Influence of head-down and lateral decubitus neck flexion on heart rate variability

C. MATTHEW LEE, ROBERT H. WOOD, AND MICHAEL A. WELSZICH
Department of Kinesiology, Louisiana State University, Baton Rouge, Louisiana 70803

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Lee, C. Matthew, Robert H. Wood, and Michael A. Welsch. Influence of head-down and lateral decubitus neck flexion on heart rate variability. J Appl Physiol 90: 127–132, 2001.—The purpose of this study was to examine the response of heart rate variability (HRV), a noninvasive index of autonomic control, to head-down neck flexion (HDNF), which engages both otoliths and neck muscle afferents, and to lateral decubitus neck flexion (LNF), in which neck afferents are activated, whereas otolith afferent input is not. HRV and forearm blood flow were evaluated in participants lying prone, during HDNF, lying in the lateral decubitus position, and during LNF. Compared with the prone position, HDNF resulted in lower high-frequency (46.9 ± 7.1 vs. 62.3 ± 6.2) and higher low-frequency (53.1 ± 7.1 vs. 37.7 ± 6.2) power, expressed as normalized units, along with higher low-frequency-to-high-frequency ratio (1.65 ± 0.3 vs. 0.78 ± 0.2), whereas LNF resulted in no alterations in HRV indexes. Furthermore, there were no significant differences in forearm blood flow or vascular resistance among any of the positions. Our data suggest that otolith organs influence autonomic modulation of the heart, supporting previous studies reporting that HDNF elicits increased sympathetic outflow. These data further suggest that HDNF results in a parasympathetic withdrawal from the heart in addition to sympathetic activation.

vestibular; otoliths; muscle afferents

VESTIBULAR ORGANS RESPOND to linear and angular accelerations of the head and provide signals to neurons in the brain stem that influence autonomic activity (20). Accordingly, animal studies have long provided evidence for a link between the vestibular system and autonomic nervous system (2, 10, 19, 20). For example, electrical stimulation of the vestibular nerve has been reported to alter autonomic outflow to a wide variety of sympathetic nerves (2, 10, 19). More recently, human research has provided evidence that head-down neck flexion (HDNF), which likely stimulates the otoliths of the vestibular apparatus, as well as neck muscle afferents and perhaps other afferents, results in augmented sympathetic outflow (4, 8, 11, 14, 15, 17). Essandoh et al. (4) reported that HDNF elicits increases in calf and forearm vascular resistance (FVR), and Shortt and Ray (17) further report elevated muscle sympathetic nerve activity in this position. Interestingly, this presumed vestibulosympathetic reflex is absent during neck flexion in the lateral decubitus (L) position (LNF) and during yaw head rotation (14, 16). Thus it appears that this reflex is mediated primarily by the otolith organs with little contribution from neck muscle afferents (4, 11, 14, 16). Whereas the HDNF model has been used to investigate sympathetic activation by the otoliths, parasympathetic-mediated activity during neck flexion has not yet been reported.

Heart rate variability (HRV) has gained recent popularity as a noninvasive tool to estimate autonomic control. Time domain measures [e.g., standard deviation of normal R-wave-to-R-wave (R-R) intervals (SDNN)] are believed to reflect vagal modulation of the heart, and spectral analysis of beat-to-beat variations in the R-R interval provides some information about sympathovagal balance regarding the neural control of the cardiovascular system (12, 18). Within the power spectra of the R-R interval, the majority of the oscillations occur as peaks within the range of 0.04–0.4 Hz. High-frequency power (HF) (0.15–0.4 Hz) seems to reflect vagal activity to the heart, whereas low-frequency power (LF) (0.04–0.15 Hz) represents vasomotor activity and has been reported to reflect both sympathetic and parasympathetic modulation (13). Therefore, this technique allows for an estimation of the relative contribution of each component of the autonomic nervous system.

