EFFECT OF HEAD-UP TILT ON CARDIOVASCULAR RESPONSES IN NORMAL YOUNG VOLUNTEERS

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Abstract : Since the cardiovascular effects of tilting are influenced by degree as well as duration of the tilt, we planned to study the time course of blood pressure and heart rate (HR) responses during 30°, 60°, 80° head up tilt (HUT). The study was conducted on 20 volunteers aged 18-20 y who were tilted on a tilting table. Blood pressure was determined by sphygmomanometer and HR was calculated from R-R interval of ECG. 30° HUT produced an insignificant decrease in systolic pressure (SP) and pulse pressure (PP) while diastolic pressure (DP), mean pressure (MP) and ratepressure-product (RPP) registered an insignificant rise. The changes produced by 60° and 80° HUT were more marked than those produced by 30° HUT. While SP and PP decreased significantly, HR and RPP increased significantly. In conclusion, 30° HUT produces insignificant changes while 60° and 80° HUT produce significant changes in SP, PP and RPP.

Key words : head-up tilt pulse pressure heart rate systolic pressure diastolic pressure mean pressure rate-pressure-product

INTRODUCTION

Tilting can be used for assessing the integrity of autonomic cardiovascular regulatory mechanisms in physiological (1-4) as well as in clinical situations (5, 6). Head-up tilt (HUT) results in gravityinduced pooling of blood in lower parts of the body and a consequent decrease in venous return, stroke volume, pulse pressure (PP) and mean pressure (MP). The result is a baroreceptor-mediated tachycardia and vasoconstriction (5). There are conflicting reports on the effect of change in posture on MP. While Lathers at al (6) have reported that standing for 5

minutes produces minimal changes in MP, Sprangers et al (7) found a 25% fall in MP upon assuming standing posture. Bie et al (4) have reported that 20° HUT does not produce any change in MP and PP while 50° HUT produces a significant increase in MP as a result of increase in sympathetic activity. Van Brederode et al (8) have suggested that the immediate change in heart rate (HR) after active change of posture is due to "muscle heart reflex" that instantaneously inhibits cardiac vagal tone. The initial heart rate increase on standing is almost exclusively mediated by withdrawal of vagal tone, whereas the sustained tachycardia of later phases

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depends predominantly on sympathetic stimulation (2, 5, 9). Rate-Pressure-Product (RPP) is a reliable index of myocardial oxygen consumption and cardiac work and it correlates well with myocardial oxygen consumption of normal subjects as well as patients with angina pectoris (10, 11). HUT is likely to influence RPP as it alters preload (due to venous pooling) as well as afterload (due to change in the arterial pressure). However, there is paucity of literature on the effect of HUT on RPP. In view of this, the present study was planned to determine the effect of HUT on blood pressure and RPP, of normal young volunteers.

The objectives of our study were:

- 1. To study the cardiovascular effects of tilting since there are conflicting reports on the effect of change in posture on arterial pressure (6, 7).
- To study the time course of cardiovascular changes during five minutes of 30°, 60° and 80° HUT since the effects of tilting are likely to be influenced by the degree as well as duration of tilt (12).
- 3. To study the effect of HUT on RPP since there is paucity of literature in this area.

METHODS

Twenty male subjects aged 18-20 years from 1st year MBBS students of our college volunteered for this study. The subjects were free of any cardiovascular abnormality as revealed by history and clinical Indian J Physiol Pharmacol 2000; 44(4)

examination. Informed consent was obtained from them after explaining the experimental design. A few days before the study, the subjects were exposed to the laboratory environment and familiarised with 30°, 60° and 80° of HUT. On the day of the test, the subject was asked to report at the laboratory 2 h after a light breakfast. The temperature of the laboratory was maintained at $27 \pm 1^{\circ}$ C. The subject was made to lie supine on a tilting table and straps were applied at the levels of knees, waist and shoulders. A foot board was provided for supporting the feet during HUT. For recording blood pressure a dial type aneroid sphygmomanometer (Enma Japan) was used because its dial could be secured on the arm cuff and its position relative to arm/heart remained constant irrespective of the degree of tilt. For calculating heart rate from the R-R interval, lead II ECG was recorded on a polygraph (Model 7 polygraph, Grass Instruments, USA).

The recordings were taken in the following sequence :

1. Basal: 10 min after supine rest on the tilting table.

2. After 30° HUT.

3. After 60° HUT.

4. After 80° HUT.

Between each tilt, the subject was tilted back to the horizontal position and allowed to rest for 5 min. The Systolic blood pressure (SP), Diastolic blood pressure (DP), PP, MP, HR and RPP (RPP = SP × HR × 10^{-2}) were

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measured/calculated at each angle tilt immediately after and at 1, 2, 3, 4 and 5 min after the tilt. The data was subjected to statistical analysis using students "t" test. P values of less than 0.05 were taken as statistically significant.

