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Physiological Responses to Dangling and Standing in Healthy Subjects

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ABSTRACT

Boone T, Crawford R, Erlandson A. Physiological Responses to Dangling and Standing in Healthy Subjects. **JEPonline** 2011;14(3):15-25. This study determined the effects of dangling in 11 subjects on oxygen uptake (VO_2) and the central (heart rate, HR; stroke volume, SV; cardiac output, Q) and peripheral (arteriovenous oxygen difference, a-vO_2 diff) components of VO_2 during dangling and standing. Dangling resulted in a significant ($P<0.05$) increase in VO_2, a-vO_2 diff, and DP, and a significant decrease in SV. Standing resulted in significant ($P<0.05$) increase in HR, DP, and SVR, and significant decrease in SV and Q. The transition from supine to dangling to standing resulted in a significant increase in HR, a-vO_2 diff, SVR, and DP, and significant decrease in SV and Q. Compared to supine, dangling increased HR, a-vO_2 diff, and VO_2. The decreased SV was offset by HR, which kept Q constant since SVR was unchanged. Compared to dangling, standing decreased SV and Q. The increased HR was responsible for keeping Q from decreasing further. Compared to the supine measurements, standing produced a greater reduction in SV and a higher HR and a-vO_2 diff than did dangling. When raising an individual from a supine position to assess the orthostatic responses to dangling, an increase in HR of 10 beats·min^{-1} or more is a common response to the decrease in SV that occurs with the immediate downward translocation of blood volume. Systolic blood pressure may increase, remain stable, or may show a small decrease. A decrease in SBP of 15 mm Hg or more and/or a decrease in HR of 10 beats·min^{-1} or more with dangling should be evaluated as abnormal orthostatic responses.

Key Words. Cardiovascular and Metabolic Physiology

INTRODUCTION

Dangling is generally defined as raising a patient from a supine position in bed to a sitting position with the legs hanging over the edge of the bed (35). It is a common nursing intervention used to assess and promote a patient's physiologic tolerance to getting out of the bed (22,28,33). The patient may dangle or more typically, stand, pivot, and sit in a chair. Despite nurses' routine use of dangling (22,28,30,32,33), only three studies have investigated the practice (10,19,25). Kennedy and Crawford (19) examined the effect of dangling on heart rate (HR) in 10 healthy volunteers (7 men and 3 women; mean age of 26 yrs) and in 10 male patients (mean age of 56 yrs) with angina pectoris. They found that HR increased in the healthy subjects after dangling. This finding is in agreement with the report by McDaniel (25) who studied 30 healthy women with a mean age of 41 yrs. Interestingly, though, Kennedy and Crawford (19) reported that HR did not increase in the patients after dangling. This finding was consistent with the findings from a recent study of 24 cardiac rehabilitation patients (8 men and 16 women; mean age of 74 yrs) by Dossa and Owen (10).

Dossa and Owen (10) and Kennedy and Crawford (19) found there was no significant difference in the systolic blood pressure (SBP) measurements obtained with dangling as compared to McDaniel (25) who reported a statistically significant decrease in SBP. The diastolic blood pressure (DBP) responses demonstrated even wider variability with dangling (e.g., unchanged in the cardiac rehabilitation patients (10), significantly increased in healthy subjects but not in the angina pectoris patients (19) and significantly decreased in healthy women subjects (25). This lack of consensus may reflect the limited research on dangling, lack of uniformity in data collection, and the wide variability cardiovascular responses to changing posture (35).

Although the hemodynamic data are considered indicators of orthostatic tolerance (and thus intolerance), the usual measure of the capacity of the body to deliver and utilize oxygen in response to position changes is oxygen uptake (VO_2). VO_2 can be expressed by the Fick principle: VO_2 = cardiac output (Q) x arteriovenous oxygen difference (a-vO_2 diff). The cardiopulmonary system is therefore defined by a central component (Q), which describes the capacity of the heart to function as a pump, and by peripheral factors (a-vO_2 diff), which describe the capacity of the muscles to extract oxygen from the blood.