The purpose of this investigation was to examine HRV and vascular responses to HDNF and LNF. HRV and forearm blood flow (FBF) were evaluated while the subjects were lying prone, during HDNF in which both neck afferents and otoliths are reportedly active, while the subjects were lying in the L position, and during LNF in which neck afferents are activated, whereas otolith afferent input is not. The specific aim of this study was to determine whether HRV can detect changes in autonomic activity elicited by HDNF and LNF. We hypothesized that, consistent with previous reports, vasoreactivity would be apparent during HDNF but not with LNF. Similarly, we hypothesized that measures of HRV would be consistent with heightened sympathovagal balance during HDNF but not during LNF.
METHODS

Participants

Twelve healthy college-aged participants (3 men and 9 women), aged 19–23 yr, responded to an invitation to participate in this study and completed all aspects of the protocol. All were free of any documented cardiovascular, cochlear, vestibular, or neurological diseases or conditions. Written informed consent was obtained from participants, and all procedures were approved by the University Institutional Review Board of the host site. All participants were instructed not to take any medications, including over-the-counter drugs, caffeine, and alcohol 12 h before testing.

Experimental Design

On arriving at the laboratory, participants provided informed consent and were assessed for HRV and FBF in the prone, HDNF, L, and LNF positions, as described below.

Prone. Each participant was instructed to lie prone on a table with the left arm extended at 180° relative to the anatomic position and the right arm slightly elevated above the level of the heart (15°) to allow for sufficient venous drainage. A mobile support and foam pads were used to support the forehead and arms and to keep the dorsal aspect of the head aligned with the back. The participant remained in this position for 15 min, with measurements taken during the last 5 min.

HDNF. While the subject was lying prone, the mobile support was moved from under the participant’s head and the arms still supported. The transition from prone to HDNF lasted ≈5 s and was not included in the analysis. The participant remained in the HDNF position for 5 min.

L. Participants were positioned lying on their left side with the left arm placed by their side and slightly anterior with the left wrist placed at the level of the heart. The right arm was placed by their side and slightly anterior with the right wrist placed at the level of the heart. The dorsal aspect of the head was aligned with the back, and the mobile support was placed under the head to keep it parallel to the table. The participant remained in this position for 10 min, with measurements taken during the last 5 min.

LNF. The participants continued to lie on their left side as described above. The neck was then passively moved by the investigator to a flexed position (chin to chest) with the head still supported. The transition from L to LNF lasted ≈5 s and was not included in the analysis. The participant remained in this position for 5 min.

Hemodynamic and Respiratory Measurements

A Hokanson EC-5R plethysmography system (Bellevue, WA) was used to measure FBF. Before each experiment, a venous-occlusion cuff was positioned around the participant’s upper right arm, and a pedicuficuff was placed around the participant’s right wrist. A mercury-in-Silastic strain gauge was placed around the forearm ~10 cm distal to the olecranon process. FBF was measured during the second and fourth minute of each experimental position, and the average is reported. One minute before the measurement of FBF, the wrist cuff was inflated to 240 mmHg to occlude hand blood flow and thus isolate the forearm. FBF measures were acquired immediately on inflation of the upper arm cuff to 50 mmHg and were calculated as described by Hokanson et al. (7). The FBF values are reported as milliliters per 100 milliliters of tissue per minute.

Continuous measurements of arterial blood pressure were made throughout the entire experimental protocol via a Colin 7000 (San Antonio, TX) noninvasive arterial tonometry device. This device consisted of a sensor positioned over the left radial artery at the wrist and an occlusion cuff positioned around the upper left arm. The Colin 7000 was interfaced with a Biopac MP100 data-acquisition system (Santa Barbara, CA) using the AecKnowledge ACK100 software program (Santa Barbara, CA) and was calibrated before each experiment. FVR was calculated by dividing the mean arterial pressure (MAP) at the second and fourth minute of each position by the FBF and for simplicity is reported in units. Additionally, a Cosmed K4b2 pulmonary gas exchange system (Rome, Italy) was used to monitor respiratory frequency (RF) and tidal volume (VT) continuously during the entire experiment.

