Anesthesiologist as Physiologist: Discussion and Examples of Clinical Waveform Analysis

Aymen A. Alian, MD

Anesthesiologists are in a unique position to observe human physiology. We encounter a wealth of clinical information expressed as waveforms and other metrics (eg, arterial pressure, electrocardiography [ECG], and photoplethysmography [PPG]) on a daily basis as we take care of our patients during the perioperative period. It is estimated that globally >500,000 surgical procedures requiring anesthesia occur everyday.[1] These surgical procedures often put stress on the cardiovascular system. With rare exceptions,[2] the clinical waveforms (eg, arterial pressure, ECG, and PPG) used to guide therapy are discarded within seconds of their generation. Even the introduction of AIMS (automated anesthesia records) systems, and the recent emphasis on "big data" exploration, has failed to recognize the value of waveform analysis done after a procedure.

This article examines human cardiovascular physiology under stress. It introduces different methods of clinical waveform analysis during different clinical scenarios (eg, postpartum hemorrhage requiring massive blood transfusion and pheochromocytoma resection requiring vasodilator use). The waveforms come from standard clinical monitors, which have both advantages (broad applicability) and disadvantages (proprietary filters).[3] The aim of this analysis is to improve the understanding of the physiology that generates these peripheral waveforms. This improved understanding will hopefully allow for the development of new methods of monitoring. In turn, these new clinical monitors could ideally be used to guide pharmacologic and fluid therapy with an ultimate goal of improving patients' outcome. The hope is that this article will make it apparent that the clinical anesthesiologist has the potential of becoming an effective frontline physiologist. As anesthesiologists, we often observe and treat people under conditions (eg, hypovolemic shock) that can test their physiological limits. It would be unethical to expose people to these extreme conditions as part of a controlled experiment. By the same token, some might consider it unacceptable to not observe and learn from these events during surgical procedures. To quote 1 of the founding fathers of modern medicine, William Osler (1849–1919): "Listen to your patient, he is telling you the diagnosis." To paraphrase within the context of this article: "Observe your patient, he is telling you his physiology."

Arterial and PPG Waveform Analysis

Arterial waveform analysis has provided the anesthesiologist and intensivist with a relatively noninvasive means of estimating ventricular contractility and stroke volume on a beat-to-beat basis. Furthermore, the use of a generalized transfer function that can help in the estimation of central arterial pressures (eg, aortic) can be produced from peripheral (eg, brachial and radial) tracings, thus helping in the development of several continuous cardiac output monitors.[4] PPG offers the clinician more than just a determination of oxygenation status and heart rate.

In mechanically ventilated adults, functional hemodynamic parameters derived from arterial blood pressure (such as pulse pressure variability [PPV], systolic pressure variability, stroke volume variability) and from the pulse oximeter waveform (changes in the amplitude of pulse oximeter plethysmograph [ΔPOP] and pleth variability index) have been shown to be useful guides for fluid administration in adults. The prediction of fluid responsiveness is crucial for adequate fluid management and goal-oriented hemodynamic optimization because it has been associated with decreases in the length of hospital stay and morbidity.[5–13]

Baseline modulations of the PPG, which occur at the respiratory frequency, are increased during hypovolemia and are related to venous blood movement.[14–18] One method of isolating respiratory-induced oscillations of the PPG is frequency analysis utilizing the fast Fourier transform (FFT) algorithm. The resulting Fourier transform shows the spectrum of frequencies from the PPG waveform plotted as amplitude density against frequency. This method of analysis can also be used to determine heart rate, respiratory rate, and regional venous saturation noninvasively.[19–23] Although hypovolemia is 1 of the leading causes of increased respiratory-induced modulations of the arterial and PPG waveform, one should consider the potential for other causes such as cardiac tamponade, asthma, high airway pressures, and tension pneumothorax.[24]

Peripheral Venous Pressure Monitoring

The vast majority of hospitalized patients have a peripheral venous line. It is placed to allow fluids and medications to be given directly into the circulatory system. In the context

From the Department of Anesthesiology, Yale University School of Medicine, New Haven, Connecticut.

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Address correspondence to Aymen A. Alian, MD, Department of Anesthesiology, Yale University School of Medicine, 333 Cedar St, P.O. Box 208051, New Haven, CT 06520. Address e-mail to aymen.alian@yale.edu.

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of avoiding additional invasive intraoperative monitoring, peripheral venous pressure (PVP) monitoring has been tested as an alternative to central venous pressure (CVP) monitoring. There has been a strong correlation between PVP and CVP during various types of surgery, even during laparoscopic surgery.[25–29]

PVP frequency analysis via a standard intravenous catheter may provide a low-cost, minimally invasive monitoring solution for monitoring and resuscitating patients with perioperative hemorrhage. Frequency analysis of the PVP demonstrated a greater sensitivity for detecting early hypovolemia (as little as 6% estimated blood loss) compared with standard monitoring. There was a reduction in modulation of PVP waveforms at the cardiac frequency.[30,31] Similar findings have been noted during lower body negative pressure-induced hypovolemia in healthy volunteers. There was a significant reduction in the modulation of PVP waveform at the cardiac frequency with −30 mmHg lower body negative pressure, whereas there was no significance change in the arterial blood pressure values.[32]

PPG and Vascular Tone

Pulse pressure, a surrogate marker of increased arterial stiffness, is a powerful predictor of cardiovascular events.[33] It has been observed that the amplitude of the reflected arterial waveform will increase as the large arteries stiffen with age or disease process.[34] The PPG waveform comprises a pulsatile (AC) waveform attributed to cardiac synchronous changes in the blood volume with each heartbeat.

PPG reflection index (RI) is derived as a ratio of pulse inflection peak amplitude (second peak) over the pulse maximum amplitude. RI can provide a window into arterial compliance and vascular age. RI has shown to be a noninvasive indicator for vascular assessment. Although PPG augmentation index (AI) is derived as a ratio of the amplitude of the second peak and the amplitude of pulse maximum peak, PPG AI can be used as a measure of arterial stiffness. PPG AI has proven to be a noninvasive indicator for vascular assessments.[35,36]

Finger arterial compliance is another interesting parameter that has been studied on a beat-to-beat basis by using a digital arterial pressure (Finapres monitor) waveform and PPG volume waveform, where dynamic finger arterial compliance is defined as the volume change produced by a unit change in a pulsatile pressure. Finger arterial compliance is considered to be a useful noninvasive indicator of peripheral vascular compliance in humans.[37–39] Peripheral pressure–volume loops analysis is another interesting parameter. These loops are created by plotting the pulse oximeter waveform versus the blood pressure waveform. The authors of this article emphasize that this method of analysis may provide a useful measure of vascular compliance.[40]

THE APPROACH TO DATA COLLECTION AND ANALYSIS

Data Collection

With institutional review board approval, arterial blood pressure (BP), finger PPG, and PVP, and airway pressure were recorded at 100 Hz from operating room clinical monitors (GE, Fairfield, CT) with a data acquisition system (collect 5/S; GE). In addition to the collection of the waveforms, there were trained observers in the operating rooms recording events in real time with an emphasis on fluid and pharmaceutical administration. These observers helped to add accuracy to both the timing and amounts of fluid and pharmaceuticals given during these events beyond what is obtainable from the typical anesthesia record. Afterward, the waveforms were analyzed using both time-domain and frequency-domain digital signal processing techniques.