The physiologic response to dangling requires the integration of cardiac and peripheral components and, therefore, knowledge of HR, stroke volume (SV), and Q is essential to validating the use of dangling. It is of special interest to determine the interrelationship between the central (HR, SV) and peripheral adjustments (systemic vascular resistance; SVR) as mediators of the change in Q during dangling. The purpose of this study was to describe VO_2 and the central (HR, SV, Q) and peripheral (arteriovenous oxygen difference, a-vO_2 diff) components of VO_2 during dangling and subsequently while standing.

METHODS

Subjects

Eleven subjects (5 men and 6 women) volunteered to participate in this study. All subjects were in good health, exercised daily, and had no history of cardiorespiratory disease (Table 1). The Human Research Committee at The College of St. Scholastica approved the study.

Table 1. Physical characteristics of the subjects.

	Women (n = 6)	Men (n = 5)
Age (yr)	23 ± 2	21.5 ± 2
Height (cm)	162 ± 3	175 ± 4
Weight (kg)	63 ± 8	76 ± 6

Procedures

The experimental design was explained to all subjects, and informed consent was obtained. Following a brief familiarization period, each subject was placed in the supine position with the head slightly elevated with a small pillow for 10 min in a quiet, thermally neutral room (70 °F). With one hand placed on the subject's top arm (as a guide only), each subject assumed the sitting position with legs hanging over the side of the bed. Without arm and/or back support, the subjects dangled for 5 min with no movement (i.e., wiggling toes and contracting the calf muscles were not permitted). Following measurements, the subjects moved to the standing position for 5 min. While in the standing position, the subjects were instructed to stand motionless. Subjects were not allowed to engage in active maneuvers such as tiptoeing, shifting of body weight from one foot to the next, or contraction of the thigh and leg muscles to improve orthostatic tolerance.

Oxygen consumption data were averaged across the second 5 min of the supine position to ensure an adequate rest period prior to establishing a resting physiological baseline. All metabolic measurements were taken using a MedGraphics CPX/D analyzer (Medical Graphics Corporation, St. Paul, MN), which was calibrated using medical grade gases and according to the CPX/D Manual. The analyzer computes real-time, breath-by-breath VO_2 and other gas exchange data that were averaged across a 1-min time period. The CO_2 and O_2 gas analyzers feature stability and sensitivity that permit accurate and rapid breath-by-breath analysis. The CO_2 analyzer is an infra-red absorption analyzer that is linear from 0 to 10% CO_2, with an accuracy of ±0.2% at full scale, and a response time less than 100 ms in the sensor. The O_2 analyzer is linear from 0 to 100% with an accuracy of ±0.05% O_2 at full scale, and a response time less than 100 ms at the sensor.

Experienced users made all cardiovascular and metabolic measurements in a temperature and humidity controlled laboratory where subjects were under constant observation. During data collection, one investigator was responsible for the operation of the metabolic analyzer, another was responsible for the CO_2 rebreathing procedure and blood pressure measurements, and the third was responsible for other aspects of data collection. Cardiac output was determined noninvasively during the 10th-min of the supine position and during the 5th-min of dangling and standing using the indirect CO_2 rebreathing (equilibrium) technique. The 5th-min was used to determine Q each period, given that the subjects had rested in each position long enough to estimate a steady-state response.

The Fick Q procedure using CO_2 as a reference gas yields the following formula: Q = CO_2 production ÷ (venous-arterial) CO_2 content; that is, Q = VCO_2 ÷ (C_vCO_2 - C_aCO_2), where Q = cardiac output ($L \cdot min^{-1}$), VCO_2 = CO_2 production ($mL \cdot min^{-1}$), C_vCO_2 = CO_2 content in mixed venous blood ($mL \cdot L^{-1}$), and C_aCO_2 = CO_2 content in arterial blood ($mL \cdot L^{-1}$). The CO_2 rebreathing software determines the partial pressure of the end-tidal pulmonary CO_2 ($P_{ET}CO_2$), converts it to arterial CO_2 (P_aCO_2), and then to arterial CO_2 content (C_aCO_2). The analyzer also determines the partial pressure of CO_2 in the rebreathing (bag) procedure, converts it to mixed venous pulmonary CO_2 (P_vCO_2), and then to mixed venous CO_2 content (C_vCO_2) (13,18,36). The MedGraphics analyzer displayed the CO_2 signal graphically to ensure the $PvCO_2$ equilibrium.

