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The Effects of Roll vs. Pitch Rotation in Humans Under Orthostatic Stress

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ORIGINAL RESEARCH

The Effects of Roll vs. Pitch Rotation in Humans Under Orthostatic Stress

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Background: It has been known since 1953 that pre-exposure to less than $+1\ G_z$ will reduce subsequent $+G_z$-tolerance. With few exceptions, during operational flying, the transition from hypogravity to hypergravity involves roll as well as pitch rotation. We examined the effect of roll vs. pitch rotation while undergoing transition from hypogravity to $+1\ G_z$ on a tilt table. ***Methods:*** Twelve subjects (28–47 yr old) were rotated at $45° \cdot s^{-1}$ from head-up (HU) at 15° relative to gravitational vertical to 135° head-down (HD) and back to the HU position after different HD dwell times. HD dwell times were set at 7, 15, and 30 s. The subject was rotated about the interaural axis (pitch) and about the naso-occipital axis (roll). Both the HD dwell times and axes of rotation were randomized within and across subjects. BP and heart rate were recorded during the HU-HD-HU maneuver. ***Results:*** Analysis of variance, repeated measure design revealed that the rate and magnitude of BP decrease induced by the HD to HU maneuver is significantly higher ($p < 0.01$) in roll than in pitch during all HD dwell times. The decrease of BP at 7s is significantly ($p < 0.01$) higher than at 15s and 30s. Heart rate increases significantly higher ($p < 0.01$) in pitch than in roll at 7s-dwell time. ***Conclusion:*** Our results suggest that the compensatory mechanism to orthostatic stress is more efficient in response to pitch than roll rotation. This is reflected from the findings that the mean magnitude of OH (orthostatic hypotension) and the rate of BP decrease induced by the HD-HU maneuver is significantly greater in roll rotation than pitch rotation. The mean HR increase post HD-HU rotation is significantly higher in the pitch than the roll rotation. The significant rate of BP decrease during HD-HU roll rotation could have important implications for maintaining G-tolerance and spatial orientation during subsequent exposure to hypergravity.

Keywords: orthostatic hypotension, vestibular, G-tolerance.

As early as 1953, von Beckh reported that during in-flight research, alternating weightlessness and hypergravity resulted in decreases of G-tolerance and in the efficiency of physiological recovery (4,5). Another in-flight study (21), a centrifuge investigation (16), and an anecdotal report (9) also indicated that there is a definite deleterious effect on $+G_z$ tolerance when the pilot is subjected to the transition of negative to positive G_z. A recent survey by the USAF indicated that maneuvers of G-transition ranging from 0 to $+0.5\ G_z$ for 3.5 to 5s are common in air combat training missions (18).

A laboratory study using the Coriolis Acceleration Platform (CAP, Naval Aerospace Medical Research Laboratory) demonstrated that subjects' $+G_z$ tolerance was also reduced when acceleration was preceded by zero G_z or $-G_z$. The effect was termed the "Push-Pull Effect" (3). In order to achieve the zero-G or $-G_z$ to $+G_z$ acceleration gradient, the supine subject in the CAP was exposed to a linear translation across the diameter of the platform, and a simultaneous rotation of the subject in the roll (frontal) plane. The resulting acceleration gradient depended on the speed of rotation of the platform. Moreover, the angular accelerative force was primarily at the head level when the subject was in the "head-out" $-G_z$ position.

In the fighter aircraft, one of the common methods used to acquire tactical advantage over the opponent is the split-S maneuver which involves "unloading" the aircraft prior to accelerating. With few exceptions, this maneuver involves rolling and pulling all the G aerodynamically available. A recent Canadian Forces (CF) accident involved a one vs. one neutral engagement. As both aircraft were approaching merge from opposite directions, the wingman maintained less than $+1\ G_z$ for approximately 11s. As the lead lost visual contact of the wingman, the wingman aggressively rolled his aircraft in an inverted position in 2 s and commenced a high G split-S maneuver to capitalize on the tactical opportunity. The aircraft subsequently impacted the ground in a near vertical attitude. There was no attempt at ejection.

The effects of the point roll or unloaded barrel roll maneuver on the subsequent increase in acceleration need to be investigated. Vestibular receptors respond to gravitational forces and postural changes. These vestibular inputs constantly provide the central nervous system with information needed to produce corrections during on-going movements. The fact that BP and oxygenation remain reasonably stable during rapid changes in posture suggests that corrections start at the beginning of a movement that is likely to affect ho-

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meostasis. For example, recent studies in decerebrate cats (28) demonstrated that changes in sympathetic outflow were elicited by pitch rotation (about the interaural axis) but not by roll rotation (about the naso-occipital axis). In humans, clinical observations (19) indicated that a significant number of patients with peripheral vestibular disease were susceptible to orthostatic hypotension after standing from a supine position. Therefore, it is reasonable to postulate that there exists a vestibulo-cardiovascular reflex including those that can affect circulation under orthostatic stress.

In addition, the vestibular after-sensation of rolling is another factor which should not be ignored. Typically, in a roll of two rad $\cdot$ s^{-1} (or 100°), the sensation of roll dies away in 10 to 15 s (6). Recent surveys of civilian aerobatics pilots revealed that 12.7% reported persistent vertigo after aerobatics flight associated with $-G_z$ exposure (26).

