Indian J Physiol Pharmacol 1991; 35(4): 255-259

EFFECT OF HEAD-UP TILTING ON SYSTOLIC TIME INTERVALS IN NORMAL YOUNG VOLUNTEERS

S. VIJAYALAKSHMI*, MADAN MOHAN, D. P. THOMBRE, BHARATHI BALAKUMAR AND N. KRISHNAMURTHY

Department of Physiology,
Jawaharlal Institute of Postgraduate Medical Education and Research,
Pondicherry - 605 006

(Received on April 24, 1991)

Abstract : Systolic time intervals (STI) are sensitive indices of myocardial function. Passive tilting is a rapidly reversible and non-invasive method for inducing cardiovascular stress. The present work was conducted to study the effect of graded head-up tilt (HUT) on STI. 20 male medical students were subjected to 30°, 60° and 80° HUT on a tilting table. ECG, phonocardiogram and carotid pulse were recorded simultaneously on Grass polygraph. Electromechanical systole (QS_2), left ventricular ejection time (LVET), pre-ejection period (PEP), PEP/LEVT ratio, heart rate (HR) and corrected STI were determined immediately after and at 1, 2, 3, 4 and 5 min after each angle tilt. HUT produced a decrease in QS_2 which was more pronounced at higher angle tilt. LVET decreased after 60° and 80° HUT. PEP and PEP/LVET ratio decreased after each angle tilt. These changes in STI can be explained on the basis of sympathetic stimulation-induced increase in the inotropic state of the heart.

Key words : head-up tilt cardiovascular reflexes

INTRODUCTION

Systolic time intervals (STI) are sensitive indices of myocardial performance and are ideal for assessment of the inotropic state of normal as well as diseased heart (1,2). There are a number of reports on the effect of heart diseases on STI (3-7). However, the effect of passive tilting on STI has not received due attention and the normal data in Indian population is not adequate. Passive tilting is an easily gradable, rapidly reversible and sensitive method for producing postural stress on the cardiovascular system. Tilting can be used for assessing the integrity of autonomic cardiovascular regulatory mechanisms in physiological as well as clinical situations. Tilting as well as measurement of STI are non-invasive and sensitive tests. Hence, we planned to study the effect of heat-up tilt (HUT) on STI in normal young healthy volunteers. The effects of tilting are likely to be influenced by the degree of the tilt as well as the duration for which the subject is tilted (8). Therefore, we studied the time course of STI changes after 30°, 60° and 80° HUT.

METHODS

This study was conducted on 20 healthy male medical students aged 18-20 yr. A few days before the study, the subjects were acquainted with the laboratory environment and familiarised with 30°, 60° and 80° HUT. After explaining the experimental design, informed consent was obtained from them. On the day of the test, the subject reported at the laboratory 2 h after a light breakfast. The laboratory temperature was maintained at 27±1°C. After 30 min of rest and instructions, the subject was made to lie supine on a tilting table and straps were applied at the levels of knees, waist and shoulders. During the HUT the subject's feet were supported by a foot board. Care was taken to see that there was no discomfort to the subject or interference with his breathing. Lead II

*Corresponding Author

ECG, phonocardiogram and carotid pulse were recorded simultaneously on Grass model 7 polygraph. Electromechanical systole (QS_2) was measured as the time (ms) from the onset of QRS complex to the first high frequency vibration of the second heart sound. Left ventricular ejection time (LVET) was measured as the duration (ms) between the beginning of the upstroke to the trough of the dicrotic notch of the carotid pulse tracing. Pre-ejection period (PEP) was calculated as the difference between QS_2 and LVET (PEP = QS_2-LEVT). Heart rate (HR) was calculated from RR interval of the ECG. To calculate STI indices, the values were corrected for HR using Weissler's original HR-STI regressions (9). The recordings were taken in the following sequence.

1. Control (before tilt) : 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 horizontal position and rested for 10 min. At each angle tilt the recordings were taken immediately after and at 1, 2, 3, 4 and 5 min after the tilt.

The data was subjected to statistical analysis using Student's "t" test. P values of less than 0.05 were taken as statistically significant.

RESULTS

The results are given in Table I, II and III.

