Using the Morphology of Photoplethysmogram Peaks to Detect Changes in Posture

Stephen P. Linder **Suzanne M. Wendelken** **Edward Wei** **Susan P. McGrath**

Department of Computer Science
Thayer School of Engineering
Dartmouth College
Hanover, NH, USA

ABSTRACT. 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 the baroreflex response to orthostatic stress have not been investigated. 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. 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.

Key Words. Photoplethysmogram, baroreflex, pulse oximeter, orthostatic stress.

INTRODUCTION

Temporal variation in blood volume of peripheral tissue, and thus blood flood, can be measured noninvasively using an optically-based pulse oximeter (Cook 2001; Wisely and Cook 2001). The changes in light absorption caused by the volumetric change in blood in the tissue underlying the senor gives a photometric-based plethysmogram (PPG). As seen in Figure 1, a PPG clearly shows the pulsatile waveform caused by the pressure wave from the cardiac cycle, and the respiratory sinus arrhythmia induced by breathing (Rusch, Sankar et al. 1996; Shelley and Shelley 2001). Because the pulse oximeter is noninvasive and relatively inexpensive, much research has been done in extracting additional biometric information from the sensor, with the goal of using the pulse oximeter as a primary sensor in an affordable, wearable health monitoring system (Budinger 2003; Anliker, Ward et al. 2004; Johnston and Mendelson 2004; Montgomery, Mundt et al. 2004).

The morphological techniques used in this study allow the detection of 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.

The baroreflex is a response to the orthostatic stress of standing which causes a sudden decrease in arterial blood pressure when blood pools in the lower extremities. The reflex increases heart rate and sympathetic tone, which constricts the venous system and the vasculature of the periphery, thus reestablishing a nominal blood pressure.

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

This section provides background on the cardiovascular system, the baroreflex, and pulse oximetry as they relate to this study.

A. Physiological response to orthostatic stress

When a person in the supine position (lying flat on ones back) rises to a sitting or standing position, hydrostatic pressure forces blood to the lower extremities where it pools in