It is understood that ground-based devices are not capable of duplicating the precise maneuvers that could be performed by an aircraft. However, to our knowledge, the primary effects of roll vs. pitch on cardiovascular response in the human are not known. This study was designed to investigate these effects under orthostatic stress, by exposing subjects to head-up (HU) to head-down (HD) and back to HU maneuvers using a tilt table. Based on results of animal studies (28), we hypothesized that in humans, the cardiovascular response to pitch and roll rotation might be different.

METHODS

Subjects

Twelve healthy volunteers (1 female and 11 males) ranging from 28 to 47 yr of age (mean weight = 73 ± 7 kg, mean height = 176 ± 7.7 cm) participated in this study. They had no known history of cardiovascular, ophthalmologic, oculomotor, or vestibular disorders. Before the experiment, their range of BP was 100/65 to 128/80 and the range of heart rate was 60–75 bpm. All subjects obtained medical approval from a physician to participate. The study was approved by the DCIEM Human Ethics Committee. Informed consent was obtained from each of the subjects. Prior to the experiment, subjects were instructed to abstain from alcohol, over the counter and prescribed medications for at least 24 h. All the subjects were familiarized with the experimental procedure and apparatus on separate days by exposing them to the roll and pitch rotation before data collection. Further, all of the subjects had previous experience with the tilt table. The experiments were performed in mornings in a quiet room with the lights off to limit visual cues of orientation.

Apparatus

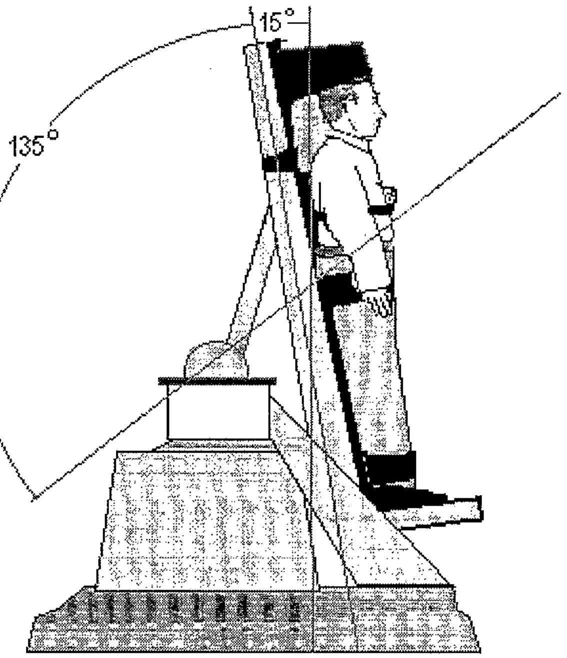
The motion stimulus is a man-rated electronic tilt table that can accommodate either a seated or standing subject with a range of 15° (HU) to 135° (HD) tilt. Zero degrees is considered to be the gravitational vertical. The angular velocity of the tilt table can be set from 5° $\cdot$ s^{-1} to a maximum of 45° $\cdot$ s^{-1} in 5° increments. The start angle, stop angle, and speed of rotation are pre-set prior to each run. A transducer attached to the tilt table registers the tilt angle, which was time-stamped for correlation with recorded physiological parameters. Axis of rotation for pitch and roll passed approximately through the mid body axis (at the umbilicus level) of the subject.

Procedures

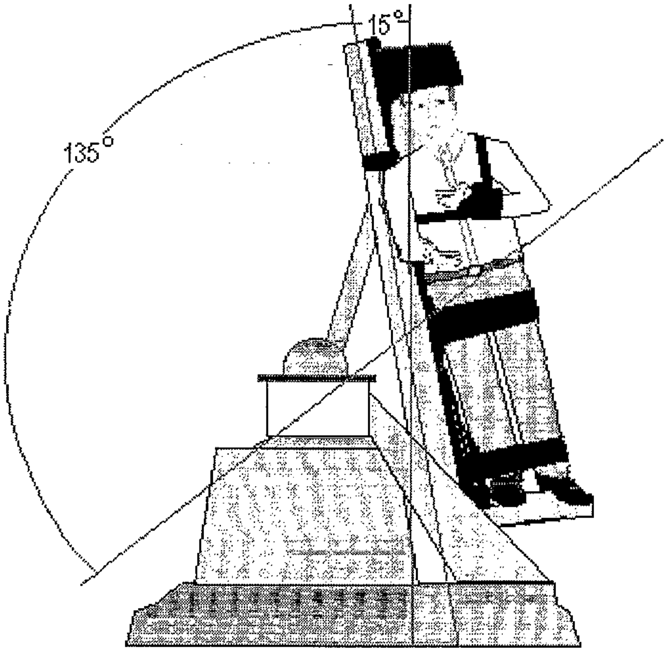
A repeated measure, factorial design was employed with two factors: 1) orientation (pitch and roll); and 2) HD dwell time (7, 15, and 30 s). Each subject participated in three experimental sessions, that were held at least one day apart, one for each of the HD dwell times. The order of the HD dwell times was randomized across the subjects. Within each session, the subject was exposed to the two trials of different orientation; the order of the two orientations was also randomized. Each trial within each session was separated with a rest period of at least 10 min or until the subjects' BP stabilized and returned to their initial baseline. In the pitch orientation, the subject faced forward with their back against the tilt table and in the roll orientation, the subject faced sideways with the shoulder against the tilt table as shown in **Fig. 1**.

