Journal of Clinical Monitoring and Computing (2006) 20: 81–87
DOI: 10.1007/s10877-006-9010-7

THE USE OF JOINT TIME FREQUENCY ANALYSIS TO QUANTIFY THE EFFECT OF VENTILATION ON THE PULSE OXIMETER WAVEFORM

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J Clin Monit Comput 2006; 20: 81–87

ABSTRACT. Objective. In the process of determining oxygen saturation, the pulse oximeter functions as a photoelectric plethysmograph. By analyzing how the frequency spectrum of the pulse oximeter waveform changes over time, new clinically relevant features can be extracted. **Methods.** Thirty patients undergoing general anesthesia for abdominal surgery had their pulse oximeter, airway pressure and CO_2 waveforms collected (50 Hz). The pulse oximeter waveform was analyzed with a short-time Fourier transform using a moving 4096 point Hann window of 82 seconds duration. The frequency signal created by positive pressure ventilation was extracted using a peak detection algorithm in the frequency range of ventilation (0.08–0.4 Hz = 5–24 breaths/minute). The respiratory rate derived in this manner was compared to the respiratory rate as determined by CO_2 detection. **Results.** In total, 52 hours of telemetry data were analyzed. The respiratory rate measured from the pulse oximeter waveform was found to have a 0.89 linear correlation when compared to CO_2 detection and airway pressure change. the bias was 0.03 breath/min, SD was 0.557 breath/min and the upper and lower limits of agreement were 1.145 and −1.083 breath/min respectively. The presence of motion artifact proved to be the primary cause of failure of this technique. **Conclusion.** Joint time frequency analysis of the pulse oximeter waveform can be used to determine the respiratory rate of ventilated patients and to quantify the impact of ventilation on the waveform. In addition, when applied to the pulse oximeter waveform new clinically relevant features were observed.

KEY WORDS. pulse oximeter, waveform analysis, plethysmograph, non-invasive monitoring.

INTRODUCTION

The pulse oximeter has become the most commonly used patient monitors both in and out of the operating room. This popularity is undoubtedly due to the pulse oximeter's ability to monitor both arterial oxygen saturation as well as basic cardiac function (i.e. heart rhythm) non-invasively. In addition, it is remarkably easy to use and comfortable for the patient. It would only seem logical that we should strive to maximize the benefit we derive from this device. Recent advances in digital signal processing combined with improving pulse oximeter technology, is allowing for a reexamination of this device. This paper introduces a new method of analysis of the pulse oximeter waveform.

In the process of determining oxygen saturation, the pulse oximeter functions as a photoelectric plethysmograph. In this role, it non-invasively measures minute

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Received 8 September 2005, Accepted for publication 10 January 2006.

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changes of the blood volume of a vascular bed (i.e. finger, ear or forehead) over time. The photoelectric plethysmograph is not a new invention [1]. While the plethysmograph has been examined previously as a potential anesthesia monitoring device [2], until recently remarkably little research has been done on this ubiquitous signal. It is important to understand that the typical pulse oximeter waveform that is presented to the clinician is a highly filtered and processed signal. It is normal practice for equipment manufacturers to use both auto-centering and auto-gain routines on the displayed plethysmographic waveforms. Despite this fact, the pulse oximeter waveform is still rich in information regarding the physiology of the patient. It contains a complex mixture of the influences of arterial, venous, autonomic and respiratory systems on the peripheral circulation. There is interest in quantifying the impact of ventilation on the pulse oximeter waveform in order to determine the respiratory rate [3–5]. It has also been suggested that the degree of ventilation-induced fluctuation is related to the respiratory tidal volume [6] and blood volume [7, 8]. Key to the successful interpretation of this waveform is the ability to separate it into fundamental components. Harmonic analysis using short-time Fourier analysis is one method of studying waveforms. It allows for the extraction of underlying signals that contribute to a complex waveform and to study how these underlying signals vary over time. Similar methods have been used previously, with the pulse oximeter and photoelectric plethysmograph to improve the accuracy of the oxygen saturation measurement [9], to monitor tissue perfusion [10] and detect individual breaths (using wavelets) [11]. This paper will examine the use of joint time frequency analysis to quantify the influence of ventilation on the pulse oximeter waveform. By quantifying how the frequency spectrum of the pulse oximeter waveform changes over time, it is hoped that new clinically relevant features can be extracted.