Heart rate data were monitored by bipolar electrocardiogram, using the Physio-Control Lifepack 9 (Physio-Control Corporation, Redmond, WA). Heart rate was recorded from a strip recording that was made during the last 10 sec of each min of each position. The data were averaged across the last 5 min of the supine position and across each 5-min period of dangling and standing. Systolic and diastolic blood pressures were measured by auscultation of the left brachial artery with the upper limb positioned at the side of the subject's body. A standard mercury sphygmomanometer was used during the 9th-min of the supine position and during the 4th-min of dangling and standing. Systolic pressure was determined as the point of appearance of Korotkoff sounds, while the point of disappearance of these sounds was considered to be the diastolic pressure. Mean arterial pressure (MAP) was calculated by adding one-third of the pulse pressure (the difference between SBP and DBP) to the diastolic pressure. Systemic vascular resistance (SVR) was estimated by dividing MAP by Q. Stroke volume was estimated by dividing Q by HR. Arteriovenous oxygen difference was calculated by dividing VO_2 by Q. Double product (DP) was determined by multiplying .01 times HR times SBP.

Instrumentation

The noninvasive CO_2 rebreathing method of determining Q is well established as a valid and reproducible scientific procedure (12,13,17,18,20,23,36). Franciosa (11) reported a high correlation coefficient (r = 0.95) between the standard invasive (Fick) method and the equilibrium rebreathing method in measuring Q in 14 patients with acute myocardial infarction. Similarly, when evaluating patients in an intensive care unit, Davis and associates (8) reported a correlation of r = 0.94 between the CO_2 rebreathing method and the direct Fick. Muiesan and co-workers (27) compared the direct Fick technique with the CO_2 rebreathing (equilibrium) method in patients at rest in the supine position. They, too, reported a high correlation (r = 0.94) between the two methods.

The reproducibility studies that have examined the CO_2 rebreathing (equilibrium) method have also reported high test-retest correlations for Q. Franciosa et al. (12) and Heigenhausen and Faulkner (16) reported a correlation coefficient of .96 and .91, respectively. Similarly, Boone et al. (5) reported intraclass correlation coefficients of .79 and .93 for Q responses at rest and during exercise. The lack of significant differences in Q at rest and in the related CO_2 rebreathing values (VCO_2, P_vCO_2, and P_aCO_2) during 4 separate tests indicates good reproducibility for estimating Q.

Statistical analysis

Data analysis was performed with the Statistics with Finesse software (4). The level of significance for all tests was set at $P \leq 0.05$. A repeated measures ANOVA was performed to compare the

supine, dangling, and standing data. When significant F-ratios were obtained, the post-hoc Scheffe comparisons for treatments were used to identify significant differences between paired group means.

RESULTS

Results are given as means ± SD, percent increase, decrease, or no change in the subjects' responses to posture change and mean percentage change of dangling and standing responses from the supine values. From supine to dangling, there were statistically significant ($P \leq 0.05$) increases in VO_2, HR, a-vO_2 diff, and DP, and a significant decrease in SV. From dangling to standing, there were statistically significant increases in HR, DP, and SVR, and significant decreases in SV and Q. From supine to standing, there were significant increases in HR, a-vO_2 diff, SVR, and DP, and significant decreases in SV and Q.

DISCUSSION

The SBP responses are consistent with previous reports by Dossa and Owens (10) and Kennedy and Crawford (19) but contrary to the results of McDaniel (25) where SBP decreased with dangling. We found no significant effect of dangling on DBP, which agrees with the results of Dossa and Owens (10) but is contrary to the increase reported by Kennedy and Crawford (19) and the decrease reported by McDaniel (25). Our HR results are in agreement with the response of other healthy subjects, as reported by Kennedy and Crawford (19) and McDaniel (25) but disagree with the reported no change in angina pectoris (19) and cardiac rehabilitation patients (10). None of the three reported studies (10,19,25) on dangling agrees completely either with each other or the present study. We conclude that the hemodynamic responses are variable. Dangling for 5 min was found to have a significant effect on VO_2 and the central and peripheral components of VO_2. We showed that the increase in VO_2 was due to a significant increase in HR and a-vO_2 diff. Stroke volume was significantly decreased, which left Q unchanged (Table 2). Hence, dangling for 5 min elicited a slightly higher metabolic response than found in the supine position, and the percentage increase in VO_2 was met by both central and peripheral circulatory adjustments.