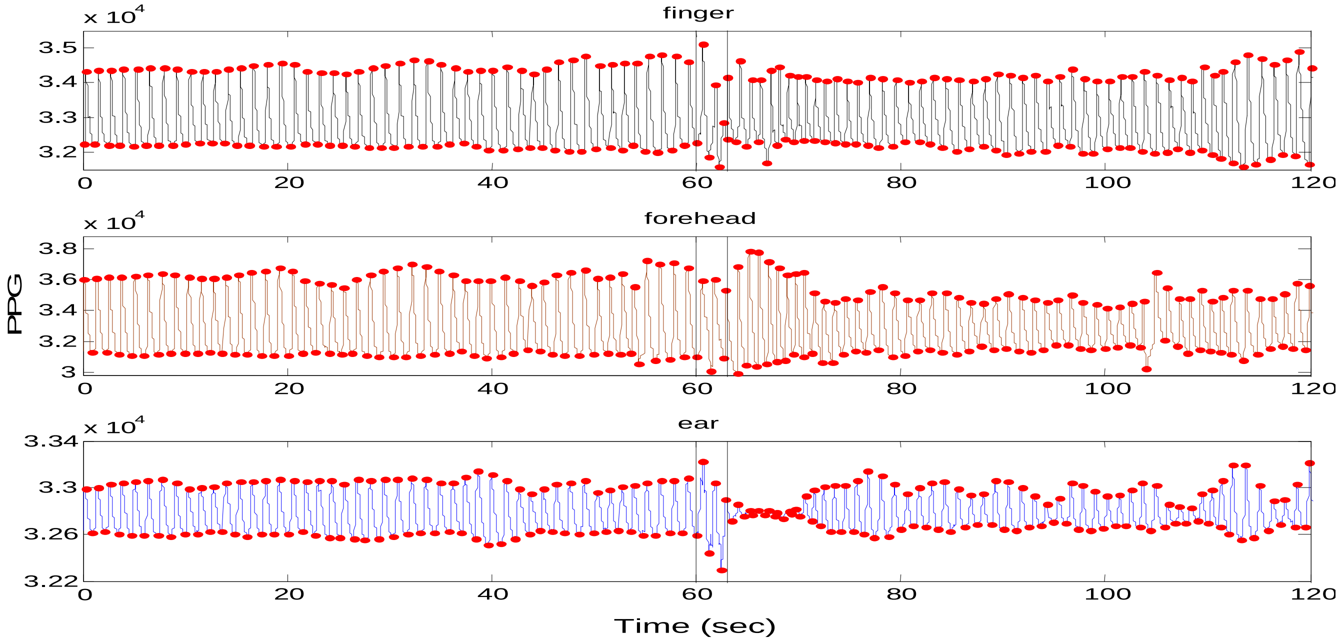

Figure 1. PPG from finger, forehead and ear from Subject 11, trial 1. The subject was initially supine and then commenced standing at 60 seconds, finishing at 65 seconds. The subject then remained standing for the final 60 seconds. The dots marked the detected peaks and valleys. Notice that all three sensors record a different response to standing.

the peripheral veins. Often referred to as *orthostatic stress*, this shift in blood volume results in a large drop in the central venous pressure, which, in turn, reduces the right ventricular filling pressure and results in a lower stroke volume for the heart. The reduced stroke volume cause the cardiac output and arterial pressure to fall and reduces cardiac output by up to 20% (Mohrman and Heller 2003; Klabunde 2005).

If arterial pressure falls by more than 20 mm Hg, the perfusion of the brain might fall to the point of inducing neurally mediated syncope, where the individual loses consciousness. Normally the baroreceptors of the arteries will detect this rapid pressure loss and reflexively increase sympathetic activity, which causes the cardiovascular system to compensate by increasing heart rate and restricting the blood flow of the periphery arterioles. Without the later response, the increase in capillary pressure in the lower limbs would cause significant edema, and loss of blood volume.

If the cardiovascular system responded only by compensating with arterial-side reflexes, up to a half liter of blood would still pool in the peripheral venous system of the legs. However, sympathetic activity and myogenic response to the stretching of the smooth muscles of the veins causes constriction of the vessels of the venous system. Along with the action of the venous valves, contraction of skeletal muscles in the extremities and the pumping action of breathing, the cardiovascular system can reduce the capillary pressure in the lower limbs to only 10-20 mm Hg above normal.

B. *Effect of Respiration on the Cardiac Cycle*

Respiration affects the cardiac cycle by varying the intrapleural pressure — the pressure between the thoracic wall and the lungs (Yasuma and Hayano 2004). This effect is often referred to as Respiratory Sinus Arrhythmia (RSA). During inspiration, intrapleural pressure decreases by up to 4 mm Hg, which distends the right atrium, allowing for faster filling from the vena cava, increasing ventricular preload, and increasing the stroke volume. Conversely during exhalation, the heart is compressed, decreasing cardiac efficiency and reducing stroke volume. When the frequency and depth of respiration increase, the venous return increases leading to increased cardiac output (Mohrman and Heller 2003), and is often referred to as *respiratory pumping*.

Shamir, Eidelman, et. al. studied the interaction between inspiration and removal of 10% of a patient's blood volume for blood banking before surgery (Shamir, Eidelman et al. 1999). They found that blood loss could be detected using either the PPG and an arterial catheter. Patients showed a decrease in RSA amplitude caused by reduced cardiac preload during exhalation when the heart is being compressed (and not as much of effect during inhalation when decreased intrapleural pressure compensates for the blood loss). This is similar to effect of standing from a supine position, where approximately 10% of the blood volume temporarily pools in the lower extremities.

Understanding the effect of the RSA on the PPG is important because there is a great variation in the RSA between individuals and each individual's RSA varies with the

