

Role of the Photoplethysmographic Waveform in the Care of High-risk Surgical Patients

To the Editor:

My colleagues and I read with great interest the recent article by Hengy¹ with the accompanying editorial by Cansson.² We commend the authors for studying the relation between the photoplethysmographic waveform and arterial pressure waveform with regard to respiratory modulation. As was pointed out by the authors of the study, and the accompanying editorial, the physiology behind the photoplethysmographic waveform is complex as it results from the interactions between the cardiovascular, respiratory, and autonomic systems. These interactions cause modulations in the two pools of blood (arterial and venous) within the region being monitored (in this case, the finger).

Adding to this complexity are the inherent limitations of digital signal processing. As noted by the authors, they used a commercial pulse oximeter with preexisting proprietary filters.³ These filters are created by a biomedical engineer, based on assumptions regarding what a clinician would find clinically useful *versus* simply being an artifact. Until recently, the

respiratory modulation of the photoplethysmographic was not considered to contain useful information, and therefore, most commercial pulse oximeters suppress those signals (*via* band pass filters and autocentering routines).

In addition, the authors used a commercial program (AcqKnowledge; Biopac Systems, Goleta, CA) combined with a homemade Excel (Redmond, WA) spreadsheet for their fundamental measurement (photoplethysmographic amplitude). This does not invalidate their results, but it should be noted that their approach of using time domain data analysis exclusively is fundamentally limiting, given the complexity of the photoplethysmographic signal. As shown in figures 1 and 2, photoplethysmographic respiratory-induced modulation is made up of the combined modulations of arterial and venous blood. We believe that amplitude modulation is primarily associated with stroke-volume variation, whereas baseline modulation is the result of the movement of venous blood.⁴ This dual modulation (baseline and amplitude), which is based on two different physiologies (arterial and venous), significantly increases the difficulty of isolating stroke-volume variation, but holds the promise of providing information regarding the patient's preload conditions.

We have recently patented methods of separating the photoplethysmographic waveform into these two different

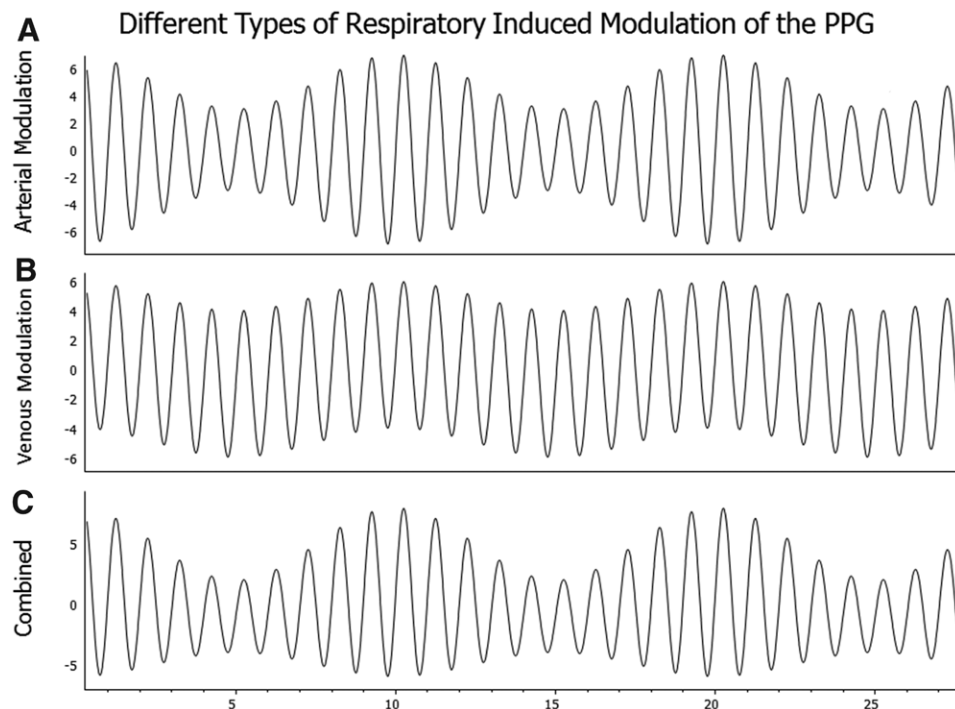


Fig. 1. Hypothetical photoplethysmographic traces demonstrating two types of modulation. (A) An example of amplitude modulation that might occur with stroke-volume variation. (B) Baseline modulation that would occur with the movement of venous blood with ventilation. Δ variation of the plethysmographic waveform of the pulse oximeter as described would measure type A modulations but completely miss type B. The spectral analysis can measure both type A and type B modulations.⁶ (C) Demonstrates the effect of combining the two types of modulation together (A and B), which occurs with patients who are significantly hypovolemic (fig. 2). PPG = photoplethysmographic.