Effect of 30° HUT (Table I): Immediately after 30° HUT, QS_2 decreased insignificantly. 1 min after the tilt, it returned towards the control value and remained relatively stable throughout the duration of the tilt. LVET increased up to 1 min after the tilt followed by a small recovery. But, this increase in LVET was not statistically significant. PEP showed a sharp but insignificant decrease immediately after the tilt. At 1 min it decreased further followed by a progressive increase throughout the 5 min study period. These changes in PEP were not significant statistically. PEP/LVET ratio decreased immediately after the tilt. 1 min post tilt the ratio showed a further and significant decrease. Thereafter, it showed a progressive recovery, but remained lower than the control throughout the duration of the tilt. The decrease in PEP/LEVT ratio at 1 and 2 min was statistically significant ($P < 0.02$ and < 0.05 respectively). Immediately after the tilt, HR was significantly higher than the control value ($P < 0.05$). However, the HR returned towards the control at 1 min after the tilt.

TABLE I : Effect of 30° head-up tilt on systolic time intervals (in milli sec).

	Before tilt	*After tilt*					
		Immediate	*1 min*	*2 min*	*3 min*	*4 min*	*5 min*
QS_2	335.2	313.3	327.0	332.2	332.2	332.2	334.4
(ms)	±13.23	±12.31	±12.82	±5.14	±7.60	±6.49	±6.22
LVET	237.6	245.5	260.0	258.8	253.3	251.1	252.2
(ms)	±13.49	±11.63	±6.41	±4.41	±5.82	±4.63	±4.32
PEP	97.64	68.88	65.88	73.33	78.88	81.11	82.22
(ms)	±14.33	±12.75	±8.36	±5.82	±5.93	±6.15	±7.02
PEP/LVET	0.46	0.33	0.24	0.28	0.31	0.32	0.32
ratio	±0.08	±0.09	±0.03**	±0.02*	±0.02	±0.02	±0.03
HR	70.33	80.15	69.8	71.55	71.2	71.6	73.15
beats/min	±1.93	±3.65*	±1.79	±1.90	±1.79	±1.84	±2.20
QS_2I	497.37	484.3	478.72	475.13	484.46	490	491.96
	±5.69	±11.14	±12.68	±12.05	±7.95	±6.00	±6.03
LVETI	364.41	384.02	366.77	373.26	377.26	375.33	374.76
	±11.53	±10.60	±10.21	±10.10	±4.67	±5.16	±5.61
PEPI	132.96	100.32	107.94	101.78	107.2	114.66	117.2
	±13.40	±16.71	±8.07	±6.88*	±7.17	±8.92	±8.79

Values are means ± SEM.

QS_2 : electromechanical systole; LVET : left ventricular ejection time ;

PEP : pre-ejection period; HR : heart rate; QS_2I : QS_2 index; LVETI : LVET index;

PEPI : PEP index.

*$P < 0.05$; **$P < 0.02$.

Effect of 60° HUT (Table II) : QS_2 decreased immediately after 60° HUT. Thereafter, it remained relatively stable upto 4 min, but decreased further ($P < 0.05$) at 5 min post tilt. LVET showed a small and insignificant decrease at 1 min after which its value remained insignificantly lower than the control. PEP decreased immediately after the tilt and thereafter its value fluctuated but remained insignificantly lower than the control throughout the 5 min duration of the tilt. PEP/LVET ratio decreased immediately after the tilt but recovered partially at 1 min post tilt, so that its value was insignificantly lower than the control during the 5 min study period. There was a significant ($P < 0.001$) increase in HR immediately after the tilt. This significant tachycardia was maintained throughout the study period. In general, the changes in corrected STI indices were not statistically significant except that immediately after the tilt, LEVTI was higher than the control and PEPI was lower than the control, both these changes being statistically significant.

TABLE II : Effect of 60° head-up tilt on systolic time intervals.

	Before tilt	*After tilt*					
		Immediate	*1 min*	*2 min*	*3 min*	*4 min*	*5 min*
QS_2 (ms)	335.2 ±13.23	313.7 ±7.89	314.1 ±8.18	305.8 ±6.58	306.6 ±7.58	308.8 ±6.51	304.4 ±6.77*
LVET (ms)	237.6 ±13.49	238.7 ±6.18	227.0 ±6.16	227.0 ±6.84	22.5.5 ±4.79	232.2 ±4.88	222.2 ±5.08
PEP (ms)	97.64 ±14.33	75.0 ±5.01	87.05 ±8.21	78.82 ±6.74	81.11 ±5.23	77.77 ±6.23	81.11 ±7.13
PEP/LVET ratio	0.46 ±0.08	0.31 ±0.02	0.39 ±0.04	0.35 ±0.03	0.35 ±0.02	0.33 ±0.03	0.37 ±0.03
HR beats/min	70.33 ±1.93	81.89 ±2.25****	78.65 ±2.37****	83.85 ±2.24****	85.0 ±2.04****	83.65 ±1.54****	84.7 ±1.67****
QS_2I	497.37 ±5.69	497.16 ±12.48	481.33 ±8.31	478.85 ±7.97	485.3 ±7.87	491.42 ±7.94	485.73 ±8.10
LVETI	364.41 ±11.53	395.98 ±8.56*	366.66 ±5.10*	370.37 ±6.02	376.1 ±5.14	376.67 ±4.77	369.46 ±4.07
PEPI	132.96 ±13.40	101.17 ±7.50*	114.66 ±10.17	108.48 ±8.88	110.53 ±5.67	117.41 ±.9.86	116.26 ±10.02