Evaluation of HRV

HRV was evaluated in accordance with guidelines set forth by the Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology (18). The Biopac MP100 data-acquisition system and AecKnowledge ACK100 software program were used to collect a continuous electrocardiogram (ECG) at a sampling rate of 200 Hz during each of the four positions. The ECG was visually inspected for noninus beats and plotted as a tachogram of the heart period. The waveform was then resampled at 4.0 Hz and evaluated for the SDNN. The spectral power density of the waveform was derived via a 1,024-point linear fast Fourier transformation using a Hamming window. The resultant power density spectra was then analyzed for total power (TP) (0.00–0.40 Hz), LF (0.04–0.15 Hz), and HF (0.15–0.40 Hz). To estimate the relative contribution of these power bands to TP, LF, and HF were normalized (LFnu, HFnu, where nu is normalized unit) by dividing each by TP minus very-low-frequency power (VLF) (0.00–0.04 Hz) and multiplying this value by 100 (18)

\[
HF_{nu} = \frac{HF}{(TP - VLF)} \times 100
\]

\[
LF_{nu} = \frac{LF}{(TP - VLF)} \times 100
\]

Furthermore, the ratio of LF to HF (LF/HF) is reported as a measure of autonomic balance.

Statistical Analysis

All statistical analyses were performed using the SAS statistical package (Cary, NC). The raw distributions of TP, LF, and HF were skewed toward large values and were, therefore, log transformed (ln). However, LFnu, HFnu, and LF/HF did not violate assumptions of normality and, therefore, did not require transformation. Repeated-measures ANOVA was used to compare each variable obtained during the four different positions. Furthermore, preplanned contrasts were used to evaluate any differences between prone and HDNF and between L and LNF. Additionally, repeated-measures ANOVA was used to compare respiratory measures within and among positions. A significance level of 0.05 was used for all tests. Data are expressed as means ± SE.

RESULTS

None of the participants reported any feelings of discomfort during any of the positions, and analysis of the ECGs revealed no cardiac ectopy in any of the
DISCUSSION

Our results indicate that HRV is significantly altered by HDNF, but not LNF, suggesting an influence of the otolith organs on autonomic modulation of the heart. We observed increases in $L_{nu}$ and LF/HF and decreases in HF, $HF_{nu}$ and SDNN during HDNF but not LNF. These data also suggest that HDNF results in a parasympathetic withdrawal in addition to sympathetic activation.

The link between the vestibular apparatus and autonomic outflow in animals is well established (2, 10, 19, 20). Cobbold et al. (2) demonstrated that electrical stimulation of the vestibular nerve in anesthetized cats resulted in increased vagal efferent outflow and sympathetic outflow to the abdominal sympathetic chain and cardiac sympathetic nerves. Yates et al. (19) further provided evidence for a vagalerespiratory reflex in decerebrate cats. By electrically stimulating the vestibular nerve, they reported elevated efferent outflow to the phrenic, abdominal, and intercostal nerves.

Recent studies in humans have used HDNF to examine the influence of vestibular stimulation on autonomic outflow (4, 8, 11, 14, 15, 17). Several investigators have reported augmented sympathetic outflow during HDNF. Essandoh et al. (4) initially demonstrated that HDNF markedly reduces limb blood flow and concluded that this movement increases sympathetic noradrenergic outflow, whereas, more recently, Ray and Hume (14) and Shortt and Ray (17) reported increases in muscle sympathetic nerve activity during HDNF. In an attempt to gain a greater understanding of this autonomic reflex, we examined the influence of HDNF and LNF on HRV, which is sensitive to changes participants. HRV and hemodynamic data can be seen in Tables 1 and 2, respectively. The results of the ANOVA and contrasts indicate that the mean R-R interval was shorter during both HDNF and LNF relative to prone and L, respectively. With regard to HRV, the ANOVA model was significant for all variables with the exception of TP and LF. Additionally, the contrasts revealed higher $LF_{nu}$ and LF/HF, and lower $HF_{nu}$ and HF during HDNF relative to the prone position (Figs. 1 and 2). However, the contrasts did not reveal any significant differences in HRV indexes between L and LNF.