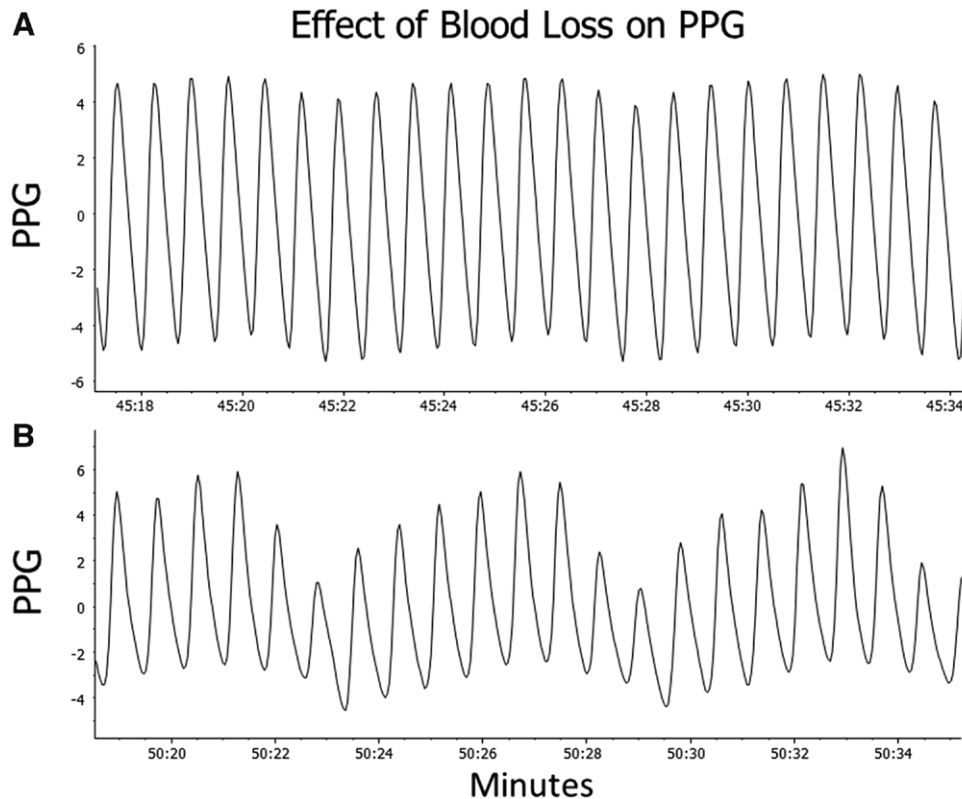


Fig. 2. The photoplethysmographic waveform from a patient who suffered a sudden blood loss (~500 cc) during a surgical procedure. It demonstrates a shift from a simple baseline venous modulation (A) to a combined arterial and venous modulation (B). One way to separate these two effects (arterial and venous) is by using Fourier analysis.⁷

types of modulations.⁵ It is hoped that useful information, regarding a patient's cardiovascular status over a wide range of clinical conditions, may be gleaned from the noninvasive photoplethysmographic waveform, using both frequency domain (Fourier analysis) and time domain (envelope analysis) approaches.

We disagree with the editorial's implication that there is something inherently limiting about the photoplethysmographic signal, which would prevent its use during high-risk surgeries. In our opinion, there are two key factors needed for the correct interpretation of the photoplethysmographic waveform: (1) a better understanding of the underlying physiology behind the waveform (in this case the influence of respiratory-induced venous modulation) and (2) better digital signal processing to allow for the correct isolation of the underlying signals (such as the use of harmonic analysis techniques). With these two factors in place, one is left with the intriguing possibility of simultaneously monitoring both the preload conditions (*via* venous baseline modulation) and stroke volume (*via* pulse amplitude modulation). Considering the noninvasive nature of the signal and the ability to measure it at multiple sites simultaneously, we believe that the potential of this waveform needs further investigation.

Kirk H. Shelley, M.D, Ph.D.,* Aymen A. Alian, M.D., Adam J. Shelley, M.S., *Yale University, New Haven, Connecticut.
kirk.shelley@yale.edu

References

- Hengy B, Gazon M, Schmitt Z, Benyoub K, Bonnet A, Viale JP, Aubrun F: Comparison between respiratory variations in pulse oximetry plethysmographic waveform amplitude and arterial pulse pressure during major abdominal surgery. *ANESTHESIOLOGY* 2012; 117:973–80
- Cannesson M, Manach YL: Noninvasive hemodynamic monitoring: No high heels on the farm; no clogs to the opera. *ANESTHESIOLOGY* 2012; 117:937–9
- Feldman JM: Can clinical monitors be used as scientific instruments? *Anesth Analg* 2006; 103:1071–2
- Walton ZD, Kyriacou PA, Silverman DG, Shelley KH: Measuring venous oxygenation using the photoplethysmograph waveform. *J Clin Monit Comput* 2010; 24:295–303
- Shelley KH, Shelley AJ, Silverman DG, Stout RG: Method of assessing blood volume using photoelectric plethysmography. USPTO August 28, 2012, Assignee Yale University, US8251912B2
- Shelley KH, Jablonka DH, Awad AA, Stout RG, Rezkanna H, Silverman DG: What is the best site for measuring the effect of ventilation on the pulse oximeter waveform? *Anesth Analg* 2006; 103:372–7
- Michard F: Using pulse oximetry waveform analysis to guide fluid therapy: Are we there yet? *Anesth Analg* 2007; 104:1606–7

(Accepted for publication February 25, 2013.)