METHODS AND MATERIALS

This study was reviewed and approved by our Human Investigation Committee. As a part of a study of the pulse oximeter waveforms 30 patients undergoing elective lower abdominal procedures (gynecological and urological procedures) under general anesthesia had their pulse oximeter waveform, airway pressure and CO_2 waveform collected. As a part of their anesthetic, patients were intubated after induction with propofol (2.0–3.0 mg·kg^{-1} IV) and vecuronium (0.1 mg·kg^{-1} IV). Sevoflurane 1–3% in combination with nitrous oxide 60% in oxygen was administered for maintenance of anesthesia. The ventilator was set to a tidal volume of 10 cc/kg at an I:E ratio of 1:2. An upper body Bair Hugger warming unit (Model 505; Augustine Medical, Inc., Eden Prairie, MN) was applied at the start of the procedure and used throughout the case. Additional medications (fentanyl, morphine and ondansetron) were given at the discretion of the anesthesia care team. At the end of surgery, inhaled anesthetics were discontinued and residual neuromuscular blockade was reversed with neostigmine (0.05 mg·kg^{-1} IV) and glycopyrrolate (0.01 mg·kg^{-1} IV). During the surgery, estimated blood loss was closely followed and recorded, as well as replacement fluid given to the patient. Three times during the surgical procedure (pre-incision, mid procedure & during closure) the ventilator was adjusted. This adjustment consisted of increasing the tidal volume till the peak pressure airway pressure was increased above baseline by 10 cm H_2O (to a limit of 35 cm H_2O). This higher setting was maintained for 5 mins and then returned to baseline.

The data were collected using a computer acquisition system consisting of a 16-bit A-to-D PC card (DAQCard AI-16XE-50, National Instruments, Austin, TX) sampling at 50Hz. BioBench (Version 1.0, National Instruments, Austin, TX) was the software used for the acquisition process. Waveform analysis was accomplished with Igor Pro (WaveMetrics, Inc. Lake Oswego, OR).

The pulse oximeter waveform was collected with a clinical pulse oximeter (OxiPleth Model 520A, Respironics, Wallingford, CT) using a standard finger probe. The pulse oximeter had its auto-gain function disabled. The pulse oximeter waveform collected consists of the AC portion of inverted infrared signal (approximate 940nm). The airway pressure and CO_2 were obtained from an Ohmeda RGM gas monitor (model 5250, Datex-Ohmeda, Madison WI).

Data analysis was preformed with Igor Pro (WaveMetrics, Lake Oswego, OR), a data analysis and graphics package. The pulse oximeter waveform was analyzed with a short-time Fourier transform (Equation (1)) using a moving 4096 point Hann window (Equation (2)) of 82 seconds duration (see Table 1 for details).

$$X[k, n_0] = \sum_{m=n_0-N+1}^{n_0} (w[m - n_0]x[m])e^{-j(2\pi N)km}$$
$$k = 0, 1, 2 \ldots, N-1 \quad (1)$$
$$w(n) = 0.5 - 0.5\cos(2\pi n/N - 1) \quad (2)$$

The frequency signal created by positive pressure ventilation was extracted using a peak detection algorithm in the frequency range of ventilation (0.08–0.4 Hz). The respiratory rate derived in this manner was compared to the respiratory rate as detected by CO_2 production which was used as the gold standard for comparison purposes.