By rearranging the Fick equation, the percentage increase in VO_2 from supine to dangling depends on the percentage change in HR, SV, and a-vO_2 diff. The decrease in SV (due to effects of gravity on the circulatory system) was offset by the compensatory increase in sympathetic drive that resulted in the increase in HR in all 11 subjects (Table 2) during dangling. The increased HR response was also accompanied by an increase in the muscles' extraction of oxygen (a-vO_2 diff), which was apparently a function of the slightly elevated muscle tension in the 11 subjects to keep the upper body in the dangling position. This is not surprising since the observed increase in VO_2 with dangling resulted from the widening of the a-vO_2 diff. This finding suggests that the non-significant rise in Q (that would otherwise accompany an increase in VO_2) resulted in, perhaps, an even greater need for the periphery to increase O_2 extraction to meet the subjects' metabolic needs. The implication is that the VO_2 response during dangling appears to be facilitated by O_2 extraction but somewhat limited by O_2 transport.

Table 2. Physiological responses (mean ± SD) to three body positions (n = 11).

	Supine (A)	Dangling (B)	Standing (C)	*F*-ratio & Prob
VO_2 ($mL \cdot min^{-1}$)	339 ± 47 AB** (17%)	390 ± 39	358 ± 64	4.1332 0.0315*
HR ($beats \cdot min^{-1}$)	68 ± 14 AB** (12%) AC** (27%)	77 ± 13	86 ± 12	27.813 0.0001*
SV (mL)	72 ± 16 AB** (-18%) AC** (-38%)	59 ± 17 BC** (-24%)	45 ± 14	23.071 0.0001*
Q ($L \cdot min^{-1}$)	4.89 ± 1 AC** (-21%)	4.54 ± .98 BC** (-14%)	3.87 ± .89	8.0135 0.0028*
a-vO_2 diff ($mL \cdot 100\ ml^{-1}$)	6.95 ± 1 AB** (24%) AC** (33%)	8.60 ± 2	9.25 ± 1.7	15.362 0.0001*
SVR ($mm\ Hg \cdot L^{-1} \cdot min^{-1}$)	20 ± 4 AC** (28%)	22 ± 6 BC** (16%)	25 ± 5	6.3423 0.0074*
SBP (mm Hg)	121 ± 5	124 ± 8	121 ± 9	1.4122 0.2622
DBP (mm Hg)	86 ± 8	87 ± 9	87 ± 10	0.1242 0.8932
MAP (mm Hg)	97 ± 6	99 ± 8	98 ± 10	0.2154 0.8034
DP	83 ± 19 AB** (17%) AC** (26%)	97 ± 21 BC** (13%)	109 ± 20	26.645 0.0001*
MVO_2 ($ml \cdot 100\ gm\ LV \cdot min^{-1}$)	5.45 ± 3 AB** (35%) AC** (63%)	7.36 ± 3 BC** (21%)	8.91 ± 3	22.887 0.0001*

Given the anticipated downward fluid shifts with dangling, the orthostatic changes were considerably milder (as expected) with dangling than previously reported with standing (1) and when compared to the standing data in the present study. In part, this result occurred because there is less downward translocation of blood with dangling versus standing since the thighs and buttocks remain horizontal to the floor. Therefore, given the reduced blood shift, there was a reduced need for the body to compensate for the "functional hemorrhage." The compensatory increase in HR, in spite of the decrease in SV, was sufficient to maintain Q and arterial pressure. Thus, at no time during dangling was a critical level reached at which circulation was not adequately controlled by the subjects' auto-regulation. Circulation remained stable and, therefore, neither the presyncopal manifestations (such as nausea, sweating, dizziness, and pallor) (1,7,9,14,31,34) nor the common manifestation of orthostatic hypotension (35) were observed.

Although myocardial O_2 consumption (MVO_2) was not measured, the degree to which dangling increased MVO_2 may be estimated via the product of SBP and HR, where $MVO_2 = 0.14$ X (HR x SBP ÷ 100) - 6.3 (21). This index of relative cardiac work, termed the double product (DP), is highly related to directly measured MVO_2 and coronary blood flow (24). This observation highlights the fact that the increase in HR, regardless of the no change in SBP, resulted in an increase in MVO_2. That is, the increased HR maintained Q, but also increased the work of the heart (MVO_2). This suggests that the subjects' ability to adjustment to an increase in myocardial oxygen demand is important demonstrating normal postural responses.

