophthalmodynamometry to measure CRA pressures and to calculate perfusion pressure is indicated whenever the patient's symptomatic profile hints at subclinical glaucomatous processes.

A comparison of IOP measurements in the upright and supine position, may serve as a useful provocative test to detect those patients with abnormal fluid dynamic relationships between the IOP and RVPP. The present data indicates that an increase in the baseline IOP of 4 to 5 mm Hg from the upright to the supine position is an expected physiological response to postural changes. Greater increases in IOP should be viewed with suspicion and initiate further investigations of IOP dynamics.

The very large increases in IOP and systemic BP on inversion should be borne in mind when counselling patients participating in various forms of exercise, muscle relaxation, or meditation involving body inversion. Patients with IOPs on the high side of a normal distribution should be advised of the possible physical, ocular, and visual consequences of such activities. Since short term elevations of the IOP can cause alterations of function of a reversible nature, longer term, repeated elevations are likely to be more detrimental to the physiological process of vision, perhaps in a more permanent way.

Ongoing studies in our lab are directed at more detailed examinations of the changes in retinal and optic nerve function subsequent to altered vascular perfusion. These studies are intended to provide new insight into the causative mechanisms of optic nerve damage in normal and elevated IOPs.
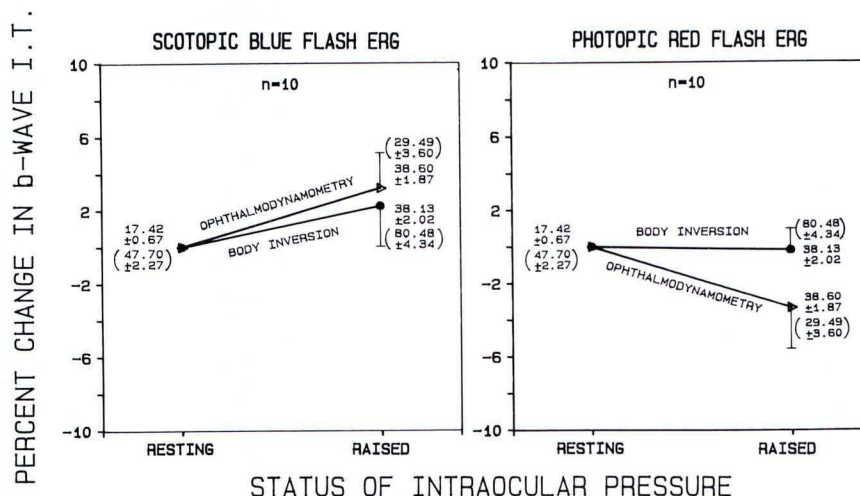
Figure 9

Grouped data for the subject population (n=10) whose ERG amplitude vs IOP function is shown in Fig. 7. These graphs show that the implicit time of the scotopic and photopic ERG b-wave changed only slightly (+/− 4%) when IOP was more than doubled by either ophthalmodynamometry or body inversion. These changes in implicit time from baseline measurements for either test procedure were not statistically significant (paired t-test, $P > 0.10$). The resting and raised IOP levels are indicated beside data points for each method. Perfusion pressure values are shown within brackets for each data point.

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