# Using PPG Morphology to Detect Blood Sequestration

**Stephen P. Linder**[1], **Suzanne M. Wendelken, Susan P. McGrath**
Department of Computer Science, Thayer School of Engineering, Dartmouth College. Hanover, NH, USA.

Changes in the morphology of the pulsatile component of the photoplethysmogram (PPG) have been shown to vary with the respiratory cycle, but changes in the morphology caused by blood sequestration has not been investigated. The morphological techniques used in this study allow the detection of changes in blood sequestration cause by changes in posture by monitoring how the shape of individual cardiac pulses change with time. Whereas frequency domain techniques can be used effectively to analyze stationary processes, analyzing the morphology of each individual cardiac cycle allows us to detect and characterize the dynamic changes in heart rate, peripheral vasoconstriction and changes in the dynamics of the blood flow to the peripheral associated with the baroreflex response to standing.

Because *volume sequestration* of blood in the lower extremities mimics the effect of rapid blood loss from traumatic injury, these results may have significance for the automatic detection of the baroreflex response to life threatening hemorrhaging in mass casualty and battlefield situations (Shamir, Eidelman et al. 1999; Olsen, Vernersson et al. 2000; Cooke, Ryan et al. 2004).

## BACKGROUND

The DC component of the PPG signal is attributable to the bulk absorption of the skin tissue, while the pulsatile component is directly attributable to variation in the amount of blood in the skin caused by the pressure pulse of the cardiac cycle. Historically frequency domain analysis (Bootsma, Swenne et al. 1996; Rusch, Sankar et al. 1996; Gratze, Fortin et al. 1998) was used to analyze this pulsatile component. However, the use of time domain techniques to extract information from the PPG signal has gained acceptance (Shamir, Eidelman et al. 1999; Johansson 2003; Shelley, Tamai et al. 2005).

Even thought the cardiac pressure pulse is somewhat damped by the time it reaches the skin, it is enough to distend the arteries and arterioles in the subcutaneous tissue. This corresponds to the rising edge of the pulse waveform. The shape of falling edge of the pulse and the trough between pulses characterizes the venous response to the cardiac cycle.

The height of pulsatile component of the PPG is proportional to the pulse pressure, the difference between the systolic and diastolic pressure in the arteries. A reduction of pulsatile amplitude can be directly attributable to either a loss of central blood pressure or constriction of the arterioles perfusing the dermis (Partridge 1987; Shamir, Eidelman et al. 1999). The pulse height is also constantly changing due to the RSA (Johansson 2003). A weakness of using pulse height as a feature, even for detecting RSA, is that it can not be calibrated and the absolute pulse height will vary depending on how and where the sensor was applied to the skin.

Three morphological features, labeled in Figure 1, are used: the Pulse Height (*PH*) is the difference between the maximum of a cardiac cycle and the previous minimum; the Cardiac Period (*CP*) is the difference in time between the peaks of two consecutive cardiac cycles, and the Normalized Peak Width (*NPW*), the Peak Width (*PW*) divided by the Cardiac Period (*CP*). The *PW* is the width of the peak at a Peak Threshold (*PT*). A PT value of 10% was selected because it was the mean height at which the slope of the trailing edge of the PPG begins to shift from being nearly linear, indicating rapid and active blood flow from the skin, to being approximately quadratic, indicating that the blood is stagnating between cardiac pressure waves (Wisely and Cook 2001). This is shown graphically in Figure 1 by the superimposed linear and quadratic dotted line.

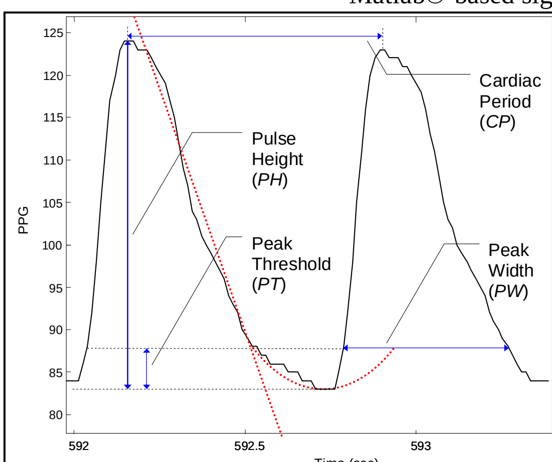


*Figure 1. Features of the pulsatile component of the PPG used to detect standing are shown: Pulse Height (PH), Cardiac Period (CP), and Peak Width (PW). A fourth feature, the Normalized Peak Width (NPW) is the ratio of PW to CP.*