With the transition from dangling to standing, which resulted in the expected downward redistribution of blood, Q decreased in response to the significant decrease in SV. Despite these responses, there was no significant difference in the blood pressure measurements between dangling and standing or between supine and standing. Therefore, it appears that the carotid, aortic, and cardiopulmonary mechanoreceptors acted together to trigger the appropriate compensatory responses to maintain arterial blood pressure and tissue perfusion (2,3,6,29,35). The physiologic responses to standing included, in particular, the significant increases in HR and SVR. The increase in HR from dangling to standing approximated the increase that occurred from supine to dangling (i.e., 10 beats $\cdot$ min^{-1}). Stroke volume was significantly reduced while standing, but was compensated by the previously elevated a-vO_2 diff that occurred with dangling. Both responses kept VO_2 constant and thus, standing after dangling was not associated with an increase in energy expenditure (Table 3).

The 21% decrease in Q and the 38% decrease in SV while standing (vs. supine) compare favorably with the standard report of 20% reduction in Q and 20% to 50% reduction in SV, respectively (2,29). The 27% increase in HR and the 33% increase in a-vO_2 diff with standing (vs. supine) kept the subjects' energy expenditure constant. Systolic pressure was identical for standing and supine, given the 28% increase in SVR. Double product was significantly increased (26%) but, again, due to the increased HR response.

CONCLUSIONS

A wide variability in human response to dangling is recognized (26). Within this variability, normal postural responses are increased HR to offset reduced SV and maintained Q, and an adequate peripheral vasoconstrictor response to maintain blood pressure. As Winslow et al. (35) pointed out,

"Research is needed to validate the use of dangling in clinical practice, to test different dangling methods, to determine normal and abnormal responses, to predict patients likely to have orthostatic intolerance, and to test methods to promote orthostatic tolerance." It is clear how a small sample of subjects responded to dangling but, in general, it highlights the need for more work in this area.

We found that the cardiovascular responses to dangling (from the supine position) are similar to but milder than standing. During dangling, as was the case with standing, there was a significant reliance on both central (HR) and peripheral components (a-vO_2 diff) to counter the decrease in SV and to meet the increased need for oxygen (VO_2), respectively. When compared to supine measurements, standing produced higher HR and a-vO_2 diff responses than did dangling. Also, the increase in SVR during the transition from dangling to standing indicates that constriction of the resistance vessels played a vital role in the subjects' arterial pressure response. Although the statistically significant increase in HR is not the primary cardiovascular adjustment during standing, it is clinically significant in maintaining the Q response.

These findings may be helpful in understanding the physiological responses to dangling. For example, if the patient's HR does not increase during dangling, the nurse may assume a more cautious role with the patient. Why, because, if HR does not increase, then, Q may be too low to meet the blood flow requirements of the central nervous system. The resulting outcome may be lightheadedness, dizziness, or other symptoms of orthostatic intolerance. Similarly, if one accepts the physiologic premise that the peripheral vasoconstriction that occurs with dangling (or standing) is important to maintaining arterial pressure, the SBP response should be a relatively stable response. This conclusion is easy to understand when the effect of peripheral vasodilation on preload and thus Q is considered.

RECOMMENDATIONS

Eloquent suggestions for future research have been presented in two *Heart & Lung* articles. The first was published in 1986 by Moore and Newton (26) and the second by Winslow et al. (35) in 1995. Their analysis of the topic demonstrates research is needed to validate and understand the physiology of dangling. Suggestions for future research include the following: (a) Determine the physiologic responses to standing with and without prior dangling; (b) Compare subjects' physiologic responses to 1-, 3-, 5-, and 10-min dangling periods (35); (c) Test the physiologic effects of variation in orthostatic tolerance during the morning versus the afternoon (26); and (d) By using bedside invasive hemodynamic monitoring technologies, continuous Q responses and other information on the patient's state should be obtained to assist the clinician with improving patient outcomes (15).

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