There were no significant differences in FBF, FVR, or MAP during any of the positions. Additionally, RF and $V_r$ remained stable throughout the experimental protocol, and ANOVA did not reveal any differences within or among positions (Figs. 3 and 4).

Table 1. Heart rate variability during the 4 positions

<table>
<thead>
<tr>
<th></th>
<th>Prone</th>
<th>HDNF</th>
<th>L</th>
<th>LNF</th>
<th>$P$ Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean R-R interval, ms</td>
<td>953.0 ± 29</td>
<td>892.4 ± 34*</td>
<td>997.7 ± 31</td>
<td>951.2 ± 28†</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>SDNN, ms</td>
<td>78.7 ± 8</td>
<td>54.7 ± 5*</td>
<td>67.2 ± 7</td>
<td>70.7 ± 9</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>$ln$ HF, ms²</td>
<td>6.4 ± 0.3</td>
<td>5.4 ± 0.3*</td>
<td>6.4 ± 0.3</td>
<td>6.3 ± 0.3</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>$ln$ LF, ms²</td>
<td>5.7 ± 0.2</td>
<td>5.4 ± 0.2</td>
<td>5.8 ± 0.2</td>
<td>5.9 ± 0.2</td>
<td>NS</td>
</tr>
<tr>
<td>$ln$ TP, ms²</td>
<td>7.4 ± 0.2</td>
<td>6.9 ± 0.1</td>
<td>7.3 ± 0.2</td>
<td>7.2 ± 0.3</td>
<td>NS</td>
</tr>
<tr>
<td>HF, ms²</td>
<td>985.8 ± 320</td>
<td>368.6 ± 112*</td>
<td>886.8 ± 209</td>
<td>899.7 ± 254</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>LF, ms²</td>
<td>352.5 ± 61</td>
<td>286.9 ± 62</td>
<td>416.4 ± 77</td>
<td>525.0 ± 122</td>
<td>NS</td>
</tr>
<tr>
<td>TP, ms²</td>
<td>2,185.2 ± 531</td>
<td>1,118.5 ± 174</td>
<td>1,802.8 ± 339</td>
<td>2,211.9 ± 643</td>
<td>NS</td>
</tr>
<tr>
<td>$HF_{nu}$</td>
<td>62.3 ± 6.2</td>
<td>46.9 ± 7.1*</td>
<td>61.7 ± 5.8</td>
<td>58.6 ± 4.3</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>$LF_{nu}$</td>
<td>37.7 ± 6.2</td>
<td>53.1 ± 7.1*</td>
<td>38.3 ± 5.8</td>
<td>41.4 ± 4.3</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>LF/HF</td>
<td>0.78 ± 0.2</td>
<td>1.65 ± 0.3*</td>
<td>0.84 ± 0.2</td>
<td>0.85 ± 0.2</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

Values are means ± SE. R-R, R-wave to R-wave; SDNN, SD of normal R-R intervals; $ln$, natural log; HF, high-frequency power; LF, low-frequency power; TP, total power; nu, normalized units; LF/HF, ratio of LF to HF; HDNF, head-down neck flexion; L, lateral decubitus; LNF, lateral decubitus neck flexion; NS, not statistically significant. *$P < 0.05$ vs. prone; †$P < 0.05$ vs. L.

