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ACUTE EFFECT OF 30°, 60° AND 80° HEAD-DOWN TILT ON BLOOD PRESSURE IN YOUNG HEALTHY HUMAN SUBJECTS

P. VIJAYALAKSHMI* AND MADANMOHAN

Department of Physiology,
Jawaharlal Institute of Postgraduate Medical Education and Research (JIPMER),
Pondicherry – 605 006

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Abstract : There are very few reports on the acute cardiovascular responses to head-down tilt of more than 30° on blood pressure (BP) and heart rate (HR) in human subjects. The aim of the present study was to determine the BP and HR response to five minutes of 30°, 60° and 80° head-down tilt in healthy human subjects. The study was conducted on 20 healthy male volunteers aged 18–20 years. In this study, 30° head-down tilt produced no significant change in BP and HR. With 60° and 80° head-down tilt, there was a significant graded increase in diastolic pressure and a decrease in pulse pressure ($P<0.01$ for both). This pressor response is most likely due to raised intracranial tension and/or hypoxemia in this condition. The decrease in pulse pressure is possibly due to excessive cardiopulmonary blood volume resulting in a decreased stroke volume. Our study confirms earlier findings that acute 30° head-down tilt produces no significant changes in BP and HR. However, the mechanisms leading to increases in BP that occurred with 60° and 80° acute head-down tilt need to be investigated.

Key words : cardiopulmonary receptors central venous pressure cushing's reflex diastolic pressure pulse pressure

INTRODUCTION

Effects of prolonged 6° head-down bed rest, a laboratory model of microgravity, have been well studied (1). Acute head-down tilt (HDT) is an established experimental model for cardiopulmonary volume loading (2). Prolonged 20 degrees HDT and tail suspension in rats have been used as models of cardiovascular deconditioning and greater degrees of HDT model negative G (3). On the other hand, reduction of central venous pressure with infusions of atrial natriuretic factor has been demonstrated to produce reflex activation of the sympathetic nervous system (4). However, there are very few reports on the acute effect of various degrees of HDT (tilt angle > 30°) on blood pressure (BP) and heart rate (HR) in human subjects. *Thus, in this study, we aimed to examine the acute effects of 30°, 60° and 80° HDT on BP and HR in healthy human subjects.*

SUBJECT AND METHODS

We studied 20 healthy young male

*Corresponding Author :

subjects aged 18-20 yr. The Institute Research Council, JIPMER, approved the study protocol. All subjects gave informed consent. Lead II ECG was recorded on a polygraph (Grass Instruments Inc, USA). BP was measured with a calibrated aneroid sphygmomanometer (Enma, Japan). All BP measurements were made by a single observer. A manually operated tilt table with footplate support was used. Subjects were familiarised with the HDT procedure before actual recordings were taken. Subjects who were unable to tolerate the procedure were excluded from the study. Tests were done 2 h after a light breakfast. Subjects refrained from smoking on the day of the tests. Subjects were not taking any medication at the time of testing.

Head-down tilt procedure : With the subject's head resting on the footboard, straps were applied at the level of knees, waist, and shoulders. Additional straps were applied to the ankles over the soft pads. Care was taken to see that there was no discomfort to the subject or interference with the subjects' breathing.

The sequence in which recording was done is as follows :

- Baseline : 10 min supine rest on the tilt table.
- 30° HDT for 5 minutes
- 5 minutes rest in the supine position
- 60° HDT for 5 minutes
- 5 minutes rest in the supine position
- 80° HDT for 5 minutes
- Supine position

Thus, the subjects were kept in each degree of tilt for 5 min. Between each degree of tilt, the subject was tilted back to the horizontal position and allowed to rest for 5 min. The systolic pressure (SP), diastolic pressure (DP), pulse pressure (PP), mean pressure (MP) and HR were measured at each angle of tilt immediately after and at 1, 2, 3, 4 and 5 min after tilt. Pulse pressure (PP) was calculated as the difference between SP and DP and rate-pressure product (RPP) was calculated as $SP \times HR \times 10^{-2}$ as in reference 5.