Table 1. Method of calculation of the joint time frequency of the pulse oximeter waveform

1. Pulse oximeter waveform was converted to a numeric series by analog to digital conversion with sampling of the continuous pulse oximeter output at a rate of 50 Hz. The sampled waveform was collected into a digital buffer, presently 4096 points - 82 seconds.
2. A windowing function was used on the data in the digital buffer (presently Hann window is used). The windowing function was designed to minimize the effect of the finite range of the sample set.
3. A Fourier analysis was performed on the data set in the digital buffer. The data was then expanded in a logarithmic fashion. The logarithmic expansion was done to compensate for the otherwise overwhelming signal strength for the heart rhythm.
4. The result was transferred to a display buffer. The digital buffer then accepted new data from the pulse oximeter on a first-in, first-out basis. The amount of new data added was determined by respiratory rate measured up to that point. Specifically, the amount of data associated with the time of one breath was added to buffer. The new data were then analyzed by the method outlined in steps 2 & 3.
5. The resulting data were plotted with Y axis - frequency & X axis- time. A number of different techniques can be used to display the results (false color, gray scale, "waterfall" or as a surface plot).

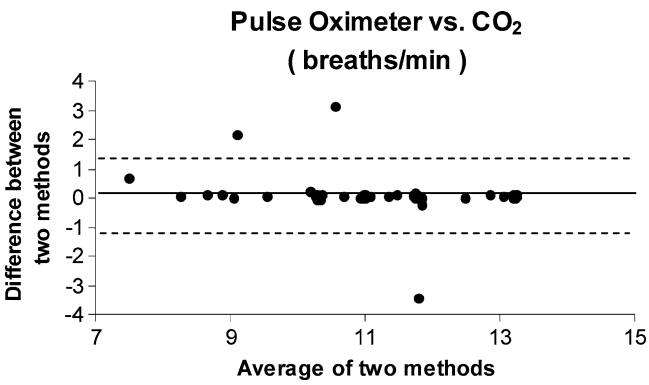

Fig. 1. Plot of the difference between the respiratory rate as determined by pulse oximeter waveform analysis and CO_2 detection. The solid line is the bias, and the two dashed lines are the upper and lower limits of agreement.

RESULTS

In total, 52 hours of telemetry data were analyzed. The respiratory rate measured from the pulse oximeter waveform had a 0.89 linear correlation (Pearson coefficient of correlation) when compared to CO_2 detection as a method of detecting ventilation. Further analysis was done using the method described by Bland and Altman [12]. The differences between each pair of values obtained using the two different methods were plotted as the Y-axis, whereas the averages of the each pair of values obtained using the two different methods were plotted as the X-axis. The mean (bias), standard deviation (SD) (as a measure of precision), and the upper and lower limits of agreement were calculated. As seen in Figure 1, the bias was 0.03 breath/min, SD was 0.557 breath/min and the upper and lower limits of agreement were 1.145 and −1.083 breath/min respectively. Review of the data revealed that the presence of motion artifact proved to be the primary cause of failure of this technique. In addition, the presence of random heart beats (i.e. atrial-fibrillation & frequent PVC/PAC) disrupted the regularity of underlying waveform created by the pulse (Figure 3A).

Ventilation was observed to have two distinct impacts on the pulse oximeter waveform. The most commonly seen was a shift of the baseline with each breath. Baseline is often referred to as the "DC" component of the pulse oximeter waveform. Shifts in the baseline are felt to be associated with changes in the venous bed (non pulsatile blood) [2]. This type of DC modulation was observed in all studied patients during both periods of controlled and spontaneous ventilation (Figure 2A).

The less commonly seen phenomenon was a change of the amplitudes of the pulse beats (AC component) with each breath. This type of AC modulation has been associated with hypovolemic states [7, 8]. Modulation of the amplitude of the pulse beat is manifested on the JFTA as secondary harmonics surrounding the pulse harmonics. The distance between the secondary harmonics and the pulse harmonic is equal to the respiratory rate. These secondary harmonics were present in all patients (eight) who had more than a 300cc blood loss (Figure 2B).