RESULTS

30° HUT

30° HUT produced a marginal and insignificant decrease in SP immediately after the tilt (Table I). 1 min after the tilt there was a further small decrease in SP which remained insignificantly lower than the basal value throughout the 5 min period of the tilt. DP registered a marginal increase immediately after the tilt and subsequently returned towards the basal value. PP decreased immediately after the tilt. It showed a further decrease at 1 min and remained lower than the basal value throughout the 5 min tilt period. These changes in PP were not statistically significant. MP showed an immediate, insignificant increase after the tilt and was close to the basal value from 3 min onwards.

HR registered an immediate rise which was statistically significant (P<0.05). However, during the remaining period of the tilt, HR was not significantly different from the basal value. Immediately after the tilt, RPP showed an appreciable increase. However, this increase was not statistically significant. Subsequently, remained close the it to basal value throughout the 5 min duration of the tilt.

60° HUT

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The changes produced by 60° HUT were more pronounced than those produced by 30° HUT (Table II). Immediately after the tilt, SP decreased significantly and remained lower than the basal value (P<0.05) throughout the duration of the tilt. DP increased immediately after the tilt and remained elevated throughout the tilt. Although the elevation of DP was more than that recorded during 30° HUT, it was not statistically significant. PP which registered an immediate and significant fall after the tilt, remained significantly lower than the basal value throughout the duration of the

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

	В	Imm	No.SaTeres	2	3	151 S + 00 ⁴ ie al	Leonard 5	
Sp	104.80 ± 1.96	103.50±2.35	101.20 ± 2.92	101.85 ± 2.42	101.70 ± 2.20	101.40±2.30	101.60±2.58	
DP	65.00 ± 2.44	66.63±2.43	67.90 ± 2.04	67.80±2.08	66.60 ± 1.76	66.70 ± 1.59	65.57 ± 1.72	
PP	39.30 ± 2.62	36.30 ± 2.70	33.30 ± 3.05	33.15 ± 2.43	34.20 ± 2.07	34.10 ± 2.45	35.57±2.68	
MP	78.60 ± 1.95	79.42 ± 2.01	79.55 ± 1.87	79.65 ± 1.91	78.80 ± 1.69	78.50 ± 1.43	77.94 ± 1.59	
HR	70.33 ± 1.93	$80.15 \pm 3.65^*$	69.80 ± 1.79	71.55 ± 1.90	71.20 ± 1.79	71.60 ± 1.84	73.15 ± 2.20	
RPP	73.35 ± 2.81	81.43 ± 4.16	70.45 ± 2.52	72.78±2.50	72.59 ± 2.18	72.78 ± 2.14	75.85 ± 2.56	

Values are mean ± SE. Pressure values are in mmHg. B: basal values before the tilt; Imm: immediately after the tilt;

1, 2, 3, 4, 5: minutes after the tilt.

*P<0.05 compared to basal value.

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TABLE II:	Effect of 60	° head-up til	t on systolic	pressure (SP)	, diastolic	pressure	(DP), pulse pressure	в
10. 0. 01. 11	(PP), mean	pressure (M	?), heart rate	e (HR) and ra	te pressur	e product	(RPP).	

101	В	Imm	1 10099	2	3	4	5
Sp	104.80 ± 1.96	98.30±2.23*	98.50±2.07*	97.40±2.62	97.60±2.42*	97.10±2.66**	98.10±2.66*
DP	65.00 ± 2.44	69.10±2.17	69.70±2.32	68.80 ± 2.08	69.80 ± 2.22	69.60±2.04	68.80±2.06
PP	39.30 ± 2.62	28.55±2.54***	28.00±2.40***	27.60±2.46***	26.90±2.34***	27.00±2.49***	28.30±2.62***
MP	78.60 ± 1.95	79.25 ± 1.77	79.40 ± 1.73	79.10 ± 1.93	79.60±1.99	79.10 ± 1.82	78.80 ± 1.91
HR	70.33 ± 1.93	$81.89 \pm 2.25^{****}$	78.65±2.37****	83.85±2.24****	85.00±2.04****	83.65±1.54****	84.70±1.67****
RPP	73.35 ± 2.81	79.42 ± 2.33	78.11±2.22	81.15±2.19*	82.55±1.96**	80.48±1.99*	82.07±2.06**

Values are mean \pm SE. Pressure values are in mmHg. B: basal values before the tilt; Imm: immediately after the tilt; 1, 2, 3, 4, 5: minutes after the tilt.