During each trial, the subject was positioned onto the tilt table with a loose harness and his feet secured to the stand plate. The subjects were instructed to close their eyes, relax, remain passive, and not to tense their muscles, especially during the rotation phase. Baseline measurements of arterial BP (BP) and electrocardiogram (ECG) were recorded for at least 2 min at 15° relative to gravitational vertical. The subject was tilted to 135° head-down (HD) and remained in the HD position according to the assigned dwell time for that particular trial. The subject was then returned to the HU position. The upright posture is one in which the utricular maculae are nearly horizontal. The direction of body tilt specifies the direction of the linear-acceleration vector acting on the statoconical membrane, and the sine of the angle of tilt specifies the magnitude of the shear force. Therefore, at 135° HD tilt, the magnitude of the linear acceleration vector acting on the statoconical membrane of the utricle is sine 135° or −0.7071 G. The velocity of the tilt table was set at 45° $\cdot$ s^{-1}. BP and ECG were monitored throughout the HU-HD-HU maneuver, and after the tilt, until the BP returned to the baseline level.

A fingertip BP monitor (Finapres, Ohmeda Inc., Englewood, CO) was used for continuous measurement of systolic and diastolic BP with direct analog output to a Gould RS3600 polygraph (Valley View, OH). The servo self adjust function was disabled to ensure that the Finapres did not reset during critical measurement. The monitoring cuff was placed on the subject's middle finger of the left hand and secured on the third intercostal space at the sternal-costal junction with a modified work glove. ECG was continuously monitored using a Tectronics 408 electrocardiograph (Beverton, OR), sampling at 100 Hz. Three bipolar limb leads of VR (right arm), VL (left arm), and VF (left foot) were em-



Pitch Rotation



Roll Rotation

Fig. 1. During pitch rotation the subject faces forward with his back against the tilt table. During roll rotation the subject faces sideways with his shoulder against the tilt table.

ployed in this procedure. A reduction or an increase of mean successive difference of the RR interval determined heart rate. Consequently, the ECG, and BP signals were continuously monitored and displayed during the 120° HU-HD-HU trapezoidal movements of the body. These physiological measures were also used to identify impending pre-syncope during orthostatic stress.

During head-up, in the roll orientation, as mentioned previously, the start angle of the tilt table is 15° to the gravitational vertical; therefore the position of the Finapres is below that of the heart level. Similarly, when the subject is HD during roll, the position of the Finapres is also below that of the heart level. By applying appropriate trigonometry and hydrostatics we determined a correction factor of 1.67 mmHg based on the greatest measurement of the position of the Finapres relative to the heart among all subjects. Considering the accuracy of the Ohmeda Finapres is 2 mmHg (according to the manufacturer), the correction factor is insignificant and therefore not included in our final analysis when roll vs. pitch BP changes were compared.

We conducted a number of preliminary studies to select an appropriate experimental profile such as the rate of rotation and maximum angular displacement from upright. Subjects were tilted to 90° HD with HD dwell time set at 7s. These subjects also participated in HD tilts to 135° where the angular velocity of the tilt table was set at $5° \cdot s^{-1}$, $15° \cdot s^{-1}$, $30° \cdot s^{-1}$; again, the HD dwell time was set at 7s. Since the cardiovascular system is anatomically asymmetrical, during roll orientation, a comparison was also made between lying with the right and left shoulder down on the tilt table. With the left shoulder down, arterial BP was monitored with the Finapres on the subject's right hand. One subject was exposed to the HU-HD-HU maneuver in the seated position.

Data Analysis

From the arterial BP data, we determined the changes in BP under all the conditions described above using a modified Schellong method (22). The Schellong test defined orthostatic hypotension (OH) as the systolic BP just before head-up tilt minus that systolic BP in the head-up position equal to or more than 21 mmHg. That is, a systolic BP decrease of more than 21 mmHg is the criteria for OH. The corresponding heart rate was determined by measuring the R-R interval. We employed an average of 6 BP and 6 HR measurements immediately before the start of the tilt and immediately after the completion of the tilt to evaluate against the criteria for OH. The initial increase in BP induced by HU-HD tilt and the initial decrease in BP induced by HD-HU tilt were compared between the pitch and roll orientation of the standing subject. The rate of change of BP was obtained by dividing the magnitude of BP increases or decreases by the duration of the transition, which is 2.6 s. Onset latency to compensatory response is defined as the time from when the subject is brought back to the head-up position to the time when compensation begins (as observed from the rise in BP). Analysis of variance followed by Contrast Analysis (planned comparison) was performed using Statistica by StatSoft. P-values for factors with more than two levels were