Two factors influenced the strength of the respiratory signal contained in the pulse oximeter waveform; 1) the airway pressure and 2) the volume status of the patient. For any given patient higher airway pressures always resulted in a stronger respiratory signal. In addition, as significant blood loss (>300cc EBL) occurred during a surgical procedure (seen in 8 cases) the respiratory signal would increase strength to only return to baseline as fluid replacement was given. Though not studied this method of harmonic analysis appeared to successfully detect spontaneous ventilation that occurred at the end of the procedures (Figure 3B).

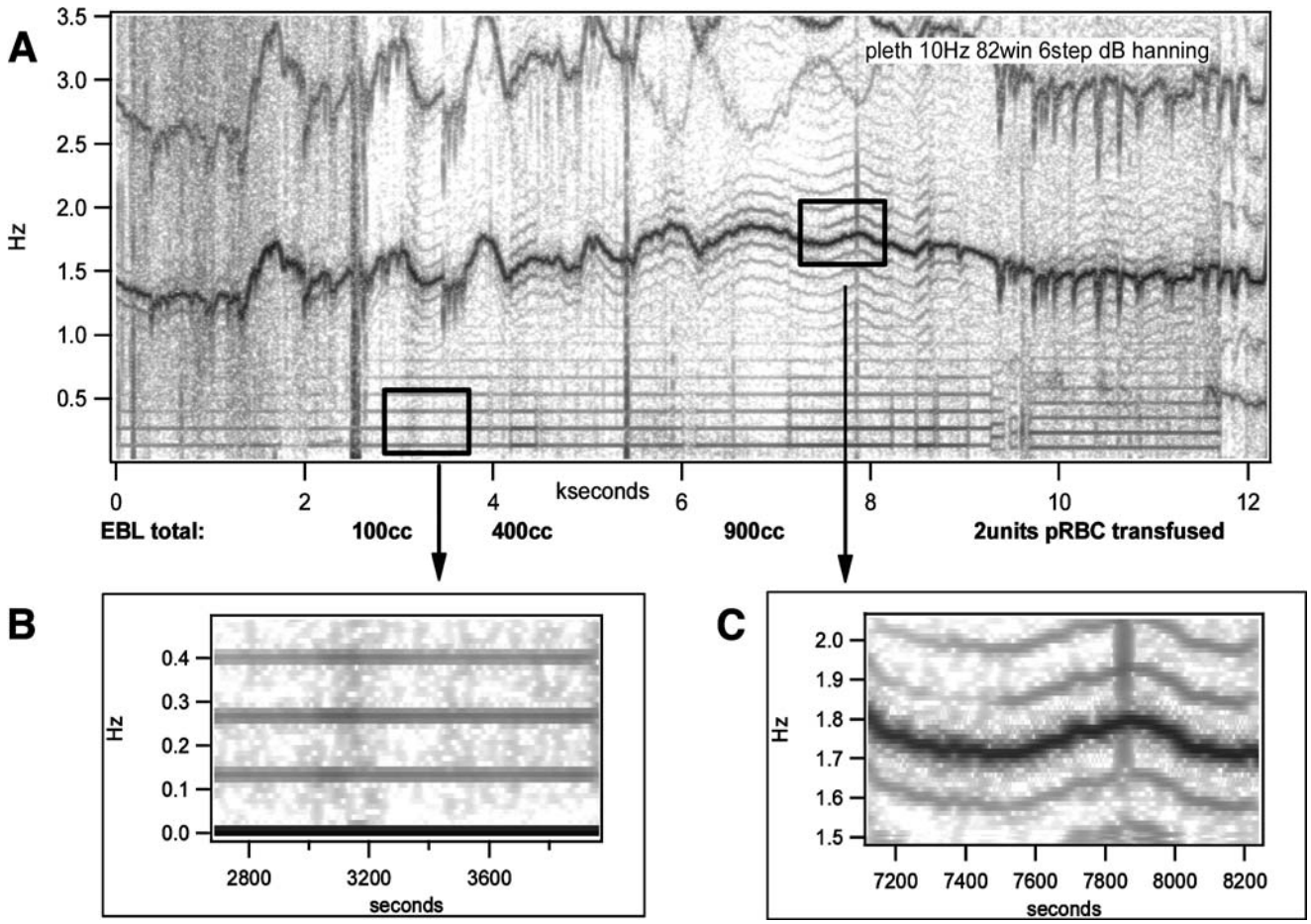

Fig. 2. (A) The joint time frequency analysis of the pulse oximeter waveform from a 3.3 hour uterine myomectomy with a total estimated blood loss of 900cc. The X-axis is time and the Y-axis is frequency. The dark band that is present in the 1.0 Hz to 1.5 Hz range corresponds to the heart rate. (B) The parallel lines running along the baseline are due to the effect of ventilation on the nonpulsatile DC component of the pulse oximeter waveform (baseline modulation). The frequency of the primary harmonic is equal to the respiratory rate (0.12 Hz = 8.3 breath/min) (C) When the blood loss is great enough, the pulse oximeter waveform develops modulation of the pulsatile component – pulse amplitude (AC modulation). The distance between the signal generated by the heart rate and the modulation once again corresponds to the respiratory rate.

There were a number of features noted in the JTFA of pulse oximeter that, while not formally studied, were nevertheless interesting (Table 2). The pulse rate is given by the frequency of the primary harmonic (i.e. 1 Hz = 60 beats/min). The thickness of the primary harmonic band corresponds to the heart rate variability (narrow ~ low heart rate variability; wide ~ high heart rate variability). The presence of venous pulsation in the plethysmographic waveform appeared to reduce the relative intensity of the primary pulse harmonic when compared with the higher pulse harmonics. Overall, in the presence of a venous pulse there was an orderly shift of power to the higher frequencies as seen in Figure 4. This occurs due to the increased complexity of the venous pulse (displaying A, C and V components) when compared to the simpler arterial pulse [13, 14].