Statistical analysis : All data given as mean ± SD. Changes during tilt were compared with baseline values using Student's paired t-test. A two-tailed P value <0.05 was considered significant.

RESULTS

Results are given in Tables I–III. With 30° HDT, there was no significant change in SP, DP, MP, HR and RPP throughout 5 minutes compared to pre-tilt supine values ($P>0.05$ for all). Changes in SP, HR and RPP were insignificant for all angles of tilt ($P>0.1$). *The increase in DP was directly proportional to angle of tilt and statistically significant for 60° & 80° ($P<0.01$ for both), whereas it remained unchanged at 30° HDT.* A decrease in PP occurred at all angles of tilt, the decrease was directly proportional to angle of tilt and statistically significant for 60° & 80° ($P<0.01$) but not 30°.

TABLE I: Effect of 30° head-down tilt on systolic pressure (SP), diastolic pressure (DP), pulse pressure (PP), mean pressure (MP), heart rate (HR) and rate pressure product (RPP).

Parameters	*Supine*	*Imm*	*1*	*2*	*3*	*4*	*5*
SP (mm Hg)	104±8.9	102±13.4	104±8.9	104±8.9	105±8.9	105±8.9	105±8.9
DP (mm Hg)	67±9.8	69±10.7	71±8.9	71±9.4	72±8.9	72±8.9	72±8.5
PP (mm Hg)	37±8.5	33±10.7	33.0±11.2	33±10.3	34±10.7	33±9.8	33±10.7
MP (mm Hg)	79±8.5	80±9.8	82±7.6	82±8.5	83±7.6	83±8.0	83±7.6
HR (beats/min)	74±15.2	71±9.4	68±8.0	69±7.6	68±8.0	68±8.0	67±8.0
RPP (units)	76±16.5	73±14.3	72±11.6	73±11.6	73±11.2	73±11.2	72±12.5

Imm: immediately after tilt; 1, 2, 3, 4, 5: minutes after tilt. Data are expressed as mean±SD for 20 subjects. P>0.05 for all comparisons between tilt and supine values.

TABLE II: Effect of 60° head-down tilt on systolic pressure (SP), diastolic pressure (DP), pulse pressure (PP), mean pressure (MP), heart rate (HR) and rate pressure product (RPP).

Parameters	*Supine*	*Imm*	*1*	*2*	*3*	*4*	*5*
SP (mm Hg)	104±8.9	104±13.4	105±12.5	106±12.5	106±11.6	107±11.2	106±11.6
DP (mm Hg)	67±9.8	75±8.9*	75±8.5**	76±7.6**	77±8.9**	77±7.2***	77±8.9**
PP (mm Hg)	37±8.5	29±12.1*	29±10.7*	30±11.2*	29±8.9**	30±8.9*	29±11.2*
MP (mm Hg)	79±8.5	85±8.5	86±9.8*	86±8.5**	86±8.9**	87±8.0**	87±8.9**
HR (beats/min)	74±15.2	66±8.0	68±7.6	69±9.8	69±8.9	69±8.0	70±9.8
RPP (units)	76±16.5	70±13.4	72±13.4	75±14.3	73±13.0	74±13.0	76±12.5

Imm: immediately after tilt; 1, 2, 3, 4, 5: minutes after tilt. Data are expressed as mean±SD for 20 subjects. *P>0.05, **P<0.01, ***P<0.001 compared to supine values.

TABLE III: Effect of 80° head-down tilt on systolic pressure (SP), diastolic pressure (DP), pulse pressure (PP), mean pressure (MP), heart rate (HR) and rate pressure product (RPP).

