

# **PACIFIC JOURNAL OF MEDICAL SCIENCES**

**{Formerly: Medical Sciences Bulletin}**

**ISSN: 2072 – 1625**



**Pac. J. Med. Sci. (PJMS)**

[www.pacjmedsci.com](http://www.pacjmedsci.com). Email: [pacjmedsci@gmail.com](mailto:pacjmedsci@gmail.com).

---

## **EFFECT OF DIFFERENT DEGREES OF TILT ON HEART RATE, PULSE PRESSURE AND MEAN ARTERIAL BLOOD PRESSURE IN YOUNG MALE AND FEMALE NIGERIANS**

**Rapheal A. Oguntola and \*Bamidele V. Owoyele**

Department of Physiology University of Ilorin, Ilorin, Nigeria

*Running title: Different degrees of tilt and cardiovascular parameters*

\*Correspondence author: [deleyele@yahoo.com](mailto:deleyele@yahoo.com) or [owoyele@unilorin.edu.ng](mailto:owoyele@unilorin.edu.ng)

**EFFECT OF DIFFERENT DEGREES OF TILT ON HEART RATE, PULSE PRESSURE AND MEAN ARTERIAL BLOOD PRESSURE IN YOUNG MALE AND FEMALE NIGERIANS****Rapheal A. Oguntola and \*Bamidele V. Owoyele**

Department of Physiology University of Ilorin, Ilorin, Nigeria

*Running title: Different degrees of tilt and cardiovascular parameters*\*Correspondence author: [deleyele@yahoo.com](mailto:deleyele@yahoo.com) or [owoyele@unilorin.edu.ng](mailto:owoyele@unilorin.edu.ng)**ABSTRACT:**

It is well documented that changes in cardiac output are lower in the upright than supine positions. This study investigates the effect of different angles of tilt on the heart rate, pulse pressure and mean arterial pressure (MAP) of healthy male and female subjects in the 18 to 24 years age group and heights between 150 -180cm. All subjects were students in the University of Ilorin. Each participant was studied and the parameters were determined at the supine position and head up tilts at 15°, 30°, 45° and, 60° for 15 minutes interval. Additionally the supine position variables were taken as the resting value against each of the head up tilt angle degree. The result shows that there was progressive and significant increase in heart rate from supine  $59.5 \pm 1.3$  beat/min to  $62.2 \pm 1.4$  beat/min ( $p < 0.05$ ) at 15°;  $59.5 \pm 1.3$  beat/min to  $63.8 \pm 1.4$  beat/min at 30° tilt ( $p < 0.05$ );  $59.5 \pm 1.3$  beat/min to  $65.9 \pm 1.4$  beat/min at 45° tilt ( $p < 0.05$ );  $59.5 \pm 1.3$  beat/min and  $68.2 \pm 1.6$  beat/min at 60° tilt ( $p < 0.05$ ). The results also showed that there were significant ( $p < 0.05$ ) increases in pulse pressure at all angles which peaked at 30° tilt. Furthermore, it was observed that MAP significantly ( $p < 0.05$ ) increased with corresponding increase in the angle of tilt from supine to 60° ( $76.5 \pm 2.2$  mmHg (supine) to  $80.6 \pm 2.1$  mmHg (15 °) to  $83.8 \pm 2.1$  mmHg (30 °) to  $85.4 \pm 2.1$  mmHg (45 °) to  $85.4 \pm 2.2$  mmHg (60 °). Our results demonstrated that the heart rate, pulse pressure and MAP were significantly ( $p < 0.05$ ) increased in males and females for all the variables.

**Keywords:** Tilt, heart rate; pulse pressure; mean arterial blood pressure*Submitted: May 2015, Accepted: August 2015*

**INTRODUCTION:**

Many workers have shown that the cardiac output in a healthy individual is less in the upright than in the recumbent position [1, 2]. During a moderate posture change in human from sitting to supine, mean arterial blood pressure (MAP) decreases [1, 2]. The mechanisms for this involve cardiopulmonary low and arterial high pressure receptor stimulation which induces peripheral vasodilatation and decrease in heart rate [3]. The muscle vascular bed is sensitive to changes in baro-receptor stimulation [4]. Many senescent individuals demonstrate an ability to regulate MAP in response to standing or head-up tilt [5]. In some individuals, sudden standing causes a fall in blood pressure, dizziness, dimness of vision and fainting. The causes of this orthostatic (postural) hypotension are multiple. The major compensation on assuming the upright positions are triggered by drop in blood pressure in the carotid sinus and aortic arch [3, 6, 7, 8]. The heart rate increases helping to maintain cardiac output. There is relatively little vasoconstriction in the periphery and arterial pressure as head level drops but jugular venous pressure falls reducing the drop in perfusion pressure (arterial pressure- venous pressure) [9].