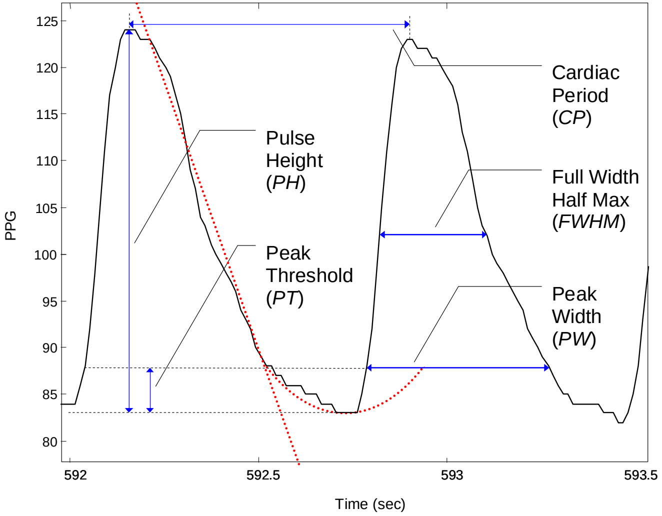

Figure 2. Features of the pulsatile component of the PPG used to detect standing are shown: Pulse Height (PH), Cardiac Period (CP), and Full Width Half Max (FWHM) and Peak Width (PW). A fourth feature, the Normalized Peak Width (NPW) is the ratio of PW to CP.

tidal volume of each respiration (Nilsson, Johansson et al. 2000; Nilsson, Johansson et al. 2003). These RSA effects on PPG can mask other physiological events, such as the baroreflex response to blood loss.

C. *Pulse Oximeter*

A conventional pulse oximeter monitors the perfusion of hemoglobin to the dermis and subcutaneous tissue of the skin. The vascular network of the subcutaneous tissue, feeds a profusion of capillaries that supply the dermis with nutrients, and feeds the venous plexus which has as its primary purpose the thermoregulation of the body.

The wide spread use of pulse oximeters to measure the oxygen saturation (SpO_2) of arterial blood in clinical settings is relatively modern, but initial experiments with such devices were conducted in the 1930's. During World War II research was conducted to have the SpO_2 of pilots monitored (Shelley and Shelley 2001).

Because the skin is so richly perfused, it allows for the relative easy detection of the pulsatile component of the cardiac cycle. This is done by illuminating the skin with light from a Light Emitting Diode (LED) and measuring the amount of light either transmitted or reflected to a photodiode. The shape of the pulsatile component of the resulting PPG differs from subject to subject, and varies with the location and manner in which the pulse oximeter is attached. As seen in Figure 1 and Figure 6, the ear, forehead and finger pulse oximeters generate different shaped pulses for the same cardiac cycle.

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 (Angelone and Jr. 1964; anonymous 1996; 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; Cook 2001; 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. Because the venous system is at a much lower pressure as the blood moves through the capillaries, a slight change in tissue compression can change its morphology. When the applied pressure is small, a secondary pulse can also be seen as the venous plexus distends after the blood drains from the arterial system (Shelley, Tamai et al. 2005). Changes in the venous return can be clearly seen in the finger PPG in Figure 6.

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). However, the constriction in the ear PPG, as seen in Figure 1, can directly be attributable to vasoconstriction of the ear, and not the RSA, since it does not occur at the finger or forehead.

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. Also, as seen in Figure 1, the pulsatile amplitude from the PPG from a finger, forehead and ear do not correlate well, even when the subject is supine, as in the first 60 seconds of data. While we use pulse height as feature, we will detect relative changes in the pulse height of the ear PPG.

The morphology features that are used in our study to characterize each pulse are labeled in Figure 2. 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. The Full Width Half Max (*FWHM*) is the width of the peak at half the maximum value of the cardiac cycle. The Peak Width (*PW*) is the width of the peak at a predetermined Peak Threshold (*PT*). The Normalized Peak Width (*NPW*) is the *PW* divided by the

Cardiac Period (*CP*).

Determining a single value of PT for all trials required us to analyze the normalized PPG pulses from all trials. 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 2 by the superimposed linear and quadratic dotted line.

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.

Three FDA approved Nonin® pulse oximeters were placed on the subjects as shown in Figure 4: a forehead reflectance probe placed horizontally on the forehead and attached with a Nonin® holder[1]; a reflective ear-clip sensor placed on the left earlobe; and a transmission finger clip placed on the left index finger. In order to prevent motion artifact from cord movement and rotation, the cords from the ear and forehead probes were tethered to the subject's shirt with a clip.

Each sensor was connected to a Nonin OEM III interface module which generated data packets at 75 Hz of filtered 16-bit PPG data. The PPG signal was pre-processed by the OEM III with high pass and notch filters. A serial RS232 interface allows a personal computer to record data.