Parameters	*Supine*	*Imm*	*1*	*2*	*3*	*4*	*5*
SP (mm Hg)	104±8.5	104±12.5	108±14.3	108±12.5	107±12.1	108±13.0	109±12.5
DP (mm Hg)	67±9.8	77±8.0***	77±8.0***	79±8.0***	80±7.9***	80±8.9***	80±9.4***
PP (mm Hg)	37±8.5	26±11.2**	31±11.6	30±10.3*	27±10.3**	28±10.7**	31±10.3*
MP (mm Hg)	79±8.5	86±8.5**	88±8.9**	89±8.5***	89±7.6***	89±8.9***	90±9.4***
HR (beats/min)	74±15.2	68±10.7	71±10.3	70±9.4	70±9.8	70±8.5	70±8.9
RPP (units)	77±16.5	71±16.5	77±16.1	76±13.9	75±15.2	75±13.0	78±13.9

Imm: immediately after tilt; 1, 2, 3, 4, 5: minutes after tilt. Data are expressed as mean±SD for 20 subjects. *P>0.05, **P<0.01, ***P<0.001 compared to supine values.

DISCUSSION

Our data reflects the acute effects of 30°, 60°, and 80° of HDT in healthy human subjects. In the present study, results of 30° HDT are similar to those reported earlier (6, 7). There are no significant changes in BP and HR. Goldsmith et al (7) found no changes in HR, mean arterial pressure, plasma norepinephrine and plasma renin activity during 1 hour of sustained 30° HDT accompanied by saline infusions to achieve a significant increases in CVP. However, London et al (8) have reported a decrease in forearm venous tone in controls, although BP, HR and baroreflex sensitivity did not change in their subjects. *An increase in cardiopulmonary blood volume as occurs with 15° and 30° HDT has been shown to be associated with a reflex decrease in muscle sympathetic nerve activity and decrease in forearm vascular resistance but little change in BP and HR(6).*

The decrease in PP during 60° and 80° HDT is most likely due to a decline in stroke volume as a result of acute excessive cardiac loading. Olfert et al (9) assessed ventilatory homogeneity using the phase III slope of the Helium multiple breath washout technique in seven healthy human subjects during acute 60° HDT and reported that an increase in pulmonary water and compression of lung parenchyma by engorged great veins in the thorax may atleast be partly responsible for alterations in ventilatory homogeneity in the HDT position. Thus, it is likely that 60° and 80° HDT (in our study) may also be associated with impairment in gas exchange. The resultant hypoxemia could have activated the sympathetic nervous system. Furthermore, the raised intracranial tension accompanying 60° and 80° HDT reduces blood flow to the vasomotor center and elicits an increase in sympathetic outflow (Cushing's reflex) (10). Whitson et al (11) evaluated the effect of normobaric hypoxia on endocrine responses to sustained 30° HDT. They reported an increase in plasma aldosterone, and renin activity, and found that hypoxia attenuated the release of atrial natriuretic peptide during HDT. However, hypoxemia has a direct depressant effect on myocardial contractility (10). Thus, in our study, the significant increase in DP during 60° and 80° HDT could have occurred as a result of hypoxemia and or raised intracranial tension. Indeed, in a study on the effect of 70° HDT in 12 healthy male subjects, SP increased by 7.5% and DP increased by 15% (8). *However, in our study. HDT was associated with significant increase in DP but not SP. This may have been due to the fact that SP is also influenced by the effects of excess venous return on stroke volume besides the stimulatory effects of sympathetic excitation on HR and myocardial contractility. Furthermore, as stated above, hypoxia could have had a direct depressant effect on myocardial contractility. Thus, changes in SP are difficult to interpret.* In a study of the effects of 1 hour of HDT on rabbits, Wen et al (12) reported that the greatest decrease in protein concentration in plasma occurred during the first five minutes of HDT. They explained that this was due to an increase in cephalad hydrostatic pressure and leakage of plasma proteins due to disruption of tight junctions. They have also suggested that similar phenomena might also occur during this condition in the lungs, and this would lead to hypoxemia and further activation of the sympathetic nervous system. Using a spectral analytic approach, Weise et al (13) reported a decrease in absolute power of the low frequency components of heart rate variability and blood pressure variability

during 100 degrees HDT in healthy subjects suggesting an elevation in sympathetic activity.