Generally, increases in cardiac output increases the systolic pressure whereas increase peripheral resistance increases the

diastolic pressure. The physiological events underlying all these aforementioned changes are complex and depend on many variables, such as the angle of tilt and length of time the patient remains at a given angle and whether the change is from recumbence to upright position or vice-versa. Posture and its relation to cardiac output is important with the introduction of hypotensive drugs [10]. This study aimed to determine the relationship between angle tilt and changes in heart rate, pulse pressure and mean arterial blood pressure in young adult Nigerians.

**SUBJECTS AND METHODS:**

Fifty healthy volunteers (30 males and 20 females) who were students in the University of Ilorin participated in this study. The age range was between 18 and 24 years, heights between 150 to 180cm and weights between 55 to 77kg. The volunteers were normotensive (supine blood pressures; not greater than 120/80 mmHg, were not taking medications, and had no known cardiovascular diseases. The volunteers were requested to refrain from drinking caffeine and alcohol, and not to be involved in intensive exercise 24 hours before the study if they were not accustomed to physical exercise.

This study followed the guidelines approved by the Ethical committee of the University of Ilorin,

and a written informed consent was obtained from each volunteer.

The heart rate was measured using standard (automated) digital sphygmomanometer and was confirmed manually using radial artery. The systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured using the automated sphygmomanometer and the blood pressure readings confirmed by aneroid manual sphygmomanometer. The pulse pressure (PP) was estimated by the difference between systolic and diastolic blood pressure that is calculated using formula:  $PP = SBP - DBP$ . The mean arterial blood pressure was determined indirectly by  $DBP + 1/3PP$  [10].

Studies were conducted three hours after breakfast or lunch [10]. The subject was

weighed and height determined. Thereafter, the subject was placed on the tilt bed, maintained in a supine position and heart rate and arterial blood pressure were determined. All subjects were maintained in the supine position for thirty (30) minutes before readings were taken. The variables measured were; heart rate, systolic and diastolic pressures. The experiment was done for four (4) different degrees of tilt [10].

The tilt positions were 15°, 30°, 45° and 60°. Each subject was tilted to all these positions sequentially one after the other starting from the least angle; and readings were taken at 15 minutes interval for each angle. The passive postural changes were performed by manual tilt bed. The duration of the procedure was about two hours for each subject [10].

Percentage change was calculated as follows [10]:

$$\text{Percentage change (\% } \Delta) = \frac{(b - a) \times 100}{a}$$

Where a= values of variables at rest (supine position) and  
b= values of variables after each tilts

The mean values of heart rate, pulse pressure and MAP were determined and the standard errors of the means were calculated. Significant responses were evaluated by Student t-test for paired comparison [11];  $P \leq 0.05$  was taken as

statistically significant. The Student paired t-test was used to detect whether means differed at similar points in time, comparing values of corresponding series (supine vs 15° tilt, supine

vs 30° tilt, supine vs 45° tilt and supine vs 60° tilt).

## RESULTS:

### Effect of different degrees of tilt on the male subjects:

All the male subjects showed significant increase in heart rate at 15° tilt after 15 minutes interval of postural change (supine to 15° tilt). The mean value increase from 59.5±1.3beat/min to 62.2 ± 1.4beat/min (an increase of 4.7 ± 1.1%, p<0.05, n=30). Continuous increase in heart rate was also observed as the tilt angles increases, after 15 minutes interval. The overall mean heart rate increases with changes in tilt angle, supine and 30° tilt: 59.5 ± 1.3 and 63.8 ± 1.4beat/min; supine and 45° tilt: 59.5 ± 1.3 and 65.9 ±

1.4beat/min; supine and 60° tilt: 59.5 ± 1.3 and 68.2 ± 1.6beat/min. The change in heart rate was significant as shown in the table 1.