DISCUSSION

Joint time frequency analysis allows the investigator to dissect out the underlying frequencies that make up a signal and to examine how these underlying waveforms change in response to clinical changes. In the case of the pulse oximeter, the plethysmographic waveform that is displayed is a highly processed and filtered signal but is still rich in physiologic information. As would be expected, the cardiac pulse waveform is the predominant contributor to the pulse oximeter signal. The second contributor, the respiratory component, when present can be subtle and difficult to monitor using conventional display methods. Using the frequency domain to display the same waveform however the respiratory effect is readily ascertained (Figure 2).

Table 2. Features of joint time frequency analysis of the pulse oximeter waveform

1) Isolation of the respiratory induced changes of the pulse oximeter, which may be related to intravascular blood volume
2) Separation of the pulsatile (arterial) and non-pulsatile (venous) components (A.C. & D.C.) of respiratory variability, allowing for an analysis of the impact of blood volume on overall cardiac function (pre-load vs. after-load effects)
3) Method to determine respiratory rate (both controlled and spontaneous)
4) Detection of spontaneous breathing patterns
5) Detects irregularity of heart rate
6) Allows for the determination of heart rate
7) Estimation of heart rate variability by the examination of the width of the primary pulse harmonic
8) Detection of the presence of a venous pulse in the plethysmographic waveform by analysis of the relative intensity of the primary pulse harmonic with higher pulse harmonics and an orderly shift of power to the higher frequencies.
9) Method of displaying a large volume of pulse oximeter waveform information in a small area. (Three or more hours of data collection easily fits on one page)
10) This method of analysis is resistant to isolated artifacts