Figure 4. Placement of forehead, ear, and finger pulse oximeters on subject.

A Java-based program was developed to simultaneously log data from multiple sensors. The annotated data was saved in text files for later analysis.

A. Experimental Protocol

In each trial, data was recorded continuously from the three pulse oximeter probes for three minutes. The subjects were instructed to breath normally, remain still, not to talk and to keep their left hand in front of them, at their waist, during data collection. During the first minute, the subject was supine on a couch. The subject was verbally prompted at 57 seconds to prepare to stand and then prompted again at 60 seconds to stand. The subject remained stationary and standing for the next minute. The subject was finally instructed to again recline on the couch for the final minute.

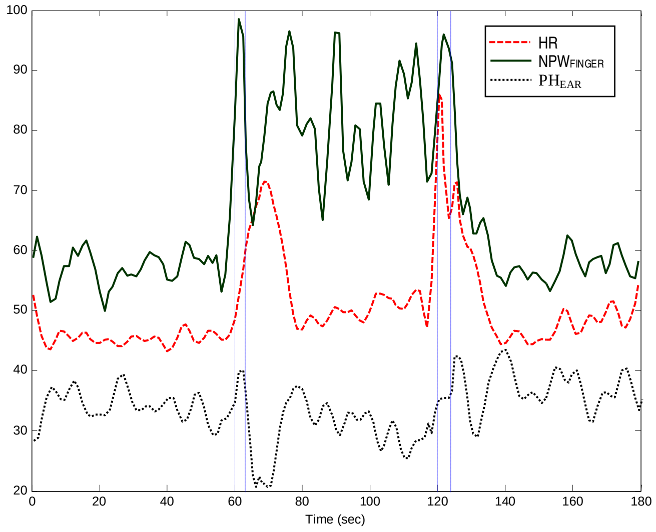

Figure 3. Filtered Heart Rate (HR), Normalized Pulse Width (NPW$_{FINGER}$) and Pulse Width for forehead (PW$_{EAR}$) for subject 11, trial 2. Standing and recline at 60 and 120 seconds respectively are marked with vertical dotted rules. As with 21of the 33 trials the NPW begins to rise before the heart rate.

B. Data Analysis

Matlab®-based signal processing software was written to analyze the PPG data[2]. The algorithm extracts pulse morphology features from the PPG using a mixed-state feature extractor based on previous work on sequential state estimation (Schell, Linder et al. 2004). This feature extractor allows statistics about each individual pulse, including pulse height, width, area, rise and fall time, and instantaneous heart rate to obtain as shown in Figure 2. Changes in the morphology of individual pulses were analyzed and the data from the three sensors were cross-correlated for each trial. As seen in Figure 3, there is an abrupt increase in heart rate, pinching in the pulse amplitude of the ear PPG, and narrowing of the PPG peak upon standing for most subjects.

Derived PPG statistics are filtered using a Savitzky-Golay smoothing filter which fits a piecewise continuous polynomial spline to data. The Savitzky-Golay filter has the advantage of preserving sharp transitions. We used a window size of nine cardiac cycles, with a polynomial of order 4. The window size was selected to equal the length of a

---

[1] In order to apply consistent and constant pressure the Nonin holder uses a piece of elastic foam to press the sensor to the skin.

[2] Our Matlab source-code is available on request for noncommercial use.

typical respiration cycle.

C. *Event Detection*

Events associated with standing are detected using non-parametric single-tail Wilcoxon rank sum test. This statistically test was used instead of the commonly used Student t-test because the PPG features can not be parameterized as Gaussian. The Wilcoxon rank sum test was used to test the null hypothesis that one sample has a statistically significant probability of having a higher (or lower) median that another sample. Our statistical threshold was $p < 0.01$.

Our event detector uses a pair of consecutive sliding windows: $W_{baseline}$, a window used as a baseline consisting of the previous data; and W_{event}, a window of the most current data. An abrupt change in a feature, as would be caused by standing, is detected by testing if the median of the data in W_{event} is statistically different than from the data in $W_{baseline}$. Because of the RSA induced variations in the PPG statistics, the detection threshold need to be selected to maximize the probability of detection of standing for the 33 trials while minimizing the probability of false alarms from RSA induced variation in the PPG waveform.