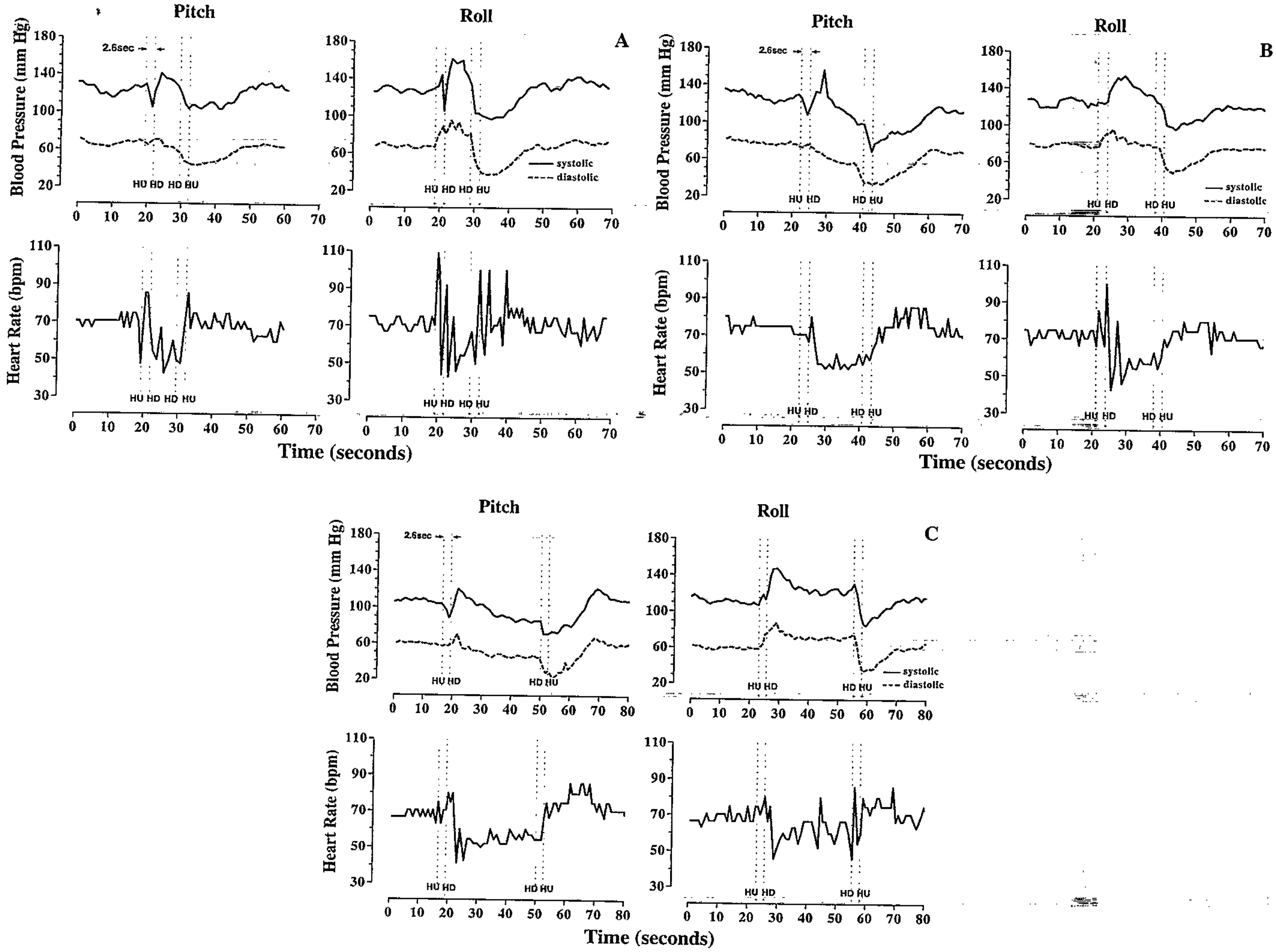


Fig. 2. A typical response of the BP in mmHg (top trace), HR in bpm (beats per minute) recorded at heart level (bottom trace) during pitch rotation and during roll rotation under different head down dwell time. Fig. 2A, 2B, and 2C for 7, 15 and 30s head down, respectively. HD = Head Down, HU = Head Up, areas enclosed by the vertical dotted line represent the transition period from HU to HD and from HD to HU.

adjusted using the Greenhouse-Geisser's epsilon correction factor.

RESULTS

All subjects reported a feeling of fullness in the head during head-down, but none complained of discomfort. Three of the subjects described a sensation of dizziness, light-headedness during roll rotations. We could not analyze the female subject's data separately for statistically meaningful results. Since her responses followed the same temporal and amplitude pattern as the male subjects, her data was included in the final analysis. Because HR data for one subject was lost, only 11 of the 12 subjects' HR data were analyzed. A considerable difference in BP, HR, and onset latency to compensatory response between pitch and roll orientation during the 120° HU-HD-HU trapezoidal movement of the head was observed. When the subjects were tilted upright from 90° HD in the preliminary studies, the difference in autonomic responses between the pitch and roll orientation was also apparent. The effect was also demonstrated in the seated subject during preliminary trials. Because we are interested in exposing the subjects to the maximum possible orthostatic stress under less than +1 Gz, we report only the results from the 120° HU-HD-HU maneuver. A typical response of the BP and HR from an individual male subject using 7-, 15-, and 30-s HD dwell time is shown in **Fig. 2A**, **2B**, and **2C**, respectively.