Table 2. Hemodynamic responses during the 4 positions

<table>
<thead>
<tr>
<th></th>
<th>Prone</th>
<th>HDNF</th>
<th>L</th>
<th>LNF</th>
</tr>
</thead>
<tbody>
<tr>
<td>MAP, mmHg</td>
<td>78 ± 1</td>
<td>75 ± 3</td>
<td>77 ± 3</td>
<td>81 ± 3</td>
</tr>
<tr>
<td>FBF, ml·100 ml tissue⁻¹·min⁻¹</td>
<td>3.2 ± 0.1</td>
<td>3.0 ± 0.2</td>
<td>3.3 ± 0.2</td>
<td>3.2 ± 0.2</td>
</tr>
<tr>
<td>FVR, units</td>
<td>24.6 ± 0.7</td>
<td>26.5 ± 2.8</td>
<td>23.4 ± 1.4</td>
<td>27.2 ± 2.4</td>
</tr>
</tbody>
</table>

Values are means ± SE. MAP, mean arterial pressure; FBF, forearm blood flow; FVR, forearm vascular resistance.
in parasympathetic and, to a lesser extent, sympathetic modulation of the heart. Power spectral analysis of HRV represents a noninvasive index of the autonomic modulation of the heart (9) and thus may allow for a better comprehension of the autonomic-mediated changes in cardiac function during head movements. Moreover, the expression of HRV in normalized units minimizes the effect of TP changes on HF and LF, resulting in a more clear representation of autonomic balance (18).

Our data indicate that HDNF results in an increased sympathovagal balance, identified by increases in LF$_{nu}$ and LF/HF and decreases in HF$_{nu}$. These findings are consistent with the hypothesis that HDNF elicits increases in sympathetic nervous system activity (4, 8, 11, 14, 15, 17). Furthermore, because SDNN and HF power are especially sensitive to vagal modulation of the heart, we provide evidence that HDNF may further elicit a parasympathetic withdrawal. This finding has not been previously reported in the literature and possibly suggests that the sympathetic branch of the autonomic nervous system is not the sole component influenced by HDNF. We did not find any significant differences in HRV between L and LNF, suggesting that LNF did not alter cardiac autonomic outflow. Normand et al. (11) postulated that, whereas HDNF stimulates both the otolithic organs and neck mechanoreceptors, LNF primarily results in neck mechanoreceptor activation. Subsequently, they reported that HDNF reduced limb blood flow, whereas LNF had little effect, and hypothesized that otoliths and neck receptors have different effects on mechanisms controlling peripheral blood flow. Similarly, whereas investigators have reported increased sympathetic nerve activity to the muscle during HDNF (14, 17), Ray and Hume (14) reported no effect of LNF on this measure and concluded that this reflex is primarily mediated by the otolith organs. Therefore, our HRV data during L and LNF support these studies and further suggest that the otolith organs play a significant role in modulating cardiac autonomic activity.

Interestingly, there was a small, but significant, decrease in R-R interval from L to LNF. Whereas the mean values for HF$_{nu}$ and LF$_{nu}$ during these positions changed in a direction consistent with increased sympathovagal balance, these changes did not reach statistical significance. Therefore, it cannot be concluded that autonomic balance shifted between L and LNF. This small change in R-R interval without a significant change in spectral parameters may be due to variations in heart rate that occur in the very-low-frequency band. Additionally, one cannot rule out the possibility that there may be a lack of sensitivity of HRV parameters for detecting very small changes in autonomic balance.

Data from the present investigation fail to reveal significant alterations in FBF or FVR during neck flexion (either HDNF or LNF). This finding appears to stand in contrast to those of Essandoh et al. (4), who reported a reduction in FBF during HDNF. However, close inspection of the data of Essandoh et al. reveals

![Fig. 2. Effect of the 4 positions on ratio of LF to HF. *Significant difference vs. prone position, $P < 0.01$.](http://jap.physiology.org/)
such changes at 30 s into HDNF but that, over the ensuing 3 min, FBF gradually returned toward baseline values. Furthermore, the observed value for FBF in the present investigation (3.0 ml·100 ml tissue⁻¹·min⁻¹) is comparable to that observed at 2 min by Essandoh et al. (2.9 ml·100 ml tissue⁻¹·min⁻¹).