To better understand the data contained in these waveforms, seen by anesthesiologists everyday, several different methods of analysis were utilized. The methods included frequency analysis utilizing FFT[41] and time domain analysis[42] and combining waveforms using X–Y plots (loops).[40]

Waveform Channel Preparation

We used LabChart 7.37 (ADInstruments, Boulder, CO) to analyze the waveforms as shown in Figure 1I, A–D. As part of the time domain analysis, PPG low-pass, PPG high-pass, arterial low-pass, and arterial high-pass waveforms were generated by applying low- (<0.5 Hz) and high-pass filters (>0.5 Hz) to finger PPG and arterial BP waveforms, respectively, as shown in Figure 1I, E, F, H, and I. This initial step was useful in separating cardiac from respiratory modulations in these waveforms. We utilized the cyclic measurement function of LabChart 7.37 (ADInstruments) to determine the amplitudes of the finger PPG (pulse height) and arterial BP (pulse pressure) waveforms. These amplitude measurements could then, in turn, be used to determine the degree of respiratory (ventilator)-induced modulations of finger PPG and arterial BP (pulse pressure modulation) as shown in Figure 1I, G and J).
The following is a summary of these channels:

(A) Clinical monitor arterial BP waveform
(B) Clinical monitor PPG waveform
(C) Clinical monitor airway pressure waveform
(D) Clinical monitor PVP waveform
(E) Low-pass filter of PPG (PPG DC): constructed by using a low-pass (<0.5 Hz) filter on the PPG raw data (B). It is the modulation of PPG baseline because of respiration (occurring at ventilator frequency).
(F) High-pass filter of PPG (PPG AC): constructed by using a high-pass (>0.5 Hz) filter on the PPG raw data (B). The modulations at these frequencies are assumed to be from arterial blood.
(G) PPG amplitude height modulation: This is generated by measuring the pulse heights from the high-pass filter PPG waveform (F). The result is PPG pulse height (AC) modulation because of respiration (occurring at ventilator frequency).
(H) Low-pass filter of arterial BP is used to determine mean arterial pressure (MAP) modulation. It is constructed by using a low-pass (<0.5 Hz) filter on the arterial BP raw data (A). This mathematically corresponds to the PPG DC and is therefore referred to as the arterial pressure DC.
(I) High-pass filter of arterial BP is used to isolate the arterial pulse. It is constructed by using a high-pass (>0.5 Hz) filter on the arterial BP raw data (A). This mathematically corresponds to the PPG AC and is therefore referred to as the arterial pressure AC.

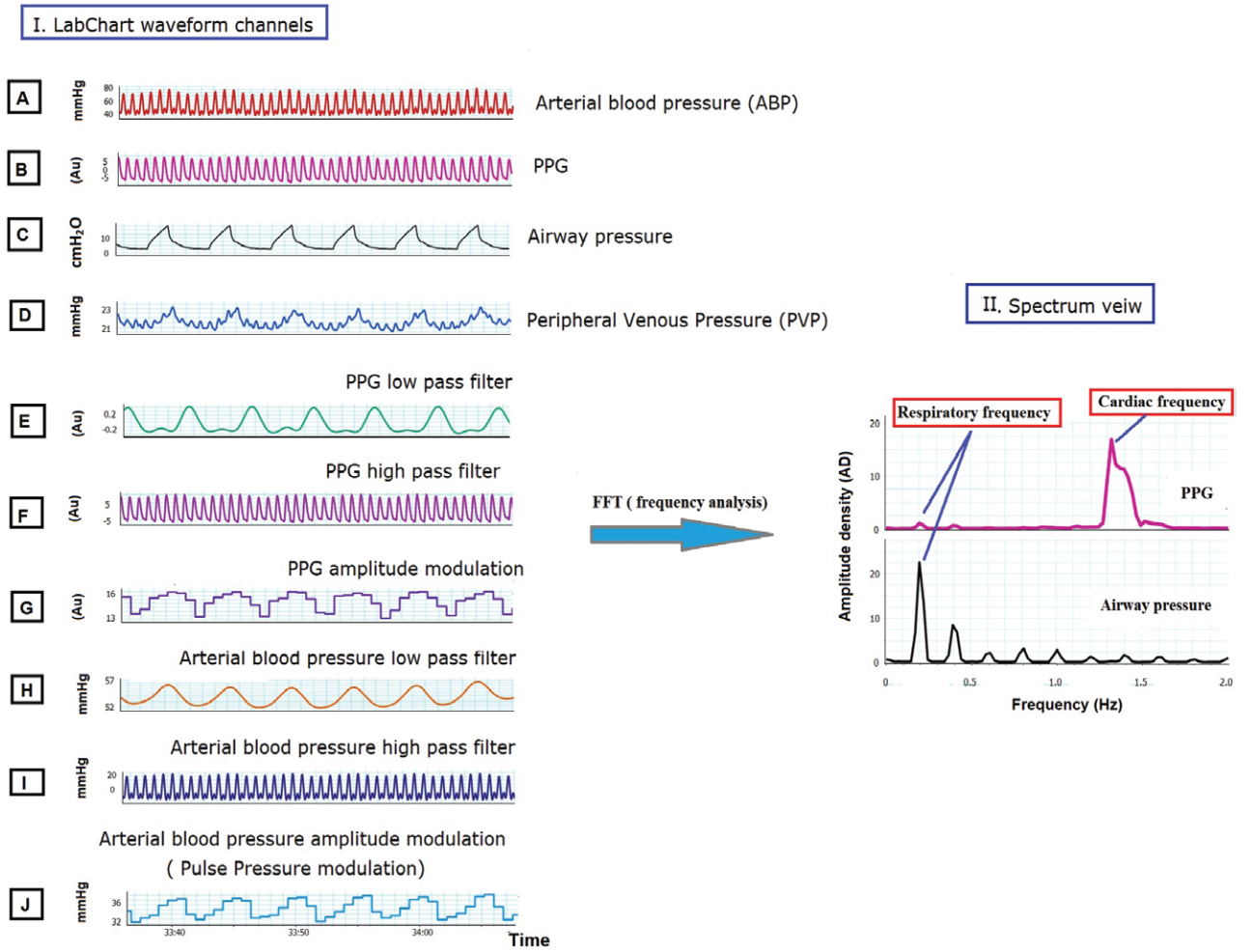


Figure 1. Waveform channel preparation and frequency analysis. I, Represents the waveform channels that are generated by LabChart 7.37 (ADInstruments). A, B, C, and D represent arterial blood pressure (BP), finger photoplethysmography (PPG), airway pressure, and peripheral venous pressure waveforms. The effect of low-pass (<0.5 Hz) and high-pass filters (>0.5 Hz) on PPG and arterial BP waveforms is shown in channels E, F, H, and I, respectively. Amplitude modulation of finger PPG and arterial BP (pulse pressure modulation) waveforms is shown in channels G and J. II, Shows PPG waveform spectrum view, which demonstrates respiratory and cardiac frequency modulations. The peak of respiratory modulation of the PPG is locked at the peak of the airway pressure waveform (0.195 Hz).

(J) Arterial pulse pressure modulation is generated by measuring pulse pressure modulation from the high pass filter arterial BP waveform (I). The result is arterial pulse pressure modulation because of respiration (occurring at ventilator frequency).

Frequency-Domain Analysis Method

In frequency-domain analysis of the waveforms, we used FFT with the spectrum view settings as shown in Figure 1II (spectrum, 4K [40 seconds at 100 Hz] Hamming window, amplitude density, 93.75% window overlap) over 3-minute windows utilizing LabChart 7.37 (ADInstruments). Modulations of the arterial BP and PPG waveforms at respiratory and cardiac pulse frequency were determined (where respiratory frequency was defined as the same frequency as the airway pressure waveform and cardiac pulse frequency was defined as the highest peak between 1 and 2.5 Hz). The strength of the waveform's modulations was measured as amplitude density, as shown in Figure 1III. Frequency analysis of arterial BP and PPG waveforms is shown in Figure 2 and includes the following variables:

1. *Arterial pressure DC*: corresponds to the MAP modulation at the respiratory frequency.
2. *Cardiac pulse height*: corresponds to the amplitude density of the arterial pressure waveform at the cardiac pulse frequency; it represents the average pulse pressure.
3. *Arterial pressure AC*: corresponds to the arterial pulse pressure modulation at the respiratory frequency and to the pulse pressure variability as measured using time domain analysis.
4. *Arterial pressure DC % and AC%*: used to normalize the arterial pressure DC and AC values, respectively. These are derived by dividing the arterial pressure DC and arterial pressure AC, respectively, by arterial cardiac pulse pressure.
   - Arterial pressure DC% is the percent of MAP modulation.

$$Arterial pressure\ DC\% = 100 \times \frac{Arterial pressure\ DC}{Arterial\ cardiac\ pulse\ height}$$