Blood Pressure (BP)

In all conditions, BP increases sharply following HU to HD, after an average latency of 2 ± 0.14s (in both pitch and roll orientations). The mean difference (±SEM) of systolic BP increase from HU to HD during the pitch and roll maneuvers was 12.89 ± 1.95 mmHg and 22.03 ± 2.21, respectively. Since it took 2.6 s to traverse 120°, the rate of BP increase from HU to HD was 4.95 mmHg $\cdot$ s^{-1} in the pitch orientation and 8.47 mmHg $\cdot$ s^{-1} in the roll orientation. A *t*-test for dependent samples was performed on the HU to HD BP data

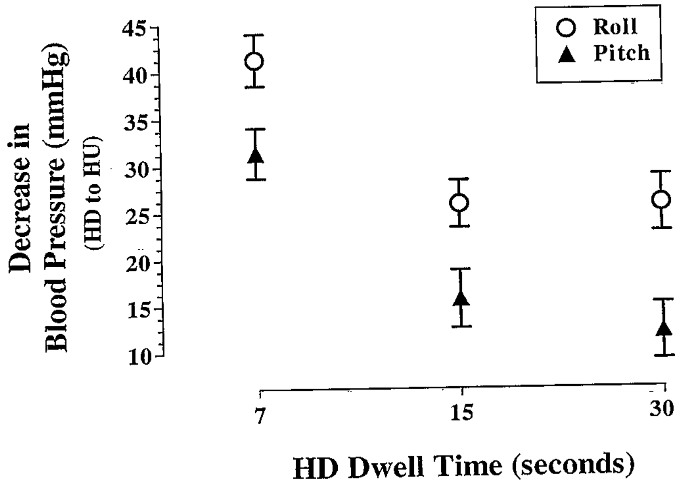


Fig. 3. Decrease in systolic BP as a function of HD dwell time and orientation during the HD to HU phase of the tilt maneuver (error bars = SEM).

under the pitch and roll orientation. The results revealed that the mean increase of BP was significantly greater during the roll than during the pitch maneuver ($t = 3.97$, df = 35, $p \leq 0.001$). After the initial rise at the beginning of the plateau phase of the HD stimulus, BP gradually diminished over the next 12.4 ± 0.55 s, for both 15-s and 30-s HD dwell time, at which time BP began to rise for the remainder of the HD period. For the 7-s dwell time, BP continued to decrease throughout the HD period.

BP immediately decreased following HD to HU in both the pitch and roll orientation. Visual inspection of the magnitude of hypotension between pitch and roll is different under all three HD dwell times. A 3 (HD dwell time) × 2 (orientation) repeated measures ANOVA was performed on the HD-HU systolic BP data. The main effects of HD dwell time and orientation were significant (F (2,22) = 31.518, $p \leq 0.001$, $\epsilon = 0.864$; and F (1,11) = 52.826, $p \leq 0.001$, $\epsilon = 0.864$, respectively). However, the interaction between HD dwell time and orientation was not significant. The main effects for dwell time and orientation are illustrated in Fig. 3. Inspection of Fig. 3 reveals three features: 1) The mean decrease of systolic BP during HD-HU is significantly greater during roll than during pitch maneuver at all HD dwell times tested. Test of significance revealed that $p < 0.01$ for 7 s, $p < 0.001$ for 15 s, $p < 0.001$ for 30 s. 2) The magnitude of OH as defined previously was observed during roll rotation at 41.2, 25.7, 25.8 mmHg for 7-, 15- and 30-s HD dwell times, respectively. However, during pitch rotation, OH was observed only during 7s HD dwell time at 31.3 mmHg. 3) Within the roll and pitch maneuvers, the decrease in BP is significantly higher at the 7-s than 15- and 30-s HD dwell time ($p < 0.001$). There is no difference in BP decrease between the 15- and 30-s HD dwell time. These observations were confirmed by Contrast Analysis. For HD dwell times of 7, 15, and 30 s, BP gradually re-gained baseline and reached a plateau in the HU position in 10.4 ± 0.91 s, 10.1 ± 0.73s, and 7.6 ± 0.73 s, respectively, as measured from the onset of compensation. More importantly, the rate of BP decrease in the roll orientation is significantly higher than the rate of BP decrease in the pitch orientation. The rate of change of BP under all conditions is summarized in **Table I.**

TABLE I. RATE OF BP CHANGES (INCREASE: HU-HD, DECREASE: HD-HU) IN THE PITCH AND ROLL ORIENTATION UNDER ALL CONDITIONS.

Dwell Time	Rate of BP Increase: HU-HD*	Rate of BP Decrease: HD-HU 7 s†	Rate of BP Decrease: HD-HU 15 s§	Rate of BP Decrease: HD-HU 30 s**
Pitch	4.95 ± 0.75	12.05 ± 1.03	6.03 ± 1.2	4.65 ± 1.15
Roll	8.47 ± 0.85	15.87 ± 1.06	9.94 ± 0.97	9.90 ± 1.16

* $p < 0.001$.
† $p < 0.016$.
§ $p < 0.001$.
** $p < 0.001$.

Heart Rate (HR)

A *t*-test for dependent samples was performed on the HU-HD roll and pitch HR data. The mean decrease of HR (±SEM) from HU to HD during the pitch and roll maneuvers was 29.7 ± 2.59 and 22.67 ± 2.32 bpm, respectively. The results revealed that the decrease in HR was significantly greater during the pitch than during the roll maneuver ($t = 4.845$, df = 32, $p \leq 0.001$).