Limitations : The BP before each degree of tilt was assumed to be the baseline BP obtained before 30 degrees HDT, this assumption could have been avoided by measuring baseline BP before each degree of tilt. Secondly, the hydration status of the subjects was not specifically controlled, and therefore confounds interpretation of results to a certain extent.

In our study, analysis of arterial blood gases during HDT would have been more informative. In conclusion, while acute 30° HDT does not produce significant changes in BP and HR, 60–80° HDT for 5 min results in a significant increase in DP and decrease in PP but little change in SP, HR and RPP. The decrease in PP is most likely due to a fall in stroke volume due to excessive preloading of the ventricles. The increase in DP could be due to sympathoexcitation due to raised intracranial tension occurring with 60° and 80° HDT. Whether such mechanisms are operative needs to be investigated.

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REFERENCES

1. Hargens AR, Tipton CM, Gollnick PD, Mubarak SJ, Tucker BJ, Akeson WH. Fluid shifts and muscle function in humans during acute simulated weightlessness. *J Appl Physiol* 1983; 54: 1003–1009.
2. Martel E, Champeroux P, Lacolley P, Richard S, Safar M, Cuche JL. Central hypervolemia in the conscious rat: a model of cardiovascular deconditioning. *J Appl Physiol* 1996; 80: 1390–1396.
3. Deklunder G, Lecroart JL, Chammas E, Goullard L, Houdas Y. Intracardiac hemodynamics in man during short periods of head-down and head-up tilt. *Aviat Space Environ Med* 1993; 64: 43–49.
4. Ebert TJ. Reflex activation of sympathetic nervous system by ANF in humans. *Am J Physiol Heart Circ Physiol* 1988; 255: H685–H689.
5. Gobel FL, Nordstrom LA, Nelson RR, Jorgensen CR, Wang Y. The rate-pressure product as an index of myocardial oxygen consumption during exercise in patients with angina pectoris. *Circulation* 1978; 57: 549–556.
6. Nagaya K, Wada F, Nakamitsu S, Sagawa S, Shiraki K. Responses of the circulatory system and muscle sympathetic nerve activity to head-down tilt in humans. *Am J Physiol Regulatory Integ Comp Physiol* 1995; 268: R1289–R1294.
7. Goldsmith SR, Francis GS, Cohn JN. Effect of head-down tilt on basal plasma norepinephrine and renin activity in humans. *J Appl Physiol* 1985; 59: 1068–1071.
8. London GM, Levenson JA, Safar ME, Simon AC, Guerin AP, Payen D. Hemodynamic effects of head-down tilt in normal subjects and sustained hypertensive patients. *Am J Physiol* 1983; 245: 194–202.
9. Olfert IM and Prisk GK. Effect of 60° head-down tilt on peripheral gas mixing in the human lung. *J Appl Physiol* 2004; 97: 827–834.
10. Ganong WF. Review of Medical Physiology. International edition. McGraw Hill: New York, 2002, p. 612: 21st edition.
11. Whitson PA, Cintron NM, Pietrzyk RA, Scotto P, Loeppky JA. Acute effects of head-down tilt and hypoxia on modulators of fluid homeostasis. *J Clin Pharmacol* 1994; 34: 427–433.
12. Wen TS, Randall DC, Zolman JF. Protein accumulation in cerebrospinal fluid during –90° head-down tilt in rabbit. *J Appl Physiol* 1994; 77: 1081–1086.
13. Weise F, London GM, Guerin AP, Pannier BM, Elghozi JL. Effect of head-down tilt on cardiovascular control in healthy subjects: a spectral analytic approach. *Clin Sci* 1995; 88: 87–93.