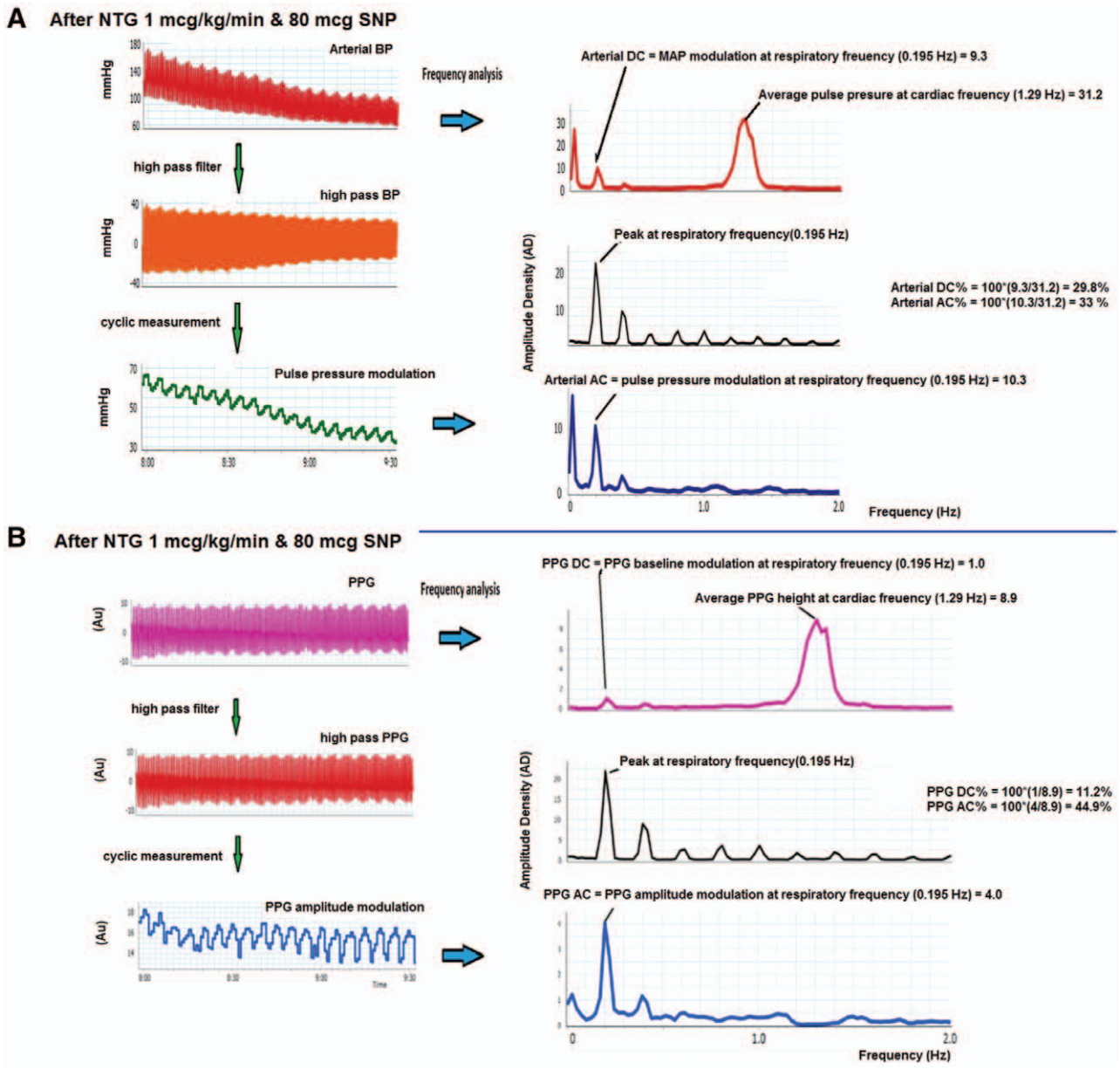


Figure 2. Arterial, photoplethysmography (PPG) waveforms, and frequency analysis after administration of a vasodilator. Effect of nitroglycerine (NTG) and sodium nitroprusside (SNP) dose on (A) arterial blood pressure (BP) waveform and (B) PPG waveform. A, On the left side (from top down): arterial BP, high-pass filter BP, and pulse pressure modulation. On the right side: the frequency analysis spectrum view of arterial BP (from above down); arterial DC (arterial baseline modulation [mean arterial pressure {MAP} modulation]) and arterial AC: (pulse pressure modulation) with respiratory frequency and average pulse pressure at cardiac pulse frequency along with the calculated arterial DC% and AC%. B, On the left side (from top down): PPG, high-pass filter PPG, and PPG amplitude modulation. On the right side: the frequency analysis spectrum view of PPG (from above down): PPG baseline modulation (PPG DC) and (PPG AC) with respiratory frequency and the average PPG amplitude at cardiac pulse frequency with examples of PPG DC% and PPG AC% calculation.

- Arterial pressure AC% is the percent of pulse pressure modulation (PPV%)

$$Arterial\ pressure\ AC\% = 100 \times \frac{Arterial\ pressure\ AC}{Arterial\ cardiac\ pulse\ height}$$

5. *PPG DC*: corresponds to the baseline modulation of the pulse oximeter waveform at the respiratory frequency.
6. *PPG AC:* corresponds to the pulse oximeter waveform amplitude modulation at the respiratory frequency.
7. *Cardiac pulse height*: corresponds to the PPG amplitude at the cardiac pulse frequency. This can be thought of as the PPG height.
8. *PPG DC % and PPG AC%*: used to normalize the PPG DC and PPG AC values to compare them between patients. These are derived by dividing PPG DC and PPG AC, respectively, by the PPG cardiac pulse height. PPG AC% corresponds to ΔPOP (in time domain analysis) by other authors.[5]

$$PPG\,DC\,\% = 100 \times \frac{PPG\,DC}{PPG\,cardiac\,pulse\,height}$$

$$PPG\,AC\,\% = 100 \times \frac{PPG\,AC}{PPG\,cardiac\,pulse\,height}$$

Time-Domain Analysis of Waveforms

The time-domain analysis of the PPG, or the arterial BP waveform, includes waveform peak, height, area (area under curve [AUC"]), width 50 (time interval at 50% of the height; Max slope) maximum slope over the whole peak, (Min slope) minimum slope over the whole peak as well as dicrotic notch (demonstrated in Figure 3). The pressure–volume (P–V) loop, at the periphery, results from the combination of arterial BP and finger PPG waveforms as shown in Figure 4. Calculating the slope, area of the loop, and the position of the dicrotic notch may provide important information regarding the cardiovascular system.

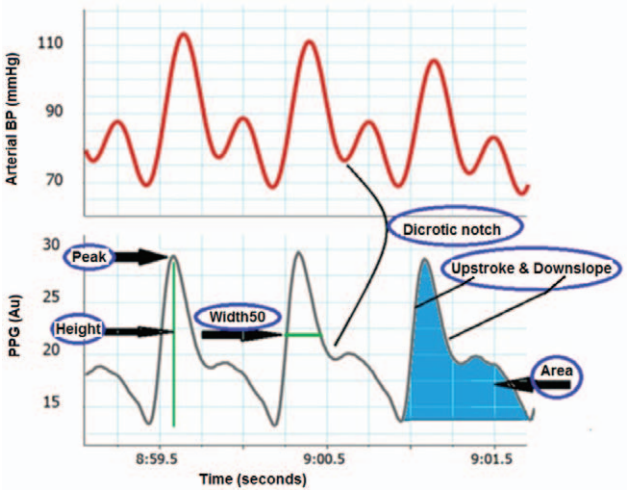


Figure 3. Time-domain analysis of waveforms. Time-domain analysis includes waveform peak, height, area (area under curve [AUC]), width 50 (time interval at 50% of the height), dicrotic notch, (Upstroke and Downslope) maximum slope and minimum slope over the whole peak respectively.

