Peripherals venous pressure waveform
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Purpose of review
The focus of intraoperative monitoring is moving away from invasive monitoring. This has been attributed to procedure time, cost, and the known risks, which include carotid artery puncture, arrhythmia, pneumothorax, and infection. Until recently, the venous system’s contribution to the circulatory system has been incorrectly identified as being insignificant. This article summarizes the unique characteristics of the peripheral venous system.

Recent findings
Numerous studies done in the last few years have paid attention to peripheral venous pressure and more specifically its pressure waveform. The popularity of, and further focus on, the field of photoplethysmography has described a strong venous component. Analysis of venous waveforms has indicated that, like arterial waveforms, they too exhibit respiratory variations and change in response to physiologic challenges.

Summary
The veins play a critical role in cardiovascular homeostasis; they do more than conduct blood to the heart. Considering the ease of measurement from a peripheral intravenous catheter, further study should be conducted to investigate the usefulness and limitations of such a minimally invasive and inexpensive monitoring device.

Keywords
circulatory system, non invasive monitoring, vascular compliance, venous pressure

Introduction
Folkow, in the 1960s, studied the characteristics of veins and noted the huge disparity which existed in the literature concerning the amount of information on the arterial compared with the venous sides of the circulation [1]. Almost 50 years later, we have still not filled the gap. Although arterial waveforms have been studied extensively, focus on the peripheral venous component has been scarce. 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. There have been attempts to use static IV pressure as an indication of central venous pressure (CVP) [2,3,4,5–7].

(1) The central venous conduit [which included the inferior vena cava (IVC) and its continuation to the large veins] holding approximately 18% of the total blood volume
(2) The reactive venous reservoir (between the capillaries and the central venous conduit) containing approximately 45% of the blood volume.

He was also able to demonstrate that there exists an intravenous gradient that facilitates the movement from the reactive reservoir to the central reservoir. He further showed that sympathetic stimulation had no significant impact on the central venous reservoir, whereas on the reactive venous reservoir it is the most dynamic.

Venous compartment
In the past, a number of investigators have advanced the concept that a small change in venous capacity, induced by venous constriction or relaxation, should markedly alter the cardiac output. In a delicately designed experiment by Bartelstone [8] involving dogs, he was able to divide the venous system into two major components:

Impact of respiration on venous reservoir
Breacher examined the relationship of respiration on the intrathoracic (Bartelstone’s central venous conduit) and extrathoracic veins (the reactive venous reservoir) [9]. His experiments were also on dogs, breathing both spontaneously and mechanically. Pressure recordings were obtained from the jugular vein, femoral artery,
intrapleural space and right atrium. He concluded that when breathing spontaneously:

1. Thoracic aspiration during inspiration causes a significant increase in blood flow to the right atrium; this is due to the emptying of the extrathoracic veins into the central veins.
2. Flow does not increase further once the collapsed state of extrathoracic veins has been reached.
3. If inspiration is long and deep enough, flow may even drop slightly below its inspiratory maximum due to the exhaustion of the extrathoracic reservoir and the progressively increasing resistance offered by the partially collapsed extrathoracic veins.

He then studied the same relationship under conditions of hypervolemia and hypovolemia and concluded that identical degrees of thoracic aspiration increase venous return only moderately in the hypovolemic state as compared with the euvoolemic state. He further noted that the greater the hypovolemia the shorter the duration and amount of the aspiratory flow augmentation and the earlier the onset of the collapsed stage [9].

Respiratory variations in the central venous waveform have been described before. The respiratory induced variation in CVP also causes variations in arterial blood pressure (ABP) and peripheral venous pressure (PVP). Valves in the venous system in the forearm may hinder hydrostatic continuity, implying that one single vein

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Figure 1 The normal morphology of a peripheral venous waveform during anesthesia with positive pressure ventilation; the capnogram and photoplethysmogram are also depicted.
might not represent the entire venous system in the forearm. Whether the respiratory variation in PVP is a forward or a backward transmission from the central venous system is unclear [10]. The degree of ventilation-induced variation (either positive pressure or spontaneous) in the PVP is directly related to the volume status of the patient. In other words, the higher the degree of pressure variation during respiration in the venous waveform, the more ‘full’ the patient’s circulation is. Conversely, as the variation disappears from the waveform the more ‘empty’ the patient is. This phenomenon can be used to help guide fluid replacement (colloid, crystalloid, and blood products) for a patient. Of note, this is the opposite of the

Figure 2 The onset of abdominal insufflation in a patient under general anesthesia and positive pressure ventilation, showing the increase in the amplitude of the venous waveform; the arterial waveform, photoplethysmogram and capnogram are also shown.
effect ventilation has on the arterial pressure (which increases as the patient loses blood volume). This creates the possibility of producing an index combining the two (e.g. arterial variation/venous variation). This would lead to greater sensitivity and specificity than either parameter alone (Figs 1–3).

There is a direct link between airway pressure and venous pressure. As airway pressure increases so does PVP (Fig. 4). During positive pressure ventilation, this effect could be used to monitor the effect of ventilation on the vascular system and assist with ventilator adjustments. During spontaneous ventilation this effect could be used to monitor respiratory health including asthma and heart failure (extent of disease and effectiveness of therapy).

