

## Monitoring Changes in Photoplethysmography During Lower Body Negative Pressure Induced Hypovolemia: Differences by Site and Sex

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**Introduction:** Lower body negative pressure (LBNP) is used as a model for induced hypovolemia by sequestration of blood in the lower extremities.<sup>1</sup> The photoplethysmography (PPG) waveform, known for its use in monitoring arterial oxygen saturation, can also be used as a non-invasive tool to measure blood volume changes.<sup>2</sup> The area under the curve (peak area (PA)) in the arterial waveform can be used to calculate stroke volume (SV).

In states of hypovolemia, peripheral vasoconstriction has been shown to interfere with peripheral blood flow monitoring. When compared to the finger, the nasal alar site is more immune to sympathetic vasoconstriction as it is supplied by the internal and external carotid arteries.<sup>3</sup> In comparison to male patients, female patients show a lower tolerance to hypovolemia due to more rapid reduction in compensatory mechanisms to maintain arterial perfusion.<sup>4</sup>

The purpose of this study was to monitor changes in PA and SV by pulse oximetry site (nasal alar (Xhale) vs. finger (Nellcor)) and by sex during progressive LBNP induced hypovolemia.

**Methods:** 30 healthy subjects (15 male, 15 female), aged 21-43 years, underwent progressive LBNP protocol (LBNP -15, -30, -45, -60mmHg). Subjects who remained asymptomatic at LBNP -60 mmHg were labeled as high tolerance (HT), while subjects who became symptomatic before LBNP -60 mmHg were labeled as low tolerance (LT).

Participants were monitored with EKG, continuous non-invasive arterial pressure (CNAP) to measure MAP and non-invasive cardiac output monitoring (NICOM) to measure SV and CO. Nasal alar (Philips-Xhale Assurance Nasal Alar SpO<sub>2</sub>) and finger (Nellcor) pulse oximeter probes were utilized to measure PPG waveforms.

LabChart (ADInstruments) was used for data collection and waveform analysis of the PPG waveform. Friedman ANOVA and Wilcoxon tests were conducted utilizing SPSS. P-value <0.005 was considered significant (Bonferroni adjustment).

**Results:** During progressive LBNP, MAP was maintained while there was a significant decline in SV (i.e. >~15%) in both HT and LT patients.

In early phases of LBNP, there was a significant drop in finger PPG PA, which is attributed to an increase in sympathetic tone and vasoconstriction in the periphery, as indicated by an increase in low frequency of heart rate variability. Nasal alar PPG demonstrated a strong correlation with changes in SV in both HT (r=0.99) and LT (r=0.94) groups.

At LBNP -30mmHg in the LT group, there was a decline in SV of 11.4% and 21.5% and a 36% and 27% reduction in nasal alar PPG PA in both female and male subjects, respectively. In the LT group, female patient showed rapid decline in nasal alar PPG PA (13%, 36%, 54%, and 65%) more than male patients (12%, 27%, 48%, and 41%) during progressive LBNP protocol.

**Conclusions:** MAP is maintained with progressive LBNP, indicating that this is a late and non-specific sign of hypovolemia.

The rapid decline in nasal alar PPG PA in females compared to males during LBNP-induced hypovolemia confirms that female patients decompensate more quickly than male patients.

The decline in nasal alar PPG PA was comparable to SV reduction in both sexes, indicating that PA can be used as a marker on arterial waveform monitors to track early changes in blood volume. Thus, nasal alar PPG waveform is a more useful clinical tool to assess early central hypovolemia compared to the finger site in both female and male patients.

**References:**

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Figure:

**Figure 1(a-d) – a) b) Percent change in PPG peak area, SV and MAP during LBNP in LT and HT groups; c) d) Percent change of nasal alar PPG PA and SV during LBNP in female and male HT and LT groups**