RESULTS

The output of real-time Wilcoxon-based detectors was tuned to discern abrupt increase in Heart Rate (*HR*), Full-Width Half Max (*FWHM*), and Normalized Pulse Width (*NPW*) from the finger sensor, and abrupt decrease in ear Pulse Height from the ear sensor (PH_{EAR}). Two sensors were needed because while the ear amplitude was suppressed we were unable to accurately estimate the other three statistics. As seen in Figure 5 the detectors can successfully detect standing while rejecting changes associate with a normal RSA. The same detector configuration was used for all subjects. We also found that the forehead PPG gave similar results to finger PPG.

The peak in *HR* was detected by testing for a 20% increase in median pulse rate , with a $W_{baseline}$ = 40 cardiac cycles and W_{event} = 5 cardiac cycles. All trials for all subjects resulted in a detection. One false positive for Subject 5, Trial 3, as seen in Figure 5, was from a strong RSA.

Constriction of the PH_{EAR} was detected for a twofold decrease in median pulse amplitude, with a $W_{baseline}$ = 35 cardiac cycles and W_{event} = 8 cardiac cycles. The constriction associate with standing 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 by testing for a 5% increase trough width, with a $W_{baseline}$ = 40 cardiac cycles and W_{event} = 5 cardiac cycles. Standing in all but two trials was detected; detection was