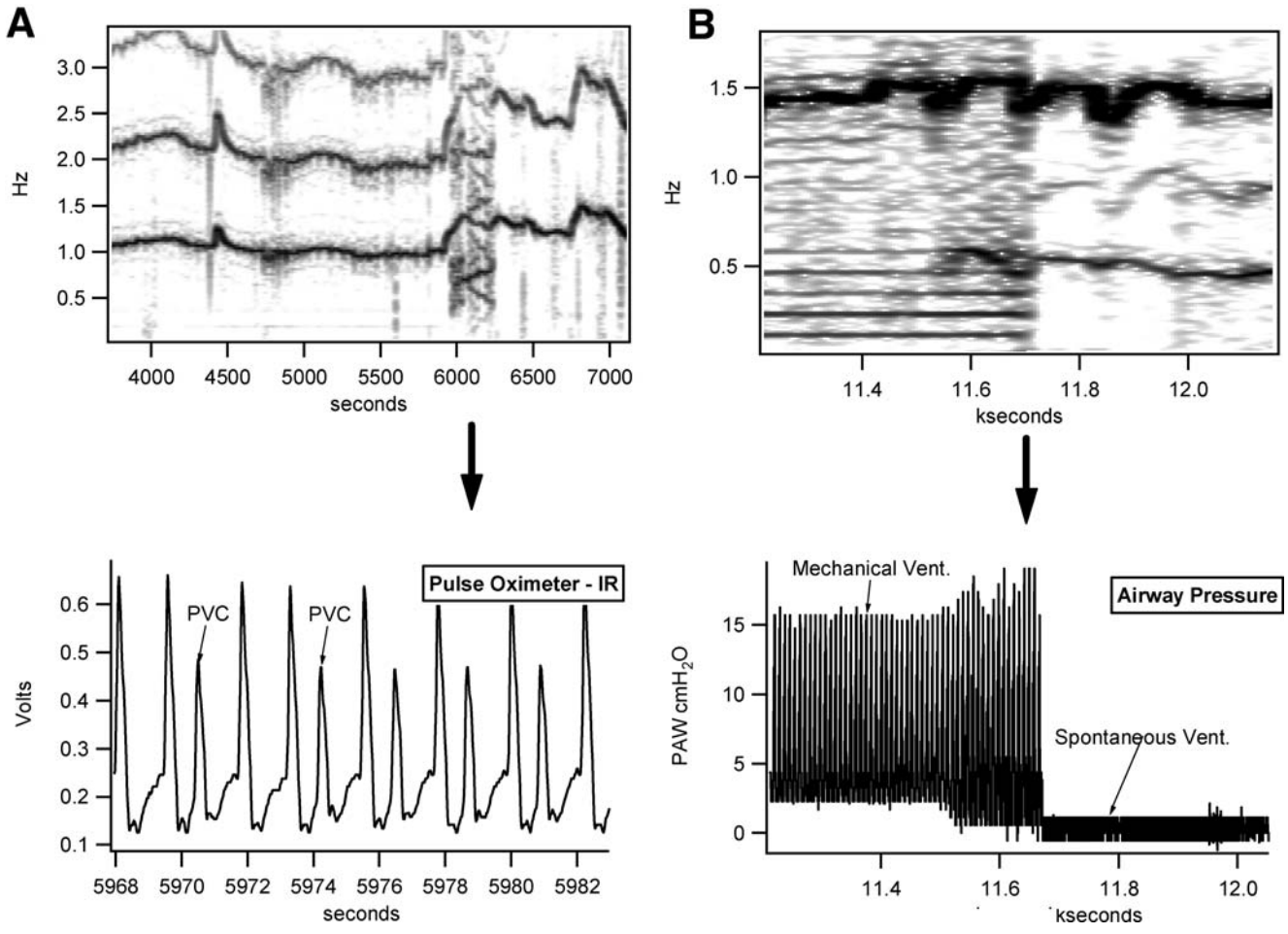

Fig. 3. (A) The effect of cardiac arrhythmia on the joint time frequency analysis of the pulse oximeter signal. (B) A demonstration of the effect of spontaneous ventilation on the joint time frequency analysis of the pulse oximeter waveform. In this case the spontaneous ventilation has a rate of 0.5 Hz = 30 breath/min.