*P<0.05; **P<0.02; ***P<0.01; ****P<0.001 compared to basal value.

tilt (P<0.01). The increase in MP was marginal and insignificant. Immediately after the tilt, there was a marked increase in HR, which remained elevated throughout the 5 min tilt period. This increase in HR was highly significant (P<0.001). There was an appreciable but insignificant increase in RPP immediately after the tilt. 2 min after the tilt, it increased further and remained significantly (P<0.02) elevated throughout the duration of the tilt. Nete was a further small decrease in SF

 80° HUT produced changes which were similar in direction, but more pronounced than those seen during 60° HUT (Table III). SP decreased significantly (P<0.05) while DP and MP increased insignificantly. PP registered a significant (P<0.001) fall, which was more pronounced than during 60° HUT. HR and RPP increased significantly and their increase was more pronounced than 60° HUT.

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

	В	Imm	1	2	3	4	5
Sp	104.80 ± 1.96	99.00±2.33	97.90±2.72*	97.90±2.80	97.57±2.69*	96.84±2.99*	97.86±3.21
DP	65.00 ± 2.44	68.50 ± 2.24	69.50±2.12	69.30±2.04	70.30±2.20	70.20±2.31	70.42 ± 2.21
PP	39.30 ± 2.62	29.05±2.68***	27.60±2.65***	27.10±2.48***	25.78±2.61****	25.44±2.99***	24.84±2.57****
MP	78.60 ± 1.95	79.31 ± 1.79	79.25 ± 1.89	79.25 ± 2.04	80.00±2.00	79.38±2.20	80.20 ± 2.38
HR	70.33 ± 1.93	83.60±2.03****	86.00±2.46****	86.45±2.07****	86.25±2.11****	86.50±3.07****	89.50±2.51****
RPP	73.35 ± 2.81	$80.86 \pm 1.36^*$	83.39±2.86**	83.71±2.56***	83.40±2.53**	84.44±2.92***	85.32±2.96***

Values are mean \pm SE. Pressure values are in mmHg. B: basal values before the tilt; Imm: immediately after the tilt; 1, 2, 3, 4, 5: minutes after the tilt.

*P<0.05; **P<0.02; ***P<0.01; ****P<0.001 compared to basal value.

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DISCUSSION

In the present study, we have recorded the effect of different degrees of HUT on cardiovascular parameters in healthy, young volunteers. SP and PP decreased while DP, MP, HR and RPP increased progressively as the angle of HUT increased. At 30° HUT, the changes were insignificant except a significant increase in HR immediately after the tilt. This agrees with the findings of Bie et al (4) who have reported that 20° HUT does not produce significant changes except a slight but significant increase in HR. Orthostatic stress is mediated by arterial baroreceptor as well as cardiopulmonary reflex mechanisms which are not markedly affected by lower levels of HUT. Hence, changes during 30° HUT were insignificant except for a transient tachycardia immediately after the tilt. The increase in HR during 60° and 80° tilt was tilt-dependent and highly significant throughout tilt period of 5 minutes. Similar findings have been reported by other workers (4, 5, 13, 14). This increase in HR may be due to withdrawal of vagal tone and/ or an increase in sympathetic tone. Marin Neto et al (9) have concluded that the initial tachycardia elicited by the upright posture is due to parasympathetic withdrawal. Jahan et al (13) also have concluded that tilting results in alteration of stimulus bound changes in parasympathetic reactivity and during the tilt there is a decrement of parasympathetic tone rather than enhancement of sympathetic tone. However, other workers have concluded that sympathetic activation plays a crucial role in increasing HR during higher grades of the tilt (2, 5, 9). Work done by Wieling et al (15) suggests that neural compensatory

mechanisms are very effective in maintaining arterial pressure in normal healthy human subjects.

In contrast to 30° HUT, 60° HUT and 80° HUT produced more pronounced and significant changes in SP, PP and RPP. However, the increase in DP and MP was not statistically significant. These changes in SP and DP were associated with highly significant decrease in PP, the decrease being tilt dependent. Our results are different from those of Jahan et al (13) who have reported a significant increase in DP after 70° HUT. This apparent discrepancy may be due to the fact that Jahan et al have incorporated procedures like Valsalva maneuver and hand grip test along with HUT in their work. Changes in MP and PP at higher degree of tilt in our study are similar to those of other studies (4, 5). These changes are associated with increase in the levels of epinephrine, norepinephrine and aldosterone (4, 16, 17). Plasma norepinephrine levels are known to increase progressively as the angle of tilt increases from 0 to 80° (17, 18).

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Tilting produced an immediate increase in RPP which was maintained throughout the 5 min duration of the tilt. This increase was highly significant at 80° HUT. RPP correlates well with myocardial oxygen consumption and is an index of cardiac work in normal subjects as well as patients with heart diseases (10, 11). The increase in RPP in our subjects suggests that tilting produces an increase in load on the heart.

In conclusion, our study shows that postural stress in the form of HUT produces sustained increase in HR and RPP. This can 472 Vijayalakshmi et al.

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be explained on the basis of sustained changes in autonomic tone as well as

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In conclusion, our study shows the postural strugg in the form of H17 produce sustained increase in SR and R147 This can hormonal levels, which are known to alter the cardiovascular responses (16-19).

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