A 3 (dwell time) × 2 (orientation) repeated measures ANOVA was performed on the HD-HU heart rate data. The main effect for orientation was significant (F (1,10) = 8.817, $p = 0.014$) and the interaction between dwell time and orientation was also significant (F (2,20) = 12.563, $p = 0.031$, $\epsilon = 0.961$). The interaction between HD dwell time and orientation of the HR data is illustrated in **Fig. 4.** Inspection of Fig. 4 reveals that the HD-HU increases in HR at the 7-s HD dwell time is significantly greater during pitch than during the roll maneuver ($p < 0.01$). These observations were con-

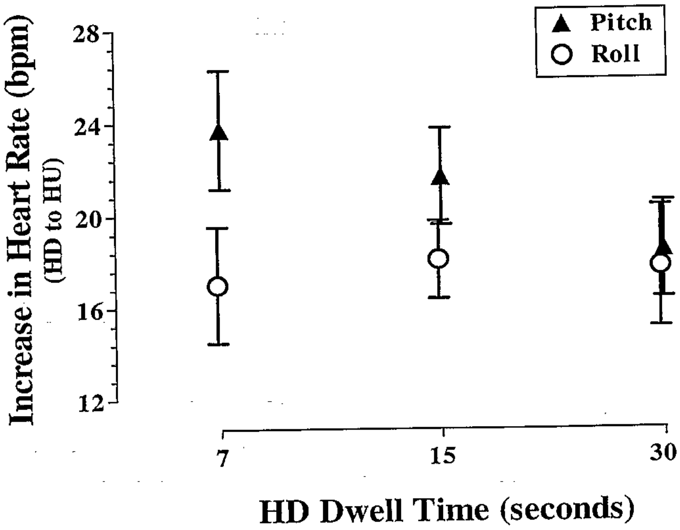


Fig. 4. Increase in HR as a function of dwell time and orientation during the HD to HU phase of the tilt maneuver (error bars = SEM).

firmed by Contrast Analysis. However, there were no significant differences in the changes in HR between the pitch and roll orientation at the 15- or 30-s HD dwell times.

Onset Latency to Compensatory Response from HD to HU

A 3 (HD dwell time) × 2 (orientation) repeated measure ANOVA was performed on the onset latency to compensation response data. The main effects for HD dwell time were significant ($F(2,20) = 9.431$, $p < 0.001$, $\epsilon = 0.951$), but no other effects were. The onset latency mean values (±SEM) during HU following the 7-s, 15-s and 30-s dwell times, are 5.3 ± 0.88, 2.3 ± 0.72, and 2.6 ± 0.83 s, respectively. Contrast analysis revealed that the latency to compensation after a 7-s HD dwell time is significantly longer than at either 15 ($p < 0.001$) or 30 s ($p < 0.001$).

Preliminary investigation of HD rotation to 90° also demonstrated that the induced OH and increased HR were significantly higher in the roll than the pitch orientation. The magnitude of the decrease during 90° HD conformed to the Schellong criteria. However, the magnitude of induced OH (HD-HU systolic pressure) was substantially greater during 135° HD (mean HD-HU = 41.2 mmHg) than 90° HD (mean HD-HU = 32 mmHg). In the roll orientation, there was no difference in BP or HR, whether the subject was placed with the left side or right side down. In other words, rolling to the left or to the right makes no difference to the parameters measured. The difference in the rate and magnitude of BP decrease between pitch and roll was also evident from a seated subject. Finally, when the tilt table velocity was set at $5° \cdot s^{-1}$, no orthostatic hypotension was induced under all conditions. When the velocity of the tilt table was set at $15° \cdot s^{-1}$ and $30° \cdot s^{-1}$, the difference in the rate of BP decrease as induced by HD-HU rotation between the pitch and roll axes were progressively higher, but less than the results obtained with $45° \cdot s^{-1}$ rotation. The decrease in BP and hence the rate of BP decrease was consistently higher in the roll than in the pitch rotation in all three velocities.

DISCUSSION

The cardiovascular system is profoundly susceptible to gravitational accelerations; simple changes in posture may initiate a decrease in venous return to the heart and pose a dramatic challenge to blood circulation. To prevent OH, rapid corrections need to be made in the circulatory system in order for blood to flow from the distal extremities back to the heart. The consequence of OH is decreased perfusion of the brain, which could result in syncope if the condition is not corrected rapidly.

A number of mechanisms exist to resist postural related OH. An increase in sympathetic outflow to selected arterial beds reduces blood flow to unnecessary regions raising vascular resistance and, hence, BP. Baroreceptors located in the carotid sinus and aortic arch respond to changes in BP instantly, however, the full expression of the reflexes is delayed. This leads reflexively to an inhibition of vagal (parasympathetic) tone and to a stimulation of the sympathetic nervous system. Both mechanisms increase the strength and rate of cardiac contraction and increase the resistance to blood flow by provoking a generalized vasoconstriction. On the other hand, when exposed to $+G_z$ acceleration, the maximum heart rate from the compensatory reflex was achieved some 15 s after the onset of acceleration (1). Therefore, a feedback mechanism helps to prevent a decrease in BP following a change in posture, but its limited temporal response is the cause of the early phase of OH.

When a person is rotated 120° from HU to HD, arterial pressure increases and heart rate decreases, as measured at the heart level in this study. Our data indicated that the increase in BP from HU to HD is less pronounced during the pitch rotation than the roll rotation. This is consistent with the findings that the decrease in HR from HU to HD is less pronounced during the pitch rotation than the roll rotation. The latency to increase in BP from HU to HD is almost immediate, about 2 s in both orientations. More importantly, the magnitude and rate of decrease in BP induced by a 120° HD to HU maneuver is significantly greater during roll rotation. The HR increase post rotation is significantly higher in the pitch than the roll rotation. Our observations suggest that pitch rotation is the preferred direction of head rotation as indicated by the seemingly more efficient compensation for orthostatic stress than roll rotation (magnitude of induced OH is lesser in pitch rotation).