**Venous compliance**
Rothe in the 1990s effectively tackled the issue of compliance in the venous compartment [11]. He further...
The relationship between CVP and venous return is known as the venous return curve. When venous tone changes so does the CVP (Fig. 7). For example, whenever PVP is elevated by increases in blood volume or by sympathetic stimulation, the venous function curve shifts upward and to the right [12**]. This is felt to be caused by a decrease in venous capacitance, which raises the mean circulatory pressure, which in turn tends to increase all intravascular pressures, and thus increases the preload of the heart [12**].

In 1955, Guyton, a man known for his valuable contributions to the field of physiology, explained the relationship between venous compliance and cardiac output. He used Starling’s law for the determination of cardiac output, which he defined as the relationship between the cardiac output and right atrial pressure and called the ‘cardiac response curve’ [14].

Figure 8 shows that peripheral venous constriction increases cardiac output by raising CVP and moving the heart’s function upward along a fixed cardiac function curve. Figure 8 also depicts the response of the vasculature to hemorrhage in progressive steps (i.e. A to B to C to D), which does not happen discretely in reality. The actual course of a patient’s net response to hemorrhage would appear to follow nearly a straight line from point A to point D.

**Response to bleeding and sympathetic activity**

The behavior of peripheral veins of the forearm, in response to hemorrhage or sympathetic activity, is conflicting. Although Zoller was able to demonstrate that the forearm veins show intense venoconstriction in the absence of changes in other hemodynamic parameters, other studies have proved that those limb veins have very little role to play in contributing to the central blood volume [15].

**Interchangeability between central venous pressure and peripheral venous pressure**

Although controversy still exists concerning the role of peripheral veins and their contribution to the central volume in face of blood loss, many studies in the late 1990s and early 2000s have shown a consistent correlation between CVP and PVP [16,17]. Although CVP waveforms characteristically show a-waves, c-waves, and v-waves, PVP waveforms appear as a more dampened sinusoidal pattern.

Munis et al. [18**] reported mean PVP values of 13 mmHg and CVP values of 10 mmHg, with a PVP–CVP difference of 3 mmHg. Amar et al. [19] observed mean PVP
values of 9 mmHg and a mean CVP value of 8 mmHg in 100 intraoperative patients. Hadimioglu et al. [20] came to the same conclusions in patients undergoing kidney transplant. Baty et al. [21] studied 29 infants and children after cardiopulmonary bypass. The difference between PVP and CVP in these patients was 11 ± 3 mmHg. No clinically significant variation in the accuracy of the technique was noted based on the actual CVP value, size of the peripheral IV catheter, its location, or the patient's weight.

Other authors have done similar assessments in patients undergoing right hepatectomy. A central venous catheter was placed through the right internal jugular vein and a peripheral venous catheter was inserted at the antecubital fossa in the right arm. A total of 1430 simultaneous measurements of CVP and PVP were recorded. They concluded the difference between PVP and CVP was within clinically acceptable agreement and the degree of difference tended to remain relatively constant throughout the right hepatectomy in living donors [3]. Hofman
Figure 7 The relation between venous filling pressure and venous return

Modified with permission from [12**].

[4] studied the correlation of both variables in patients undergoing liver transplant. The nature of the liver transplant surgery allowed the authors to test the durability of the PVP/CVP correlation during extreme derangements of physiology, including IVC crossclamp, brisk hemorrhage, and reperfusion of the donor graft. One unexpected finding, not previously reported in other studies, was the much weaker PVP/CVP correlation at low filling pressures. It was suggested that, at low filling pressures, peripheral veins intermittently collapse, interrupting their continuity with the central circulation and thus leading to PVP/CVP divergence.

Figure 8 The relationship between the venous return curve and Starling cardiac output curve

The intersection points correspond to the cardiac output. Modified with permission from [12**].

Photoplethysmography and peripheral venous component

The pulse oximeter was introduced not too long ago. Its rapid acceptance stems from its ease of use and portability. It was not long before clinical investigators starting looking into the complexity of the waveform called the photoplethysmogram. Shelley et al. [22] studied the photoplethysmogram and they observed that the PVP imparted special properties to the waveform. A photoplethysmograph has a baseline component, which is related to changes in the venous bed, and an oscillating component, which is due to the pulsatile flow [23]. In Nilsson’s study [10] of spontaneously breathing patients he noted that the respiratory modulation of amplitude of the photoplethysmogram was closely related to the respiratory variations present in the peripheral venous waveforms.

Conclusion

It is hoped that this article has opened doors for readers to appreciate the dynamic nature of the peripheral veins and the opportunities available to explore them further. It is in the best interest of the patient when the clinician is open-minded and explores all possible avenues to optimize care. The use of the intravenous catheter as a monitoring tool is intriguing and worthy of further exploration.

References and recommended reading

Papers of particular interest, published within the annual period of review, have been highlighted as:

• of special interest

•• of outstanding interest

Additional references related to this topic can also be found in the Current World Literature section in this issue (p. 828).


This is a nice demonstration of the use of this technology during difficult clinical conditions.


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12 Mohrman D. Cardiovascular physiology. 6th ed. New York: McGraw-Hill Medical; 2006. This textbook has a nice approach to cardiac physiology that is concise and quite readable.
18 Munis JR, Bhatia S, Lazada LJ. Peripheral venous pressure as a hemodynamic variable in neurosurgical patients. Anesth Analg 2001; 92:172–179. This article is an excellent example of the use of the PVP as a clinical monitor. The same author (Munis) has an interesting patent (6 623 470) that uses PVP to guide fluid therapy.