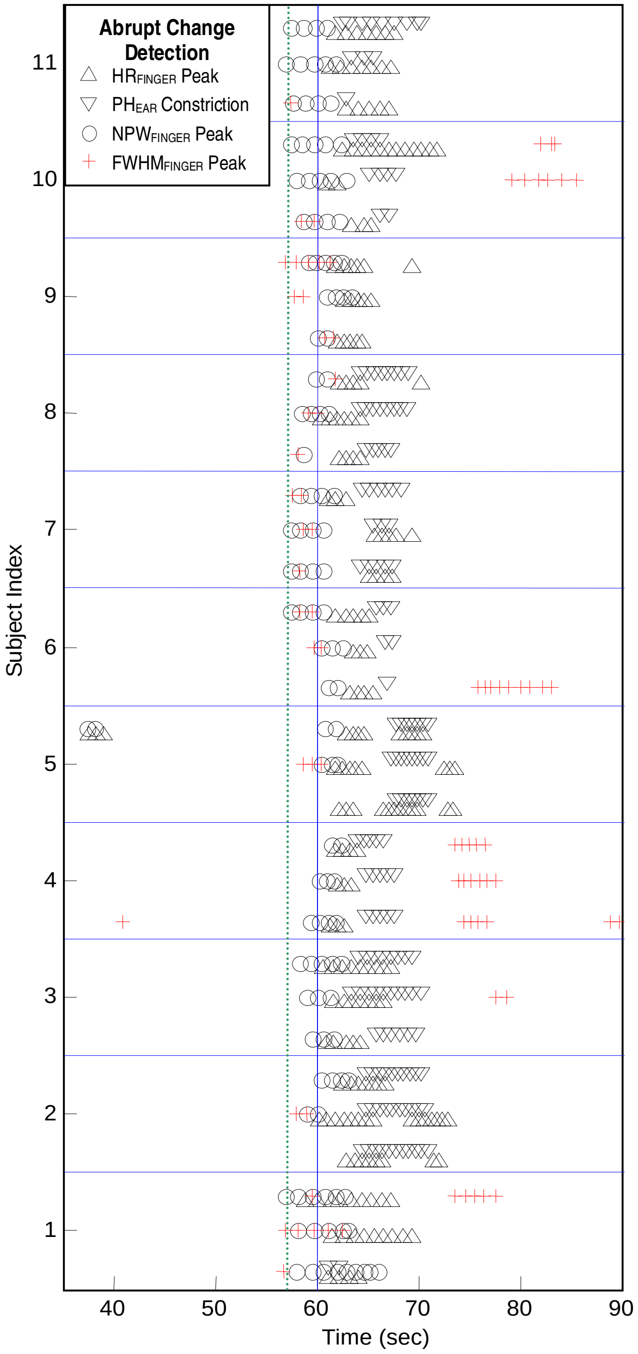

Figure 5. Output of real-time detectors tuned to detect abrupt increase in Heart Rate (HR), Full-Width Half Max (FWHM), and Normalized Pulse Width (NPW), and abrupt decrease in ear Pulse Height (PH_{EAR}). Detectors were tuned to detect standing while rejecting changes associate with a subject's normal Respiratory Sinus Arrhythmia. The horizontal rules separate the three trials from each subject, while the two vertical rules mark the three second prompt to stand, and the start of standing at 60 seconds. The time range was selected to show all false positives.

missed for Subject 2, Trial 1, and Subject 5, Trial 1. One false positive was detected coincident with false positive for *HR*. Visual inspection of Figure 6 shows that the peak becomes a comparatively wider portion of the cardiac period as the valley width decreases when subject is (a) supine, (b) preparing to stand, and (c) standing.

Finally, standing was detected using *FWHM* by testing for a 5% increase ($p < 0.01$), with a $W_{baseline}$ = 40 cardiac cycles and W_{event} = 5 cardiac cycles. As seen in Figure 5 these detections did not correlate well with standing, detecting standing in only 54% of the trials, with 10 false positives. Visual inspection of the *FWHM* graphs shows that the half height width did not peak during standing for at least half of the subjects.

DISCUSSION

Pulse oximetry is a low cost, noninvasive technique that has been successfully used in hospitals to measure oxygen saturation and average pulse rate. Because previous techniques for analyzing electro cardiogram relied predominately on frequency-domain signal processing techniques, many of the original results using pulse oximeters did the same. As and example the Using heart rate variability techniques to access cardiac health were developed using frequency-domain techniques and are still widely used and sited (Bootsma, Swenne et al. 1996). However, these techniques require a sampling window of data which usually encompasses several cardiac cycles, so any statistics gives an average of a feature, such as the heart rate, for those cycles.

More recently studies have focused on the morphology of the pulsatile component of the PPG. Research shows that with morphological-based signal processing the quality of breathing can even be ascertained from the RSA (Nilsson, Johansson et al. 2000; Johansson 2003). However, researchers predominately use the Igor Waveform Analysis package[3] (Awad, Ghobashy et al. 2001; Shelley, Tamai et al. 2005) which limits the flexibility of the features that can be studied. By extracting shape features from each individual cardiac pulse, detectors such as the one described here can be developed to monitor for transitory events.

While previous research only tried to distinguish between whether a subject was either supine or standing using frequency domain analysis, this current effort was to detect an actual response to standing. As expected, the orthostatic stress of 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