Lastly, in the present investigation, great care was taken to ensure that the participants’ heads were passively placed in the flexed position. In contrast, it appears as though the participants in the investigation of Essandoh et al. may have lowered their own heads by maximally flexing their necks, which could have evoked changes in FBF mediated by central command.

In our investigation, it is not likely that the alterations in HRV are the result of the stimulation of neck mechanoreceptors. If this were the case, we would have expected to see changes in HRV during LNF in which mechanoreceptors are more likely to influence autonomic activity than are the otolith organs (14, 17). Bolton et al. (1) reported that electrical stimulation of neck muscle afferents in cats resulted in alterations in sympathetic outflow to sympathetic (splanchnic) and respiratory (hypoglossal and abdominal) nerves. Fujimoto et al. (5) further reported a reduced arterial pressure response to passive 90° neck rotation in humans. Additionally, De Meersman et al. (3) reported significant modulating effects of mechanoreceptor stimulation on sympathovagal balance during passive movement of the legs. Although these studies suggest a significant role of mechanoreceptors in altering autonomic outflow, our data are in agreement with other studies using the LNF model that have demonstrated a minimal effect of the mechanoreceptors in eliciting autonomic changes (11, 14). Although there is no sound explanation for the different results obtained by LNF and other methodologies, it may be possible that different techniques result in differential activation of mechanoreceptors.

Whereas the findings of the present investigation support otolithic rather than mechanoreceptor-mediated changes in autonomic modulation of the heart, it is also important to rule out the influence of other mechanisms, such as baroreflexes, that are known to alter autonomic activity. Shortt and Ray (17) found no alterations in central blood volume, estimated by thoracic impedance, during HDNF, which would be necessary for cardiopulmonary receptors to be activated. Although spectral power may be influenced by a change in blood pressure due to arterial baroreceptor-mediated sympathetic outflow to the heart (9), in our study MAP was not significantly different during any of the positions for which we reported a decreased mean R-R interval (reflecting an increased heart rate) from prone to HDNF and from L to LNF. Although these factors appear to rule out the role of the arterial baroreflex, the time course of our measures may not have been rapid enough to detect alterations in baroreceptor activity. Therefore, we cannot completely rule out a role of the arterial baroreceptors in mediating this reflex.

There are several other potential limitations to the findings of the present investigation. These include the possible influences of central command, visual perturbations, respiratory pattern, and nonspecific pressure receptors in the head on autonomic activity. As to the former, the effect of central command on autonomic outflow was minimized by having the investigator passively move the subject’s head between positions. It may be speculated that changes in visual inputs may have, in part, influenced our data. However, Shortt and Ray (17) previously reported that blindfolding subjects resulted in no noticeable differences in the autonomic response to HDNF. Therefore, we do not believe that the subject’s inverted field of vision played a significant role in mediating these changes in this study. Although it has been documented that respiratory factors influence HRV (6), neither RF nor VT was altered by any of the positions, and both were consistently stable within each position. Therefore, our data suggest that respiration did not play a role in mediating the changes in HRV in this study. One last limitation of this study is that we were unable to account for the possible influence of nonspecific pressure receptors in the head, as cerebral pressure was not measured. However, Hume and Ray (8) provided evidence against the role of such

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Fig. 4. Tidal volume (VT) during prone and HDNF (A) and L and LNF (B) positions.
receptors in mediating autonomic alterations during HDNF.

In conclusion, we found that HDNF significantly alters HRV. These findings suggest that the otolith organs play a significant role in mediating autonomic outflow to the heart. Additionally, our data suggest that a parasympathetic withdrawal accompanies the sympathetic activation elicited by HDNF.

REFERENCES