ANALYSIS EXAMPLES

Case 1: Hemorrhage (Postpartum Hemorrhage With Massive Blood Transfusion)

A 24-year-old, 78-kg woman G2P1 received 2 units of packed red blood cells, 2 fresh frozen plasma, and 2.5 L of crystalloid during a cesarean delivery for placenta previa with an estimated blood loss of 2500 ml. The postoperative course was complicated with postpartum hemorrhage of an additional estimated blood loss of 700 mL. The patient received 2 units of packed red blood cells in the recovery room, and she was rushed to the operating room for a hysterectomy under general anesthesia after attempts failed to control postpartum hemorrhage. The case was divided into 5 distinct events to show the changes in vital signs as well as in PPG and in arterial and peripheral venous pressure waveforms.

PPG and Arterial Waveforms Analysis

Time-Domain Analysis of Finger PPG and Arterial Waveforms. ***Although the MAP readings were stable, there were changes in PPG and arterial waveforms as shown in Figure 5.***

- In events 1 and 2, there was continuous bleeding despite fluid resuscitation and there were significant changes in both the PPG and arterial waveforms showing respiratory modulation.
- In events 3 and 4, there was sufficient fluid resuscitation. This led to the loss of respiratory variability of the arterial and finger PPG waveforms.

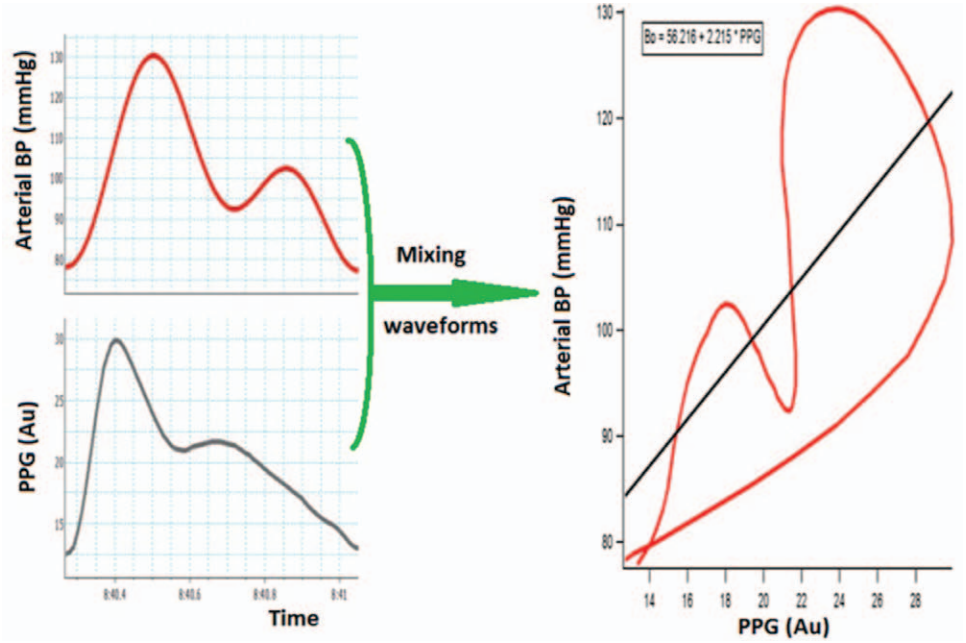


Figure 4. Mixing of arterial and photoplethysmography (PPG) waveforms to create P–V loop. On the left side: a single arterial blood pressure (BP) and finger PPG waveforms. On the right side: the pressure–volume (P–V) loop at the periphery as a result of plotting the waveforms together.

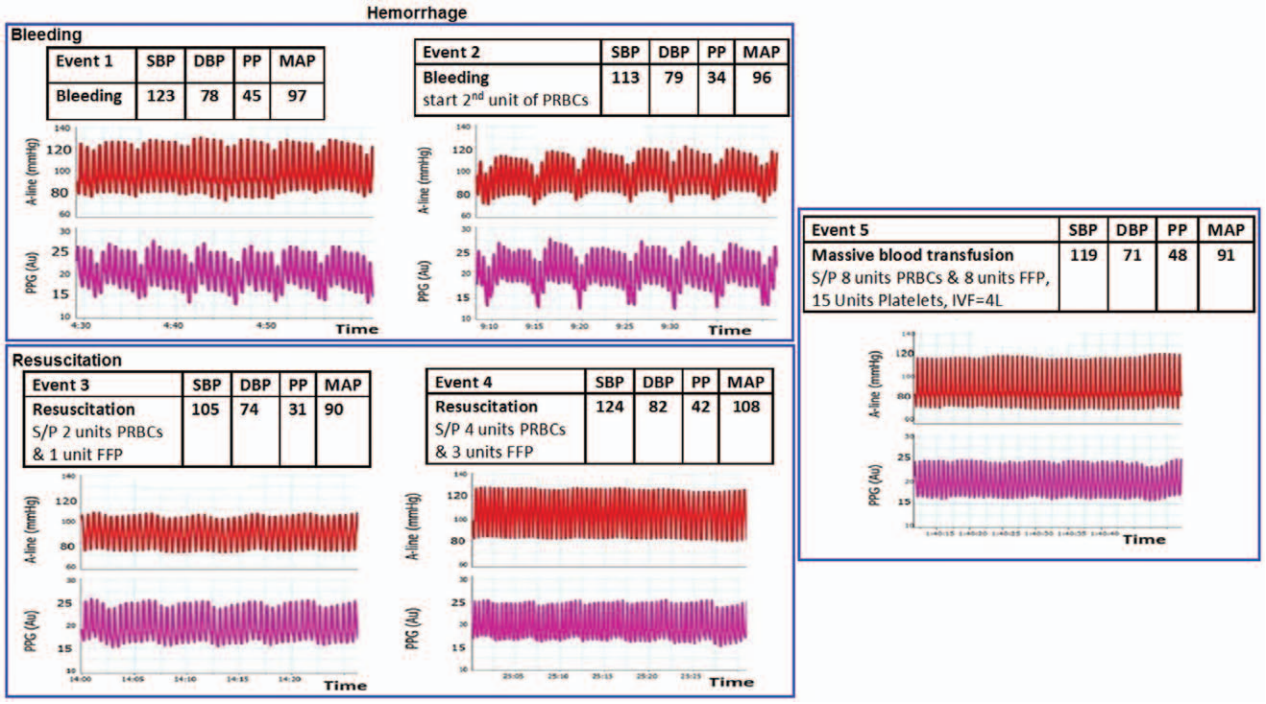


Figure 5. Changes in vital signs, arterial blood pressure (BP), and photoplethysmography (PPG) waveforms during different events of hemorrhage. The average values of blood pressure together with arterial and PPG waveforms during different events of the postpartum hemorrhage case. SBP, systolic blood pressure; DBP, diastolic blood pressure; PP, pulse pressure; MAP, mean blood pressure. Events 1 and 2 highlight the bleeding effect even in the face of resuscitation. Events 3 and 4 show the impact of resuscitation on the waveform. Event 5 showed the effect of the massive blood transfusion. Notice the changes in the PPG and arterial BP waveforms in face of stable BP readings.

- In event 5, changes showed in the PPG and arterial waveforms after massive blood transfusion.

Frequency Analysis of Finger PPG and Arterial Waveforms. PPG waveforms, together with corresponding frequency analysis during different events, are shown in Figure 6A. The arterial waveform, during different events as well as corresponding frequency analysis, is shown in Figure 6B. The changes in PPG DC% exceed the changes seen in the PPG AC%, arterial DC%, and arterial AC% during the bleeding phases of the case, as shown in Figure 6C. The respiratory modulations of the PPG and the arterial waveforms were lost with volume replacement. This leads to the hypothesis that the presence of PPG and arterial modulation reflects hypovolemia, whereas their loss reflects adequate fluid resuscitation.

Now, the question is, "What happens during a massive blood transfusion event?" (ie, can we reduce the risk of fluid overload utilizing peripheral waveforms?). This unexplored area of investigation ultimately may be the greatest contribution from this type of analysis, in effect, telling the anesthesiologist when to stop giving fluids.

Peripheral Venous Waveform Analysis

The placement of an intravenous line is a standard practice of patient care during fluid resuscitation. Monitoring of the intravenous line (PVP) may provide clinicians with valuable information.