Values are means ± SEM.
QS_2 : electromechanical systole; LVET : left ventricular ejection time ;
PEP : pre-ejection period; HR : heart rate; QS_2I : QS_2 index; LVETI : LVET index;
PEPI : PEP index.
*P < 0.05; **P < 0.02; ****P < 0.001

Effect of 80° HUT (Table III): There was immediate and significant ($P < 0.02$) decrease in QS_2 after 80° HUT. Except at 2 min post tilt, the value of QS_2 was significantly lower than the control throughout the study period. The values of LVET, PEP as well as PEP/LVET ratio decreased after the tilt and remained insignificantly lower than the control throughout the 5 min duration of the study. There was a significant ($P < 0.001$) tachycardia immediately after the tilt which was maintained throughout the study period. In general, the pattern of changes in corrected STI indices followed those of the STI changes. However, these change were not statistically significant.

TABLE III : Effect of 80° head-up tilt on systolic time intervals.

	Before tilt	After tilt					
		Immediate	1 min	2 min	3 min	4 min	5 min
QS_2 (ms)	335.2 ±13.23	296.4 ±8.26**	302.3 ±7.83*	305.8 ±7.42	295.2 ±6.75**	300.0 ±7.52*	304.7 ±6.30*
LVET (ms)	237.6 ±13.49	224.7 ±5.82	217.6 ±5.65	222.3 ±5.65	221.1 ±4.68	220.0 ±6.32	223.5 ±4.92
PEP (ms)	97.64 ±14.33	72.94 ±8.73	84.70 ±7.38	83.52 ±7.52	75.29 ±6.97	80.0 ±8.36	81.17 ±5.54
PEP/LVET ratio	0.46 ±0.08	0.33 ±0.04	0.39 ±0.04	0.38 ±0.04	0.33 ±0.03	0.37 ±0.05	0.36 ±0.02
HR beats/min	70.33 ±1.93	83.6 ±2.03****	86.0 ±2.46****	86.45 ±2.07****	86.25 ±2.11****	86.5 ±3.07****	89.5 ±2.51****
QS_2I	497.37 ±5.69	483.75 ±9.66	485.93 ±6.49	480.74 ±9.02	482.56 ±7.88	482.56 ±7.88	489.36 ±8.40
LVETI	364.41 ±11.53	370.34 ±5.24	362.2 ±7.28	364.16 ±6.99	370.45 ±5.25	363.9 ±4.35	368.04 ±6.69
PEPI	132.96 ±13.40	113.41 ±11.42	123.73 ±9.91	117.92 ±10.54	112.10 ±8.45	125.46 ±9.85	121.81 ±8.80

Values are means ± SEM.
QS_2 : electromechanical systole; LVET : left ventricular ejection time ;
PEP : pre-ejection period; HR : heart rate; QS_2I : QS_2 index; LVETI : LVET index;
PEPI : PEP index.
*P < 0.05; **P < 0.02; ***P < 0.01; ****P < 0.001.

DISCUSSION

STI correlate well with invasively determined measures of myocardial performance (1, 2). Being non-invasive and useful measures of left ventricular function (10), they have been used by a number of workers for assessing cardiac function in clinical situation (1, 5, 6, 10-12). In the present study, we have determined the effect of HUT on STI. Maximum tilt used was 80° since the hydrostatic effects of 80° tilt are quite similar to those of 90° tilt (sin 80° = 0.98; sin 90° = 1) whereas the subject feels more comfortable and relaxed at 80° tilt.