All the male subjects also showed increase in pulse pressure at various angles of tilt and the increase was highest at angle 30° tilt.

At angle 45° and 60° tilt, the pulse pressure fell from the value obtained in 30° tilt. Hence, mean pulse pressure significantly increased from supine position to 30° tilt as shown in table 1. As can be seen in Table 1, all the male subjects showed increase in MAP and the increase was in line with increase in the angle of tilt. Overall, MAP increases with change in angle of tilt. The changes were significant (p<0.05).

Table 1: Effect of different degrees of tilt on heart rate, pulse pressure and mean arterial blood pressure for the male and female subjects

Subjects		Supine (a)	15° tilt (b)	30° tilt (b)	45° tilt (b)	60° tilt (b)
Male (n=30)	Heart Rate (bpm)	59.5±1.3	62.2±1.4*	63.8±1.4*	65.9±1.4*	68.2±1.6*
	Pulse Pressure (mmHg)	39.0±1.1	42.4±1.2*	46.7±1.1*	44.1±1.0*	41.6±0.8*
	MAP (mmHg)	76.5±2.2	80.6±2.1*	83.8±2.1*	85.4±2.1*	85.4±2.2*
Female (n=20)	Heart Rate (bpm)	66.1±1.7	68.4±1.8*	69.7±1.8*	71.4±1.8*	74.3±1.7*
	Pulse Pressure (mmHg)	39.4±0.9	42.0±0.9*	44.7±0.9*	41.9±1.0*	41.0±0.9*
	MAP (mmHg)	78.4±2.1	82.7±1.8*	85.2±1.8*	85.4±2.1*	86.4±1.9*

Data are Mean ± SEM values, \*P<0.05 significantly different from control: supine vs 15 ° tilt, supine vs 30 ° tilt, supine vs 45° tilt, supine vs 60° tilt. Where a= values of variables at rest (supine position) and b= values of variables after each tilts

Table 2: Percentage changes of heart rate, pulse pressure and mean arterial blood pressure for the different degrees of tilts compared to the supine for the male and female subjects

Subjects		15° tilt (a)	30° tilt (a)	45° tilt (a)	60° tilt (a)
Male (n=30)	Heart Rate (bpm)	4.7±1.1*	7.2±1.4*	10.8±0.7*	14.7±1.3*
	Pulse Pressure (mmHg)	8.9±1.6*	21.1±3.4*	14.4±2.8*	8.0±2.4*
	MAP (mmHg)	5.8±1.1*	10.0±0.9*	12.3±1.2*	12.1±1.3*
Female (n=20)	Heart Rate (bpm)	3.5±0.5*	5.4±0.6*	8.0±0.7*	12.6±0.8*
	Pulse Pressure (mmHg)	6.8±1.1*	13.7±1.8*	6.5±1.7*	4.2±1.4*
	MAP (mmHg)	5.8±1.2*	9.1±1.3*	11.8±1.5*	10.7±1.7*
	*p-value	<0.05	<0.05	<0.05	<0.05

Data are Mean ± SEM values, a = values of variables after each tilts.

#### Effect of different degrees of tilt on the female subjects:

All the female subjects showed significant increase in heart rate as the angle of tilt increases. The mean heart rate increased from supine to 15° tilt, 30° tilt, 45° tilt and 60° tilt; the corresponding values were 66.1 ± 1.7 and 68.4 ± 1.8 beat/min, 66.1 ± 1.7 and 69.7 ± 1.8 beat/min, 66.1 ± 1.7 and 71.4 ± 1.8 beat/min, 66.1 ± 1.7 and 74.3 ± 1.7 beat/min. The changes in heart rate were significant ( $p < 0.05$ ) as shown in Table 1. Increase in pulse pressure at various angles of tilt was observed for all the female subjects; the increase was highest at angle 30° tilt (Table 1). At angle 45° tilt and 60° tilt, the pulse pressure fell from the value obtained in 30° tilt. The mean pulse pressure significantly increased

( $p < 0.05$ ) from supine position to 30° tilt as shown in tables 1 and 2. The MAP for the female subjects increased from supine to the different angles of tilts. The changes in the angles of tilts are presented in tables 1 and 2.