Time-Domain Analysis of PVP Waveforms. During hypovolemia ([event 2] of Figure 7A), the average PVP was 27 mmHg and respiratory modulations were 4–6 mmHg. However, during resuscitation ([event 4] of Figure 7A), the average PVP was 29 mmHg with respiratory modulations of 1–2 mmHg.

Frequency Analysis of PVP Waveforms. During the bleeding phase, the frequency analysis of the PVP waveforms showed an increase of signal strength (as measured by amplitude density) at the respiratory frequency with only a small signal at the cardiac frequency (Figure 7B, event 2). During resuscitation, there was a reduction in the signal strength of the PVP at the respiratory frequency, whereas the signal at the cardiac frequency did not change (as shown in Figure 7B, events 3 and 4). Finally, after massive blood transfusion, there was an increase in the AD of the cardiac modulations as well as a marked reduction of the respiratory frequency (as shown in Figure 7B, events 5 and 6). One interpretation could be that, with hypovolemia, there was significant respiratory modulation of the venous pressure, whereas during fluid overload, cardiac modulation (with CVP transmission down the vein) would become the predominant signal. Clearly, from a single case, one would not propose changing clinical practice. On the

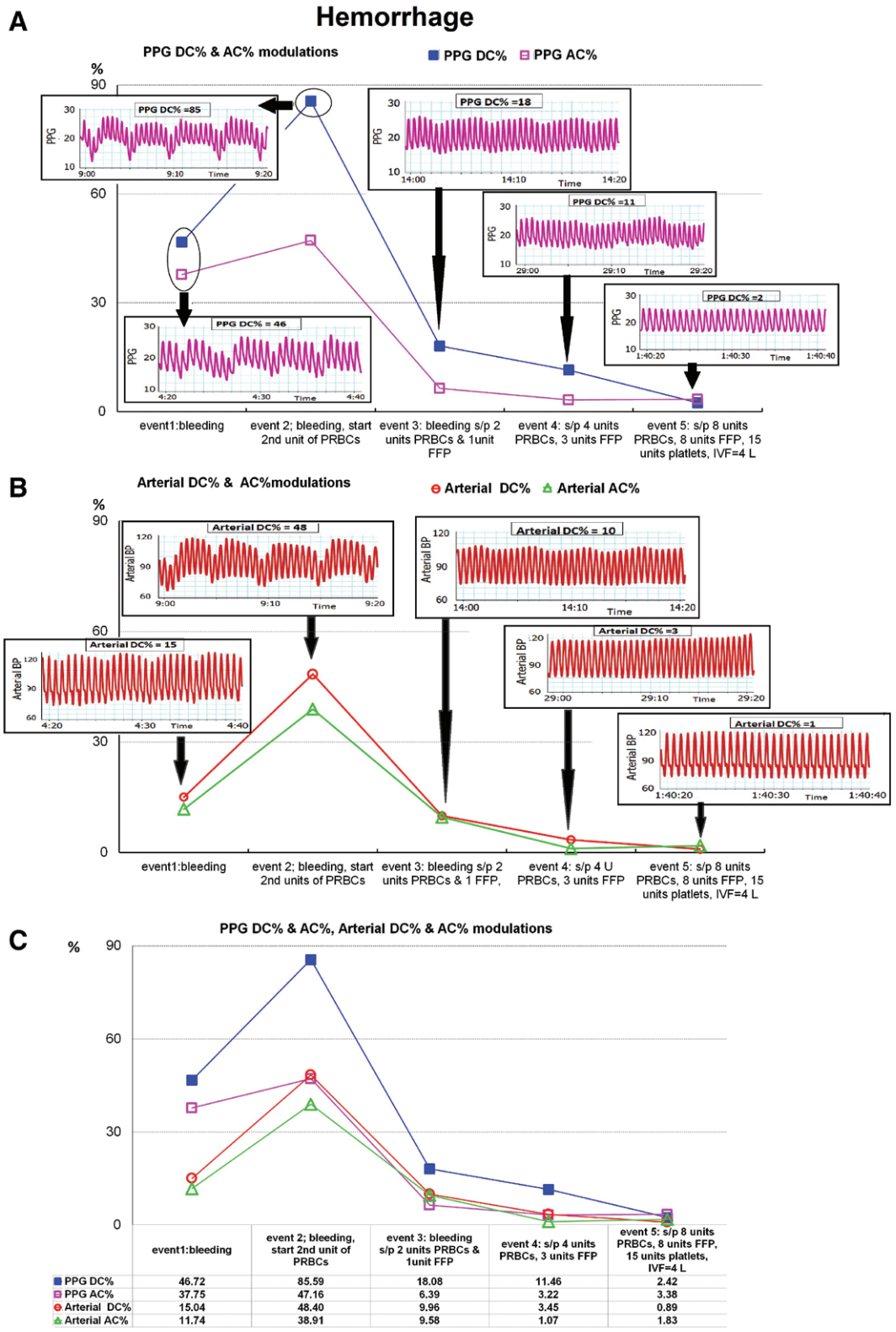
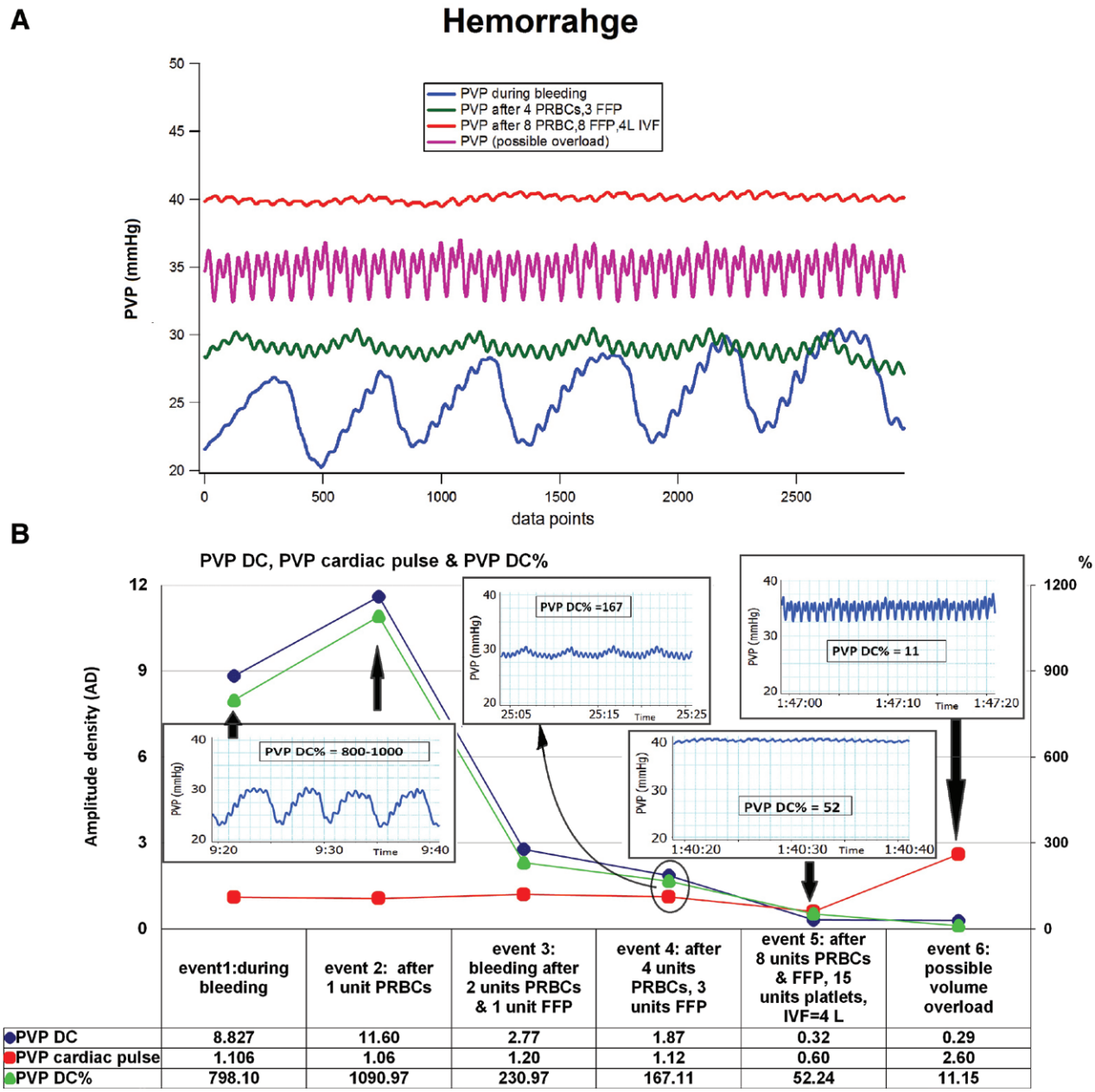