We are more accustomed to pitching than rolling movements. There is a predominance of forward locomotion and downward motion detection in humans (17). The movements normally encountered in roll are of briefer duration than those for pitch or yaw. Psychophysically, for roll tilt angles greater than 30°, humans underestimate the amount of tilt known as the Aubert effect (29). In addition, for roll tilt angles greater than 30°, the variability in making these estimates also increases significantly. It has also been shown that the perception of pitch is stronger than roll under stimulus velocities of 30, 45, and $60° \cdot s^{-1}$ (7). Therefore, it is assumed that those pitch movements maintain a high level of sympathetic nerve activity to produce the maximal possible compensation in BP responses to stimuli that excite the sympathetic nervous system.

The onset latency of the BP increases from HU-HD, regardless of the pitch and roll orientation, was 2 s. Although a direct comparison could not be made, it is of interest to note that in cats with extensive denervation to eliminate non-labyrinthine inputs, the onset latency of the increase in BP during head up tilt was 1.4 s (27). This short latency of 1.4 s was similar to that of pressor responses elicited by stimulation of vasomotor neurons in the rostral ventrolateral medulla (24). This onset latency from the beginning of the stimulus plateau, HD dwell time was on the average 1.4 s. In our study it was also observed that during the HD plateau phase, BP gradually diminished for 12.4 ± 0.55 s, for both 15-s and 30-s HD dwell times, indicating that baroreceptors have already begun the compensatory action (Fig. 2B and 2C). Therefore, the lesser change in BP and HR differences between the roll and pitch orientation at the longer HD dwell times is to be expected.

The current study has been limited by the maximum $+G_z$ level ($+0.96\ G_z$ as the upright position is 15° to vertical) one could attain using the tilt table. Since the rate and the magnitude of BP decrease from HD to HU is significantly greater during roll rotation, it is likely that the absolute BP reached at a higher $+G_z$ level (for example at $+3G_z$) could be even lower in the roll orientation. Further study is required to investigate this possibility and how it might affect G-tolerance during subsequent exposure to hypergravity. The second limitation is that the maximum rate of rotation of the tilt table is $45° \cdot s^{-1}$. Our preliminary studies indicated that at $5° \cdot s^{-1}$ there was no difference in cardiovascular response between roll and pitch. However, difference in cardiovascular responses between roll and pitch in six subjects were progressively more apparent at $15° \cdot s^{-1}$ and $30° \cdot s^{-1}$. However, it did not reach the same level of significance as in $45° \cdot s^{-1}$. It suggested that the rate of rotation could be a determining factor in inducing OH. The final limitation is that roll rotation on the ground stimulates the semicircular canals as well as a changing gravity vector detected by the otoliths. The exact mechanism of the roll rate in inducing OH remains to be investigated.

Is it reasonable to postulate that there exists a vestibulo-cardiovascular reflex? As early as the 1940s, it was shown that activation of vestibular afferents by galvanic or caloric stimulation produced changes in BP in cats, dogs and guinea pigs (23). As mentioned briefly in the introduction, recent studies in decerebrate cats (28) indicated that changes in sympathetic outflow are elicited by pitch rotation (about the interaural axis) but not by roll rotation (about the naso-occipital axis). These cats also underwent upper cervical root transection, cerebellectomy, baroreceptor denervation, and vagotomy. Therefore, sensory inputs elicited by head rotations came from the vestibular system. In addition, the gain of the vestibulo-sympathetic response to sinusoidal pitch rotation is constant across stimulus frequencies and in phase with the stimulus position. Since these response characteristics are similar to those of otolith afferents, it is likely that the otolith organs are predominantly responsible for producing the vestibulo-sympathetic response. The most direct evidence of vestibular influence on orthostatic adjustment came from experiments (12) showing that bilateral transection of the vestibular nerves in paralyzed, chloralose-anesthetized cats significantly impaired compensation for the hypotension produced by 30° or 60° head-up body tilt. In addition, static nose-up vestibular stimulation, but not ear-down stimulation produces large (20 mmHg) increases in BP. The increase in BP to nose-up tilt was abolished following the transection of the VIIIth cranial nerve (27), indicating that the changes were not the result of fluid shifts or other indirect mechanism. Recent neuroanatomical and neurophysiological studies also demonstrated direct connections between the vestibular nuclei, the locus coeruleus and the brainstem pathways that are involved in control of the sympathetic nervous system. Balaban and Porter (2) demonstrated that removal of vestibular inputs to the brainstem compromises the ability to adjust BP during unexpected changes in posture.

Since the vestibular system provides inputs signaling changes in body position, based on the above evidence, it is likely that vestibular signals provide for feed-forward adjustments of BP during unexpected postural changes, such that the compensation for OH can begin before BP drops significantly. Data from our preliminary studies suggested that the induced OH is greater during HD 135° ($-0.7\ G_z$) to HU than during supine ($-1\ G_z$) to HU. This observation appeared to be confounding to the G transition effect (reduced G-tolerance induced by post-hypogravity acceleration). Vestibular associated reflexes are known to be highly "plastic," we are accustomed to the expected and frequently practiced maneuver of rising from supine to upright every morning, regardless of whether we rise up in the pitch or roll plane. Therefore, it is reasonable that the induced OH observed is lessened in the later case.