## METHODS AND MATERIALS

A diverse group of eleven subjects, four women and seven men ages 20 – 43, participated in the study with informed verbal consent. The only inclusion criterion was that the subjects did not have a known cardiovascular condition.

Using two FDA approved Nonin® pulse oximeters placed on the finger and ear, we monitored 11 subjects, for three trials each, as they stood from a supine position. Each cardiac cycle was automatically extracted from the PPG waveform and characterized using statistics corresponding to normalized peak width, instantaneous heart rate, and amplitude of the pulsatile component of the ear PPG. A nonparametric Wilcoxon rank sum test was then used to detect in real-time changes in these features. Data Analysis

Matlab®-based signal processing software was written to analyze the PPG data. The algorithm extracts pulse morphology features from the PPG using a mixed-state feature extractor.

Derived PPG statistics are filtered using a Savitzky-Golay smoothing filter which fits a piecewise continuous polynomial spline to data. Events associated with standing are detected using nonparametric single-tail Wilcoxon rank sum test. The Wilcoxon rank sum test was used to test the null hypothesis ($p < 0.01$) that one sample has a statistically significant probability of having a higher (or lower) median that another sample.

## RESULTS

The output of real-time Wilcoxon-based detectors was tuned to discern

[1] *Email: spl@alum.mit.edu*

abrupt increase in *HR*, *NPW* from the finger sensor, and abrupt decrease in ear *PH* from the ear sensor. A peak in *HR* was detected in all trials for all subjects with one false positive from a strong RSA. Constriction of the $PH_{EAR}$ was detected was detected for 9 of the 11 subjects with no false alarms. While Subject 1 has a visually detectable pinch, it was only detected for one trial. Subject 9 shows a unique response to standing, with the peak amplitude increase for all three trials.

A peak in the *NPW* corresponding to standing was detected in all but two trials; detection was missed for Subject 2, Trial 1, and Subject 5, Trial 1. One false positive was detected coincident with false positive for *HR*.

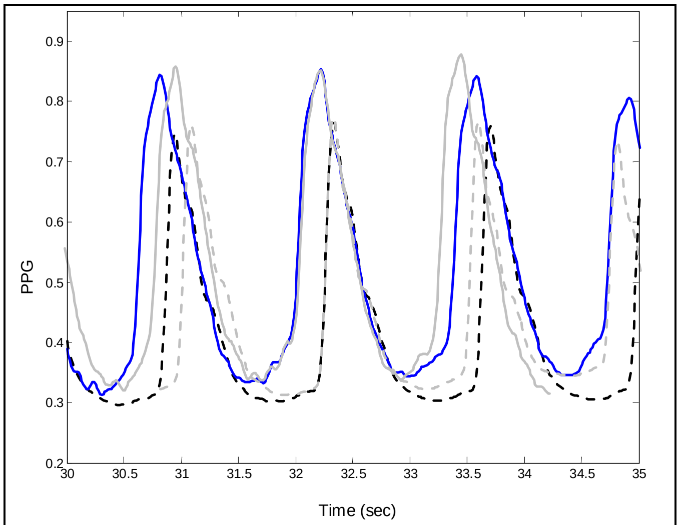


*Figure 2. Comparison of relative trough width 30 sec before standing (black) and just before standing (gray).The graphs are aligned so that the second peaks coincide with the graphs for the finger (dashed) and ear (solid) PPG. The heart rate increase before standing is almost completely attributable to the narrowing of the troughs between the peaks.*

## CONCLUSION

The blood sequestration from the orthostatic stress induced by standing from a supine position created enough of a physiological response so as to result in reliably detectable changes in the morphology of the PPG pulse. While a peak in *HR* alone would be ambiguous, the detection of a peak in *NPW* pulse along with the constriction of the $PH_{EAR}$ clearly indicates that the baroreflex associated with standing has occurred.