Figure 6. Frequency analysis of photoplethysmography (PPG) and arterial blood pressure (BP) waveforms during different stages of hemorrhage. Upper panel (A) shows changes in PPG waveforms as well as PPG DC% and PPG AC% respiratory modulations during different stages of the postpartum hemorrhage case. Middle panel (B) shows the arterial BP waveforms as well as arterial DC% and arterial AC% modulations during different stages of postpartum hemorrhage with the massive blood transfusion case. Lower panel (C) shows changes in PPG and arterial AC% and arterial DC% modulations. FFP indicates fresh frozen plasma; IVF, intravenous fluid; PRBCs, packed red blood cells.



	event1:during bleeding	event 2: after 1 unit PRBCs	event 3: bleeding after 2 units PRBCs & 1 unit FFP	event 4: after 4 units PRBCs, 3 units FFP	event 5: after 8 units PRBCs & FFP, 15 units platlets, IVF=4 L	event 6: possible volume overload
PVP DC	8.827	11.60	2.77	1.87	0.32	0.29
PVP cardiac pulse	1.106	1.06	1.20	1.12	0.60	2.60
PVP DC%	798.10	1090.97	230.97	167.11	52.24	11.15

Figure 7. Changes in peripheral venous pressure (PVP) waveforms during hemorrhage and resuscitation; time domain and frequency analysis. PVP waveforms during different events of the postpartum hemorrhage case with massive blood transfusion are shown in the upper panel (A). The amplitude density of PVP waveform frequency analysis during different degrees of hemorrhage is demonstrated in the lower panel (B). PVP DC is amplitude density of PVP at the respiratory frequency, whereas PVP at the cardiac pulse is the amplitude density at the cardiac pulse. PVP DC% is the ratio of ([PVP DC/PVP cardiac pulse]*100). FFP indicates fresh frozen plasma; IVF, intravenous fluid; PRBCs, packed red blood cells.

other hand, this might be the clue to a new technique of minimally invasive clinical monitoring.

Case 2: Hyperdynamic Circulation

A 37-year-old man was scheduled for laparoscopic removal of a left adrenal mass (pheochromocytoma). The patient's urinary and plasma metanephrines were high: 24-hour urine metanephrines were 17,000 (N <190 μg/24 h), normetanephrine 11,000 (N <482 μg/24 h) with a total of 28,000 (N <695 μg/24 h). Plasma-free metanephrine was 3027 (N <75 pg/mL), and plasma-free normetanephrine was 3024 (N <148 pg/mL) for a total of 6051 (N <205 pg/mL). Multiple doses of vasodilators (eg, sodium nitroprusside or nitroglycerine) were used to control BP during this pheochromocytoma case.

Frequency Analysis of PPG and Arterial Waveforms. There were changes in the PPG and arterial waveforms with the use of vasodilators. Both PPG AC% and PPG DC% were increased with the use of vasodilators (as shown in Figure 8A). The increase in the PPG AC% was more than the increase in the PPG DC%, and it is our observation that this magnitude was the opposite of what we noticed during hypovolemia (Figure 6A). The relation between the changes in PPG (as demonstrated by PPG AC%) and arterial

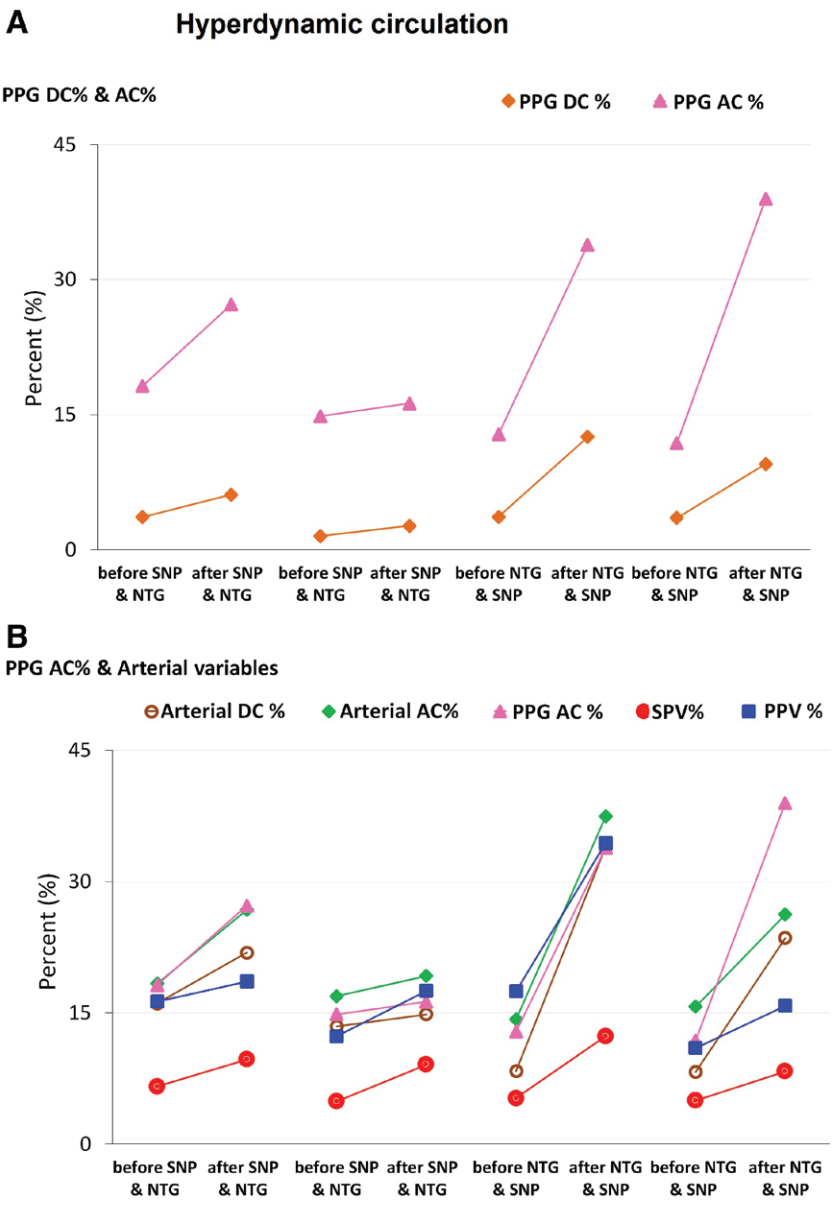


Figure 8. Effect of vasodilators on PPG AC%, PPG DC%, and arterial variables. The upper panel (A) shows the effect of multiple doses of vasodilators on PPG AC% and PPG DC%. The lower panel (B) shows the changes in PPG AC%, arterial AC%, arterial DC%, SPV%, and PPV%. The changes in PPG AC% are comparable, and even superior, to the arterial derived variables. NTG indicates nitroglycerine; PPV%, pulse pressure variability; SNP, sodium nitroprusside; SPV%, systolic pressure variability.

BP variables (as demonstrated by systolic blood pressure [SBP] variability, PPV, arterial DC%, and arterial AC%) is shown in Figure 8B. The direction and the magnitude of the changes between the PPG AC% (synonymous to delta POP) and arterial AC% (pulse pressure variability) are shown in Figure 8B.