#### DISCUSSION:

The subjects were selected based on their health status. Subjects with history of cardiovascular diseases, hypertension, smokers, and history of kidney diseases were excluded from the study. This is because the major products of tobacco combustion, nicotine and carbon dioxide are potent vasoconstrictors [12, 13]. Besides, nicotine stimulation increased secretion of catecholamine. The net effect is that there will be increase in peripheral

vascular resistance and this can lead to elevation of blood pressure [13].

The results obtained for both male and female subjects showed that slight increase in heart rate occurred during the first 15 minutes of tilting to the head-up position of 15° and thereafter increase progressively as the angle of head up position was increased to 30° tilt, 45° tilt and 60° tilt as presented in table 2. When compared with the recumbent values, the average heart rate at those angles was increased. The changes were significant at  $p < 0.05$ . The increase in heart rate which began at 15° tilt and gradually increased after, and the probable simultaneous augmentation of myocardial contractility (also caused by a reflex increase of sympathetic nervous system activity) prevented further decrease in cardiac output [14]. However, the increase in heart rate is in agreement with the findings of Tuckman and Shillingford [10]. The increase might be due, in part, to withdrawal of vagal inhibition [15]. In addition, in female, the sort of heart rate maintenance could be linked with estrogen. Estrogen is said to be involved in cardio-protection in female [16, 17].

As shown in table 1, there was significant increase in pulse pressure when comparing recumbence with changes in various angles of tilts. The pulse pressure increase gradually at angle 15° tilt ( $8.0 \pm 1.1\%$ ) and peak at 30° tilt ( $18.0 \pm 2.2\%$ ) before decline at angle 45° tilt and

60° tilt ( $11.0 \pm 1.9\%$  and  $6.5 \pm 1.6\%$ ). Generally, the average pulse pressure increased at all positions of tilt when compared to supine. The changes were statistically significant ( $p < 0.05$ ). It is well established that a change from recumbence to the vertical is accompanied by an increase in heart rate and diastolic pressure with little or no increase in systolic pressure [18]. The fall in pulse pressure at angles 45° and 60° is in agreement with findings of Tuchman and Shillingford that "It may well be that the reflexes associated with control of diastolic pressure are more active at lesser degrees of tilt than those responsible for the increase in cardiac rate" [10]. Increase in systolic blood pressure could be due in part to increase in alpha adrenergic activity [9, 15].

There was progressive increase in MAP as the angle of tilt increases. The MAP increased gradually at 15° and markedly at other angles of tilt. When comparing supine to these angles, however, the changes were significant (Table 2). When the vascular bed of the legs are separated from circulation by arterial occlusion, the decrease in MAP during posture change from upright, sitting to supine vary similar to those during posture change without occlusion [19]. This is in central effect of water immersion, where vasodilation in the legs is necessary to prevent MAP from increasing [19]. It has been observed that during posture change from sitting to supine, with a

subsequent stimulation of arterial high and cardiopulmonary low pressure receptors, MAP decreases [20]. During orthostasis which is the maintenance of an upright standing posture, the splanchnic regions accounts for some 30% of the increase in total peripheral resistance, while skin and muscles account for some 40% of the increase in total peripheral resistance [3]. It was expected that constriction in both the vascular beds would be pivotal to inducing an increase in MAP during a moderate anti orthostatic posture change.

### CONCLUSION:

The effects of different angles of head-up tilting on heart rate, pulse pressure and mean arterial blood pressure have been studied on male and female subjects in the 18 to 24 years age group. The results show that at head up tilt position of 15<sup>0</sup>, 30<sup>0</sup>, 45<sup>0</sup> or 60<sup>0</sup> major change of pulse pressure occurs at 30<sup>0</sup> and there is little change on further tilting; heart rate increase with increase in angle of tilt.

The major change of mean arterial pressure occurs at 30<sup>0</sup> and 45<sup>0</sup>.