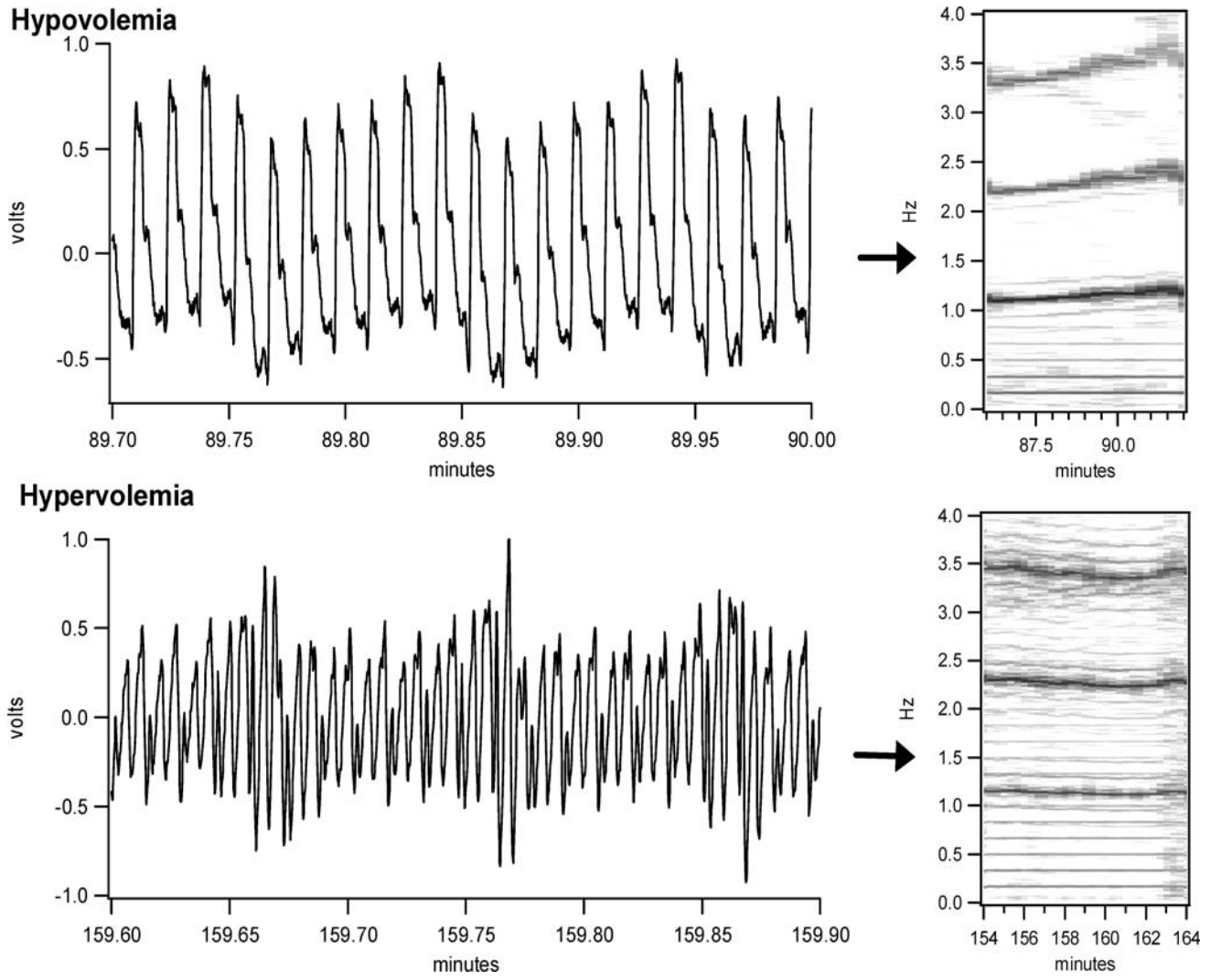

Fig. 4. The upper trace labeled Hypovolemia is the pulse oximeter waveform from a patient undergoing general anesthesia for a radial prostatectomy. The waveform shown is after approximately 400cc blood loss. It demonstrates the impact of positive pressure ventilation on the waveform. The grayscale drawing to the right is the joint time frequency analysis from the same period of time. The lower tracing labeled Hypervolemia is from the same patient after aggressive fluid replacement. It demonstrates the appearance of a venous pulse. The joint time frequency analysis of the same time (lower right) reveals an orderly shift of power to higher frequencies.