Peripheral Pressure–Volume Loops. The combination of arterial BP and finger PPG waveforms will result in a P–V loop at the periphery before (A) and after (B) vasodilator administration (Figure 9). The P–V loop slope was reduced (from 7.4 to 1.9) and the P–V loop area was also reduced (from 488 to 282) after administering the drug (Figure 9).When we aligned the foot of the finger PPG waveform with the arterial BP waveform, it resulted in a P–V loop. An important observation is that the blood volume builds first in the periphery. Once the blood volume reaches a peak, then the pressure begins to increase. This may be thought of as being analogous to the filling of a garden hose. The volume of water first enters the hose, and only after it is full does the hose become pressurized. This peripheral loop can be thought of as being the mirror image of the classical ventricular pressure–volume loop, where pressure increases first and is followed by a reduction in volume as blood volume moves forward into circulation.

Dicrotic Notch. Using the vasodilators resulted in changes in the dicrotic notch value and position. Vasodilators will dilate muscular arteries and reduce both the arterial augmented

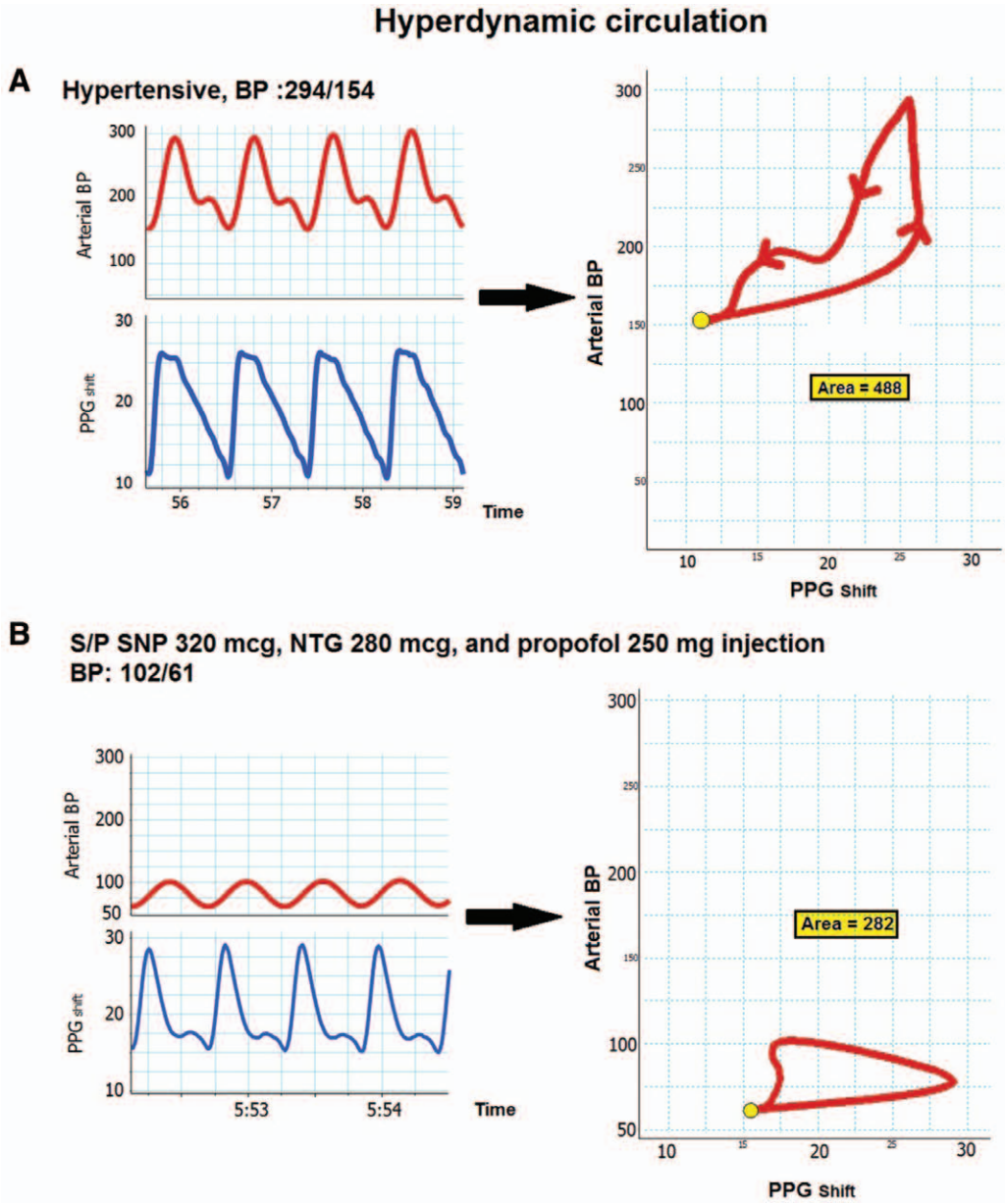


Figure 9. Pressure–volume (P–V) loop. The pressure–volume before and after vasodilator administration. Upper panel (A): the patient is hypertensive with arterial BP = 300 mmHg; the PPG dicrotic notch is close to the peak of PPG waveforms. The area of the P–V loop is 488. Lower panel (B): after the use of multiple doses of vasodilators (sodium nitroprusside [SNP], nitroglycerine [NTG]): blood pressure drop to 100 mmHg with loss of the arterial dicrotic notch, whereas PPG dicrotic notch moved closer to the baseline with the use of vasodilators. The area of the P–V loop decreased to 282. To create the P–V loop, the foot of PPG and arterial waveforms were matched.

index and the diastolic augmented index (Figure 10A). This effect will reduce the left ventricular load.[43] Elevated BP is associated with a high value of the dicrotic notch (close to PPG peak), whereas with hypotension, the dicrotic notch of the PPG waveform is closer to the baseline. Similar to the arterial augmented index, the PPG augmented index can be calculated from the following equation and is shown in Figure 10B:

$$PPG\ Augmented\ Index = \frac{(PPG\ dicroticnotch\ height - PPG\ baseline)}{PPG\ peak - PPG\ baseline} \times 100$$

Thus, the PPG augmented index is high (80%–90%) during hypertension, whereas with hypotension, the PPG augmented index is low. Correlation of SBP with PPG augmented index and arterial compliance was 0.92 and −0.92, respectively, over the wide range of BPs from 300 to 75 mmHg as shown in Figure 11.

Local Arterial Compliance. Compliance is the ratio of change in volume over change in pressure.

Time-Domain Analysis. The change in blood volume with each heartbeat can be determined by the PPG waveform amplitude, whereas pulse pressure provides a measure of