The evidence of vestibular autonomic interactions in humans is not as clear. Various methods of vestibular stimulation, such as caloric (20), optokinetic (7,14), Coriolis (25), or pseudo-Coriolis (15) stimulation, were reported to cause changes in BP, heart rate, and forearm blood flow. However, the autonomic responses elicited by these different stimuli are not consistent. These stimuli also inadvertently cause nausea, vomiting, and discomfort that could lead to psychologically induced cardiovascular effects. The autonomic consequences of vestibular stimulation may differ depending on the stimulation used and the vestibular pathway involved. Moreover, it is difficult to verify that the effects on the sympathetic nervous system are not due to the activation of the visceral receptors and baroreceptors.

In this study, rotation of the subjects about the roll and pitch axes allows one to study the effects of selective activation of vestibular receptors to elicit autonomic responses to compensate for orthostatic stress. The directions of excitatory and inhibitory accelerations for all afferents innervating the semicircular canals are specific to the morphological polarization of the hair cells (13). These directional rules are specific to the plane of rotation. Similarly, based on their polarization vectors, functional implications of directional selectivity also exist in the utricular and saccular otoliths. For example, pitching forward excites the superior canals and inhibits the posterior canals while pitching backward excites the posterior canals and inhibits the superior canals of both ears. On the other hand, rolling to the left excites the superior and posterior canals of the left ear and inhibits the superior and posterior canals of the right ear. Rolling to the right excites the superior and posterior canals of the right ear and inhibits the superior and posterior canals of the left ear (11). In the otolith organs, the effective stimulus force acts tangentially on the statoconial membrane (12). The great majority of utricular vectors lay near a horizontal plane, whereas most of the saccular vectors lay near a sagittal plane. The utricular macula, given the broad distribution of its polarization vectors, will signal to both lateral and fore-aft tilt (and linear acceleration). The saccular macula is particularly sensitive to dorsoventral acceleration and is ideally suited for pitching movements when the head is supine. Therefore, it appears that pitching involves both the stimulation of the utricular and saccular otoliths but rolling stimulates only the utricular otoliths.

It is possible that vestibular input plays a role in the upright posture, perhaps indicating subtle changes in baroreflex function, which may be necessary for normal orthostatic cardiovascular tolerance. In animal studies, the different BP pressor response between the pitch and roll orientation suggest that the BP responses were due to the activation of vestibular receptors and were not the result of fluid shifts or other indirect mechanisms (27). Direct evidence indicating that the vestibular system activates compensation to orthostatic hypotension awaits further studies testing the autonomic response of labyrinthine defective subjects exposed to orthostatic stress following a sudden postural change.

Implication of Current Findings

The current study indicated that simulating a point-roll maneuver on the ground, while transitioning from $-0.7\ G_z$ to $+1\ G_z$, resulted in a significant rate of decrease in BP which could affect subsequent G-tolerance when exposed to increased acceleration. In the aircraft, the centripetal acceleration experienced at the pilot's head level during a point roll depends on the rate and radius of rotation (distance from the head to the center of rotation). For example, rolling at $90° \cdot s^{-1}$ in the F18 at a radius of 0.8 m, the centripetal acceleration experienced is $-0.2\ G_z\ [(90° \cdot s^{-1} / 57.3° \cdot rad)^2 \times 0.8\ m / 9.8\ m \cdot s^{-2}]$. During negative barrel roll maneuver in aerobatics flight, the radius of rotation will be greater; the negative G_z experienced by the pilot will be much higher than the $-0.7\ G_z$ that was simulated in this study. As mentioned previously, the current study is limited by the maximum rate of rotation of the tilt table at $45° \cdot s^{-1}$. In some of the current and future more agile aircraft, the rate of roll rotation could be much higher. Our results suggest that the rolling maneuver could exacerbate the reduced G-tolerance caused by transition from hypo- to hypergravity. Whether this effect can be duplicated under operational environment or the effect could be attenuated by habituation remain to be investigated. We also verified that the appearance of deleterious hypotensive effects is not limited to extreme negative G_z exposure. In addition, the semicircular canals provide accurate information about angular movements of the head, as in a roll, during natural environment. The lack of sensory input about rolling at a constant rate, as well as the erroneous signal of rolling in the opposite direction on recovery from a constant rate of roll is well known. How the above factors affect subsequent exposure to hypergravity remains to be investigated.

In view of current evidence, use of the term "Push-Pull" to describe the transition from hypogravity to hypergravity is insufficient if it involves a point roll maneuver. With few exceptions, the flight maneuver under operational flying environment is seldom one-dimensional. In addition, the G-loading history prior to this post-hypogravity acceleration effect (reduced G tolerance) will undoubtedly affect its outcome (5). Scientifically, the terminology of "Push-Pull Effect" can be confusing. "Push-Pull" effect or mechanism is originally used to describe the bi-directional response exhibited by the canal afferents of the vestibular system (13). This is based on neurophysiological findings that any head movement exciting a canal on one side of the head will inhibit the synergic canal on the other side. It is the difference in the responses of synergic canals that is interpreted by the brain as an angular head motion. Hence the vestibular system is said to function as a push-pull balance system.

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