It was not expected that a rising peak in the *NPW* would be detected even before standing commenced. In 21 trials the *NPW* begins to peak after the prompt at 57 seconds, but before the subject actually stands at 60 seconds. This narrowing of this phase of the cardiac cycle is indicative of a shortening of time that blood remains stagnant in the skin between cardiac pressure waves as the cardiovascular system rapidly adapts to orthostatic stress of standing. As seen in Figure 2, the shape of the pressure pulse does not change significantly, yet the trough between the peaks shortens substantially. This would seem indicative of an increase in efficiency of the cardiac cycle. Over 31 trials *NPW* peaks on average 2.97 ($STD$ = 1.87) seconds before $PH_{EAR}$. After standing commences the *HR* quickly peaks, followed by a constriction of $PH_{EAR}$.

In all 33 trials, the standing event was detected as an abrupt change in at least two of these features, with only one false alarm. In 26 trials, an abrupt change was detected in all three features, with no false alarms. An increase in the normalize peak width was always detected before an increase in heart rate, and in 21 trials this feature peaked before standing commenced. After standing, the pulse rate always increases, and then amplitude of the ear PPG constricts by a factor of two or more. We hypothesis that the baroreflex first reduces the percentage of time blood flow is stagnant during the cardiac cycle, then increases the hear rate, and finally vasoconstricts the peripheral tissue in order to reestablishing a nominal blood pressure. These three features therefore can be used as a reliable detector of the baroreflex response to changes in posture or other forms of blood volume sequestration.

## ACKNOWLEDGEMENT


This research program is a part of the Institute for Security Technology Studies, supported under Award number 2000-DT-CX-K001 from the U.S. Department of Homeland Security, Science and Technology Directorate. Points of view in this document are those of the authors and do not necessarily represent the official position of the U.S. Department of Homeland Security or the Science and Technology Directorate.


## REFERENCES


Bootsma, M., C. A. Swenne, et al. (1996). "Heart rate variability during repeated incremental head-up tilt discloses time dependence of individual autonomic dynamics." *Clinical Cardiology* 19(1): 62-8.

Cooke, W. H., K. L. Ryan, et al. (2004). "Lower body negative pressure as a model to study progression to acute hemorrhagic shock in humans." *Journal of Applied Physiology* 98: 1249–1261.

Gratze, G., J. Fortin, et al. (1998). "A software package for non-invasive, real-time beat-to-beat monitoring of stroke volume, blood pressure, total peripheral resistance and for assessment of autonomic function." *Computers in Biology & Medicine* 28(2): 121-42.

Johansson, A. (2003). "Neural Network for photoplethysmographic respiratory rate monitoring." *Medical & Biomedical Engineering & Computing* 41: 242-248.

Olsen, H., E. Vernersson, et al. (2000). "Cardiovascular response to acute hypovolemia in relation to age. Implications for orthostasis and hemorrhage." *AJP-Heart and Circulatory Physiology* 278: H222-H232.

Partridge, B. L. (1987). "Use of pulse oximetry as a noninvasive indicator of intravascular volume status." *J Clin Monit* 3(4): 263-8.

Rusch, T. L., R. Sankar, et al. (1996). "Signal Processing Methods for Pulse Oximetry." *Comput. Bol. Med.* 26(2): 143-159.

Shamir, M., L. A. Eidelman, et al. (1999). "Pulse oximetry plethysmographic waveform during changes in blood volume." *British Journal of Anaesthesia* 82(2): 178-181.

Shelley, K. H., D. Tamai, et al. (2005). "The Effect of Venous Pulsation on the Forehead Pulse Oximeter Wave Form as a Possible Source of Error in Spo2 Calculation." *Anesthesia and Analgesia* 100: 743-747.

Wisely, N. A. and L. B. Cook (2001). "Arterial flow waveforms from pulse oximetry compared with measured Doppler flow waveforms apparatus." *Anaesthesia* 56(6): 556-61.