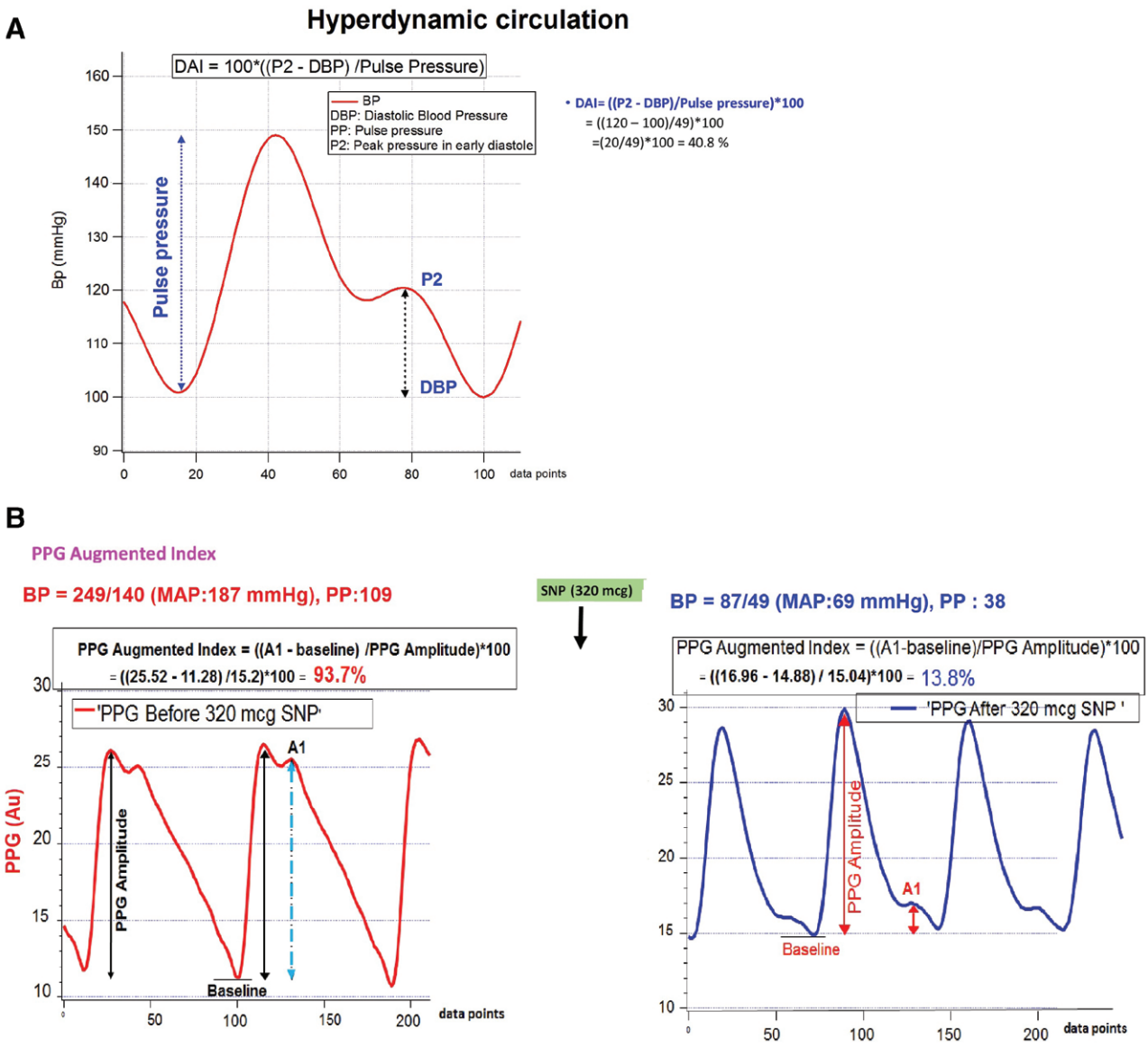


Figure 10. PPG augmented index. The upper panel (A) shows arterial diastolic augmented index calculation where P2 = the pressure at the peak of dicrotic notch and DBP, diastolic BP. The lower panel (B) shows PPG augmented index calculation before and after the effect of 320 μg of sodium nitroprusside (SNP) where PPG amplitude = (PPG peak value − PPG baseline) and A2 = (PPG value at the peak after dicrotic notch − PPG baseline). BP indicates blood pressure; MAP, mean arterial BP; PP, pulse pressure.

pressure change in the vascular bed. An index of the local arterial compliance can be calculated by dividing the PPG amplitude by the pulse pressure.[40]

$$Local\ Arterial\ Compliance = \frac{PPG\ amplitude}{Arterial\ pulse\ pressure} \times 100$$

Frequency-Domain Analysis. Another method of calculating the arterial compliance is by using frequency analysis. This is accomplished by dividing the amplitude density of the PPG at the cardiac frequency (a volume measurement) by the amplitude density of the arterial pressure waveform at the cardiac frequency (a pressure measurement), as shown in Fittgure 11.

$$Local\ Arterial\ Compliance = \frac{Amplitude\ density\ of\ PPG\ at\ cardiac\ frequency}{Amplitude\ density\ of\ arterial\ at\ cardiac\ frequency} \times 100$$

The compliance curves calculated from the time and frequency domains were almost identical. During this pheochromocytoma case, over a wide range of BP (SBP ranged from 75–340 mmHg), the compliance was inversely related to the BP waveforms.

CONCLUSIONS

The use of arterial BP, PPG, ECG, and peripheral venous pressure waveforms for patient monitoring offers a wealth of clinical information regarding patients during the

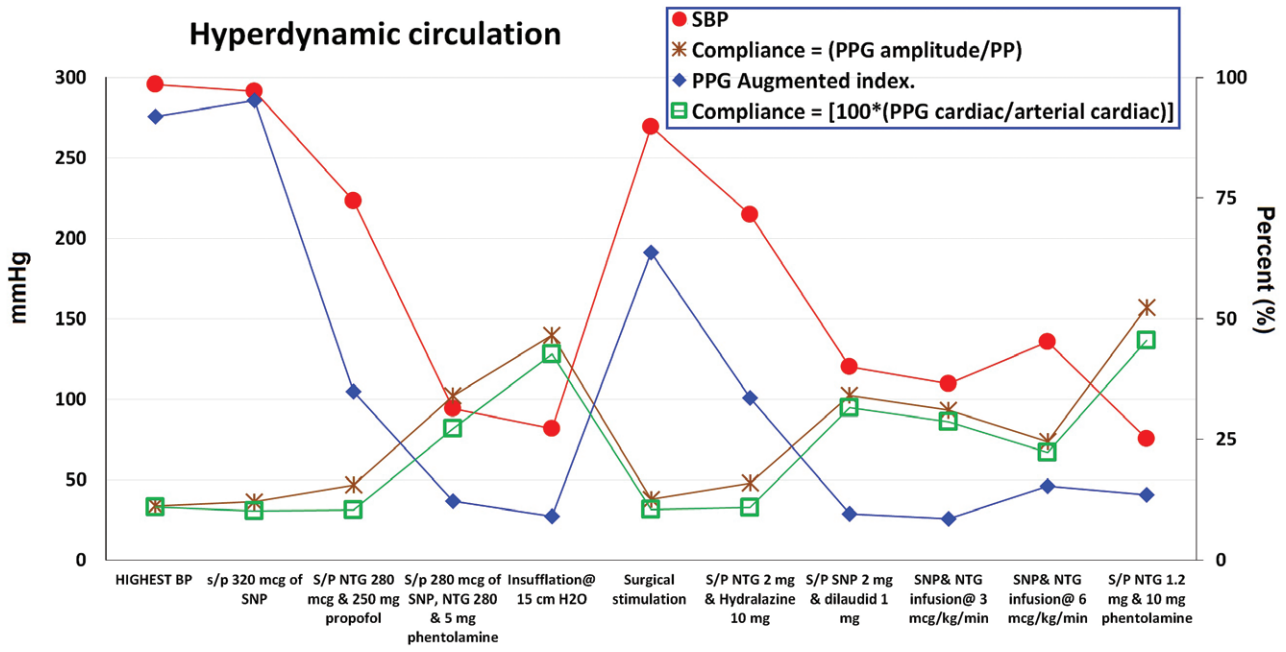


Figure 11. The relation among systolic blood pressure (SBP), PPG augmented index, and arterial compliance. The relationship among systolic BP, PPG augmented index, and arterial compliance during different events of the pheochromocytoma case. There is an inverse correlation between the arterial BP and the arterial compliance over a wide range of BP (75–340 mmHg). PP indicates pulse pressure; SBP, systolic BP.

perioperative period. These clinical waveforms are discarded as they disappear from the monitor screen. One goal of the clinician should be to develop an understanding of the underlying physiology responsible for these waveforms from both a practical and theoretical viewpoint. We believe 1 of the strengths of using frequency analysis is its ability to overcome artifacts by using larger time windows that are locked in at specific frequencies (eg, respiratory and cardiac).

The increase in baseline modulations of PPG and arterial BP waveforms will denote hypovolemia even before changes in hemodynamic vital signs, whereas the lack of baseline modulation of PPG with respiration denotes that the patient is not hypovolemic (provided adequate tidal volume is used). Not only can the PVP be used as a surrogate to CVP waveforms, but the loss of cardiac oscillation of PVP waveforms also denotes that the patient is hypovolemic. The position of PPG dicrotic notch, area, and slope of pressure–volume loop may be useful as monitors of patient's vascular tone.

The next step would be the utilization of this understanding to develop new methods of patient monitoring. It is important to keep in mind that monitors, by themselves, do not change patient outcomes. It is only changes in therapy that can do that. The results of monitoring should be presented in such a way as to allow for the guidance of therapy. This therapy might take on many forms including pharmaceutical agents, the administration of intravenous fluids (saline or blood), or the administration of oxygen.

DISCLOSURE

Name: Aymen A. Alian, MD.
Contribution: This author helped design the study, conduct the study, analyze the data, and write the manuscript.

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