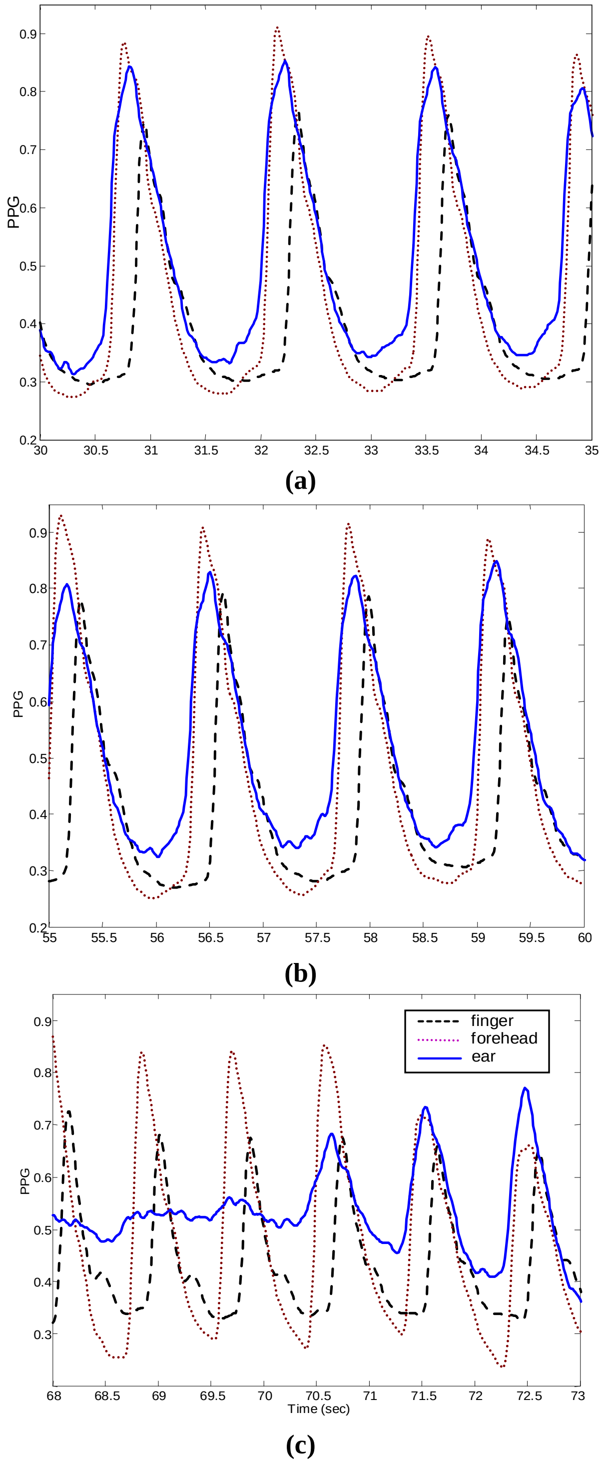

Figure 6. Changes in PPG signal from finger, ear and forehead as posture changes for subject 11, trial 1: (a) supine, (b) just after standing and (c) as standing. The ear PPG is constricted at the start of (c) and the valley of the forehead and finger PPG is narrower.

---

[3] http://www.wavemetrics.com/products/igorpro/dataanalysis/peakanalysis/peakfinding.htm

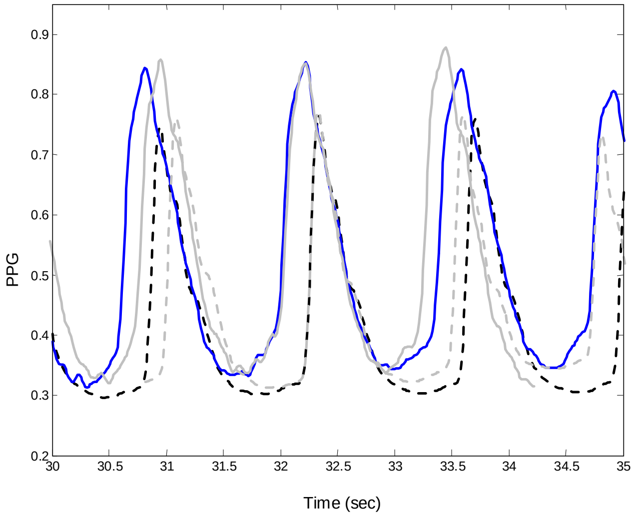

Figure 7. The trough between the peaks narrows before standing while the shape and width of the peak remains almost identical. The grayed graphs from Figure 6 (b) are aligned and scaled so that the second peaks coincide with the graphs of Figure 6 (a) for the finger (dashed) and ear (solid) PPG. The heart rate increase is almost completely attributable to the narrowing of the troughs between the peaks.

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. As seen in Figure 5, 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 7, 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 *HR*.

When standing commences the *HR* quickly peaks, followed by a constriction of PH_{EAR}. This result is support by (Jablonka, Awad et al. 2004) where they determined that the ear is particularly sensitive to vasopressins. Over 27 trials *HR* peaks on average 2.62 (*STD* = 1.72) seconds before PH_{EAR}.

While an increase in heart rate alone can have multiple physiological causes, when it occurs concurrently with a peak in NPW and a constriction of $\mathrm{PH}_{\mathrm{EAR}}$, it appears to be an unambiguous discriminator for the baroreflex response to the orthostatic stress of standing.

While we reliably detect abrupt changes in *HR*, PH_{EAR} and *NPW* during standing these changes could not be as reliably detected when the subject later reclined from standing. The reclining response was comparatively muted, with heart rate often taking over a minute to recover.