### REFERENCES:

1. McMicheal J, Sharpey-Schafer EP. Cardiac output in man by direct Fick method: effects of posture, venous pressure changes atropine and adrenaline. *British Heart Journal* 1944; 6: 33.
2. Reeves JJ, Grover RF, Blount SG, Filley FG. Cardiac output response to standing and treadmill walking. *J.appl.Physiol.* 1961; 16: 283.
3. Rowell LN. *Human Cardiovascular control.* Oxford University Press.1993
4. Öberg B. Effects of cardiovascular reflexes on net capillary fluid transfer. *Acta Physiologica Scandinavica.* 1964;62(suppl. 229):1–98
5. Burton AC. *Physiology and biophysics of the circulation.* 2nd Ed. Chicago, Year Book Medical Publishers. 1972.
6. Eckberg DL, Sleight P. *Human baroreflexes in health and disease.* Monograph of the Physiological Society . Oxford:Clarendon Press, 1992.
7. Wieling W, Karemaker JM. Measurement of heart rate and blood pressure. In: Mathias C, Bannister R, eds. *Autonomic Failure.* Oxford, UK: Oxford University Press, 2013; in press.
8. Cooper VL, Hainsworth R. Carotid baroreceptor reflexes in humans during orthostatic stress. *Exp Physiol* 2001;86:677–681
9. Ramsey, M W. Effects of head-up tilt on mean arterial pressure, heart rate and regional cardiac output distribution in aging rats. Doctoral dissertation, Texas A & M University, 2005
10. Tuckman J, Shillingford J. Effect of different degrees of tilt on cardiac output, heart rate and blood pressure in normal man. *Brit. Heart J.* 1966; 28: 32-39.
11. Kennedy JB, Neville AM. *Basic statistical methods for Engineers and Scientists* (2nd ed). Harper and Row, New York.1976; pp. 205 - 212.
12. Gerriten J, Ten Voorde BJ, Dekker JM, Kostense PJ, Bouter LM, Heethaar RM. Baroreflex sensitivity in the elder: influence of age, breathing and spectral methods. *Clin,sci (Lond).* 2000; 99: 371-381.
13. Binanay C, Califf RM, Hasselblad V, O'Connor CM, Shah MR, Sopko G, Stevenson LW, Francis GS, Leier CV, Miller LW; ESCAPE Investigators and ESCAPE Study Coordinators. Evaluation study of congestive heart failure and pulmonary artery catheterization effectiveness: the ESCAPE trial.*JAMA.* 2005;294(13):1625-1633.
14. Sarnoff SJ, Gilmore JP, Brockman SK, Mitchell JH, Linden RJ. Regulation of ventricular contraction by the carotid sinus: its effect on atrial and ventricular dynamics *Circulat Res.* 1960; 8: 1123-1136.



15. Freyschuss U. Cardiovascular adjustment to somatomotor activation. The elicitation of increments in heart rate, aortic pressure and venomotor tone with the initiation of muscle contraction. *Acta Physiol Scand Suppl.* 1970; 342:1-63.
16. Mendelsohn ME, Karas RH. The protective effects of estrogen on the cardiovascular system. *N Engl Journal Med.* 1999; 340: 1801–1811.
17. Weissman A, Lowenstein L, Tal J, Ohel G, Calderon I, Lightman A. Modulation of heart rate variability by estrogen in young women undergoing induction of ovulation. *Eur J Appl Physiol.* 2009;105: 381–386.
18. Hellebrant FA, Franseen EB. Physiological study of the ventricular stance of man. *Physiol Rev.* 1943; 23: 220.
19. Johansen LB, Jensen TV, Pump B, Norsk K. Contribution of abdomen and legs to central blood volume expansion in human during immersion. *J. Appl Physiol.* 1997; 83: 695-699.
20. Pump B, Christensen NJ, Videback R, Werberg J, Hendryksen O, Norst P, Left atrial distention and antiorthostatic decrease in arterial pressure and heart rate in humans. *Amer. J. Physiol,* 1997; 83: 695-699.
21. Hughson RL, Edwards MR, O'Leary DD, Shoemaker JK.. Critical analysis of cerebrovascular autoregulation during reported head up tilt: 2001; 32:2403-2408.
22. Zaidi A, Benitez D, Gaydecki P, Vohra A, Fitzpatrick A. Haemodynamic effects of increasing angle of head up tilt. *Heart.* 2000; 83(2): 181–184.