The use of Fourier analysis is limited by the need for a window of data. In the case of this study we used an 82 second sample. This has the advantage of limiting the effect of short isolated artifacts but makes the detection of individual breaths difficult, if not impossible. As with any Fourier method there is compromise between time and frequency resolution. To the methods credit though, once that compromise is made, the uniform spectral resolution allows for easy comparison of phenomenon occurring at different frequencies (i.e. AC vs. DC modulation).

The most common reason for inaccurate readings using this method appeared to be motion artifact. This can be explained by the mathematical assumption that is made when doing Fourier analysis, specifically that the complex waveform is stable and repeating. If that is not the case it then becomes impossible to use Fourier analysis to extract the modulating effects of ventilation. On the other hand, spontaneous ventilation appeared to be detectable and quantifiable as long as it had a regular pattern. (Figure 3B).

In conclusion, the increasing sophistication of digital signal processing techniques are allowing for a re-examination of the waveforms (i.e. CO_2, airway pressure, pulse waveform, etc) routinely used by clinicians. The observant clinician can get quite good at spotting subtle patterns within these complex waveforms. On the other hand, the clinician may have a difficult time quantifying their observations.

This is where techniques such as joint time frequency analysis can have an impact.

REFERENCES

1. Hertzman AB. The blood supply of various skin areas as estimated by the photoelectric plethysmograph. Am J Physiol 1938; 124: 328–340.
2. Dorlas JC, Nijboer JA. Photo-electric plethysmography as a monitoring device in anaesthesia. Application and interpretation. British Journal of Anaesthesia 1985; 57: 524–530.
3. Johansson A. Neural network for photoplethysmographic respiratory rate monitoring. Med Biol Eng Comput 2003; 41: 242–248.
4. Nakajima K, Tamura T, Miike H. Monitoring of heart and respiratory rates by photoplethysmography using a digital filtering technique. Med Eng Phys 1996; 18: 365–372.
5. Nilsson L, Johansson A, Kalman S. Monitoring of respiratory rate in postoperative care using a new photoplethysmographic technique. J Clin Monit Comput 2000; 16: 309–315.
6. Johansson A, Oberg PA. Estimation of respiratory volumes from the photoplethysmographic signal. Part I: Experimental results. Med Biol Eng Comput 1999; 37: 42–47.
7. Partridge BL. Use of pulse oximetry as a noninvasive indicator of intravascular volume status. J Clin Monit 1987; 3: 263–268.
8. Shamir M, Eidelman LA, Floman Y et al. Pulse oximetry plethysmographic waveform during changes in blood volume. Br J Anaesth 1999; 82: 178–181.
9. Rusch TL, Sankar R, Scharf JE. Signal processing methods for pulse oximetry. Comput Biol Med 1996; 26: 143–159.
10. Stack BC, Jr., Futran ND, Shohet MJ, Scharf JE. Spectral analysis of photoplethysmograms from radial forearm free flaps. Laryngoscope 1998; 108: 1329–1333.
11. Leonard P, Grubb NR, Addison PS et al. An algorithm for the detection of individual breaths from the pulse oximeter waveform. J Clin Monit Comput 2004; 18: 309–312.
12. Bland JM, Altman DG. A note on the use of the intraclass correlation coefficient in the evaluation of agreement between two methods of measurement. Computers In Biology And Medicine 1990; 20: 337–340.
13. Shelley K, Tamai D, Jablonka D et al. The impact of venous pulsation on the forehead pulse oximeter waveform as a possible source of error in SpO2 calculation. Anesthesia & Analgesia 2004; 98: S12.
14. Shelley KH, Dickstein M, Shulman SM. The detection of peripheral venous pulsation using the pulse oximeter as a plethysmograph. J Clin Monit 1993; 9: 283–287.