In 25 of 33 trials there was some response to reclining detected, with one false alarm, but in only 11 trials was both a peak in *HR* and *NPW* detected. Twice a constriction of the PH_{EAR} was detected. *FWHM* peaked for reclining in 19 trials, but with 11 false alarms.

CONCLUSION

The morphological analysis of the PPG from pulse oximeters provides an inexpensive tool to detect the baroreflex associated with standing in a diverse population of individuals. Our research shows that one can detect that the cardiovascular system prepares to for the orthostatic stress of standing even before the subject begins to rise. The normalized peak width becomes significantly wider after the subject is told to prepare to stand in three seconds, indicative of reflective reduction in the percentage of time the blood is stagnant between cardiac cycles. When standing does commences, a detectable increase in heart rate immediately occurs, followed by the vasoconstriction of the periphery.

Pending IRB approval, we will examine the efficacy of our approach in monitoring the cardiovascular stress and health of a subject while undergoing a tilt table test and lower body negative pressure test. Because these tests provide a more controlled environment for studying the effects of blood sequestration, these experiments will help in developing robust algorithms for the automatic detection of life threatening injuries.

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

Angelone, A. and N. A. C. Jr. (1964). "Respiratory sinus arrhythmia: a frequency dependent phenomenon." *Journal of Applied Physiology* **19**: 479-482.

Anliker, U., J. A. Ward, et al. (2004). "AMON: A Wearable Multiparameter Medical Monitoring and Alert System." *IEEE Transactions on Information Technol-*

ogy in Biomedicine **8**(4): 415- 427.

anonymous (1996). "Heart rate variability: standards of measurement, physiological interpretation and clinical use. Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology." *Circulation* **93**(5): 1043-65.

Awad, A. A., M. A. Ghobashy, et al. (2001). "Different responses of ear and finger pulse oximeter wave form to cold pressor test." *Anesth Analg* **92**(6): 1483-6.

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.

Budinger, T. F. (2003). "Biomonitoring with wireless communications." *Annual Review of Biomedical Engineering* **5**(383-412).

Cook, L. B. (2001). "Extracting arterial flow waveforms from pulse oximeter waveforms apparatus." *Anaesthesia* **56**(6): 551-5.

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.

Jablonka, D. H., A. A. Awad, et al. (2004). *The Effect of Vasopressin on Ear Pulse Plethysmography and Its Implications on Cerebral Blood Flow Regulation*. ASA Annual Meeting, Las Vegas, Nevada, Anesthesiology.

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

Johnston, W. S. and Y. Mendelson (2004). *Extracting Breathing Rate Information from a Wearable Reflectance Pulse Oximeter*. International Conference of the IEEE EMBS, San Francisco, CA, USA.

Klabunde, R. E. (2005). *Cardiovascular Physiology Concepts*. Baltimore, MD, Lippincott Williams and Wilkins.

Mohrman, D. E. and L. J. Heller (2003). *Cardiovascular Physiology*. New York, McGraw-Hill/Appleton & Lange.

Montgomery, K., C. Mundt, et al. (2004). *Lifeguard- A Personal Physiological Monitor For Extreme Environments*. International Conference of the IEEE EMBS, San Francisco, CA, USA.

Nilsson, L., A. Johansson, et al. (2000). "Monitoring of respiratory rate in postoperative care using a new photoplethysmographic technique." *Journal of Clinical Monitoring and Computing* **16**: 309-315.

Nilsson, L., A. Johansson, et al. (2003). "Macrocirculation is not the sole determinant of respiratory induced variations in the reflection mode photoplethysmographic signal." *Physiol Meas* **24**(4): 925-37.

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.

Schell, C., S. P. Linder, et al. (2004). "Tracking highly maneuverable targets with unknown behavior." *Proceedings of the IEEE* **92**(3): 558 - 574.

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. and S. Shelley (2001). Pulse Oximeter Waveform: Photoelectric Plethysmography. *Clinical Monitoring*. Carol Lake, R. Hines and C. Blitt, W.B. Saunders Company**:** 420-428.

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.

Yasuma, F. and J.-i. Hayano (2004). "Respiratory sinus arrhythmia: why does the heartbeat synchronize with respiratory rhythm?" *Chest* **125**(2): 683-90.