Tilting produced a decrease in QS_2, the decrease being more at higher angle tilt. This decrease in QS_2 could be due to shortening of cardiac cycle time as a result of tilt-induced tachycardia (8). Stafford et al (13) also have reported a decrease in QS_2 after HUT. Cokkinos et al (14) have reported that LVET decreases when heart rate is increased by right atrial pacing or by atropine administration. Stafford et al (13) have reported a decrease in LVET after HUT. However, in the present study, LVET decreased only during 60° and 80° HUT and the decrease was not statistically significant. Gillian et al (10) have reported exercise induced slight decrease in LVET in normal subjects. This suggests that the tilt induced decrease in LVET is due to an increase in the inotropic state of the heart as a result of sympathetic stimulation. The gravitation-induced decrease in venous return and stroke volume will also produce a decrease in LVET, sympathetic stimulation and release of catecholamines improve contractility and decrease PEP (11, 15). PEP includes isovolumetric contraction phase and is a good measure of contractility, since it has a significant correlation with left ventricular dP/dt (10). PEP/LVET ratio also has been shown to have an excellent correlation with myocardial contractility (1). In the present study PEP as well as PEP/LVET ratio decreased after HUT, the decrease being similar in pattern in all the three angle tilts. This decrease in PEP and PEP/LVET ratio indicates an increase in the inotropic state of the heart as a result of HUT. The pattern of changes in the calculated STI indices was in general, similar to those of STI.

30° HUT produced a transient but significant tachycardia. During 60° and 80° tilt, there was a sustained and significant increase in HR, the degree of tachycardia being more at higher angle tilt. Tilting produces a gravity-dependent venous pooling and a decrease in cardiopulmonary blood volume and pulse pressure. This results in a reduction in the inhibitory afferent traffic from the low pressure cardiopulmonary mechanoreceptors and high pressure systemic arterial baroreceptors and a consequent reflex tachycardia (16, 17).

In conclusion, our study shows that HUT produces a decrease in STI and this can be explained on the basis of increase in the sympathetic activity during the tilt.

REFERENCES

1. Ahmed SS, Levinson GE, Scharty CJ et al. Systolic time intervals as measure of the contractile state of the left ventricular myocardium in man. *Circulation* 1972; 46:559-571.
2. Weissler AM, Harris WS, Schoenfeld CD. Bedside technics for the evaluation of ventricular function in man. *Am J Cardiol* 1969; 23:577-583.
3. Khuller S, Lewis RP. Usefulness of systolic time intervals in differential diagnosis of constrictive pericarditis and restrictive cardiomyopathies. *Br Heart J* 1976; 38:43-46.
4. Lewis RP, Rittgeris SE, Forester WF et al. A critical review of the systolic time intervals. *Circulation* 1977; 56:146-157.
5. McConahay DR, Martin CM, Cheitlin MD. Resting and exercise systolic time intervals. *Circulation* 1972; 45:592-601.
6. Samson R. Changes in STI is acute myocardial infarction. *Br Heart J* 1970; 32: 839-846.
7. Weissler AM, Peeler RG, Roehall WM et al. Relationship between ventricular ejection time, Stroke Volume and heart rate in normal individuals and patients with cardiovascular disease. *Am Heart J* 1961; 62:367-378.
8. Tuckman J, Schillingford J. Effect of different degress of tilt on CO, HR, BP in normal man. *Br Heart J* 1966; 28: 32-39.
9. Weissler AM, Harris WS, Schoenfeld CD. Systolic time intervals in heart failure in man. *Circulation* 1968; 37:149-159.
10. Gillian RE, Parnes WP. The prognostic value of systolic time intervals in angina pectoris. *Circulation* 1979; 60:268-275.
11. Hamosh P, Cohn JN, Engelman K et al. STIs and left ventricular function in acute myocardial infarction. *Circulation* 1972; 45:375-381.
12. Rater D, Paugh D, Gray W. Practical significance of systolic time intervals in coronary artery disease. *Chest* 1973; 64:186-188.
13. Stafford RW, Harris WS, Weissler AM et al. Systolic time interval as indices of gravitational circulatory stress in man. *Am J Cardiol* 1967; 19(Abst): 152.
14. Cokkinos DV, Heimonar TE, Demopoulas JV et al. Influence of heart rate increase on the uncorrected PEP/LVET ratio in normal individuals. *Br Heart J* 1976; 38: 683-688.
15. Harris WS, Schoenfeld CD, Weissler AM. Effect of adrenergic receptors activation and blockade on the systolic pre-ejection period, heart rate and arterial pressure in man. *J Clin Invest* 1967; 46:1704-1714.
16. Abbound FM, Eckberg DL, Johannsen UJ et al. Carotid and baroreceptor control of splanchnic and forearm vascular resistance during venous pooling in man. *J Physiol* (London) 1979; 286: 173-184.
17. Sjostrand T. The regulation of blood distribution in man. *Acta Physiol Scand* 1952; 26:312-327.