

Ultrasound Guided Noninvasive Measurement of Central Venous Pressure

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INTRODUCTION

Central venous pressure (CVP) is a measure of the mean pressure within the thoracic *vena cava*, which is the largest vein in the body and responsible for returning blood from the systemic circulation to the heart. CVP is a major determinant of the filling pressure and cardiac preload, and like any fluid pump, the heart depends on an adequate preload to function effectively. Low venous return translates into a lower preload and a drop in overall cardiac output, a relationship described by the Frank-Starling Mechanism.

CVP is an important physiological parameter, the correct measure of which is a clinically relevant diagnostic tool for heart failure patients. In addition to other vitals such as heart rate and mean arterial pressure, accurate measures of central venous pressure through simple diagnostic instrumentation would provide physicians with a clear picture of cardiac functionality, and allow for more targeted treatment. Recent literature has also shown that measuring CVP can be an important hemodynamic indicator for the early identification and treatment of more widespread conditions, such as sepsis (Rivers, Nguyen, Havstad, & Ressler, 2001). With over five million patients (American Heart Association, <http://www.americanheart.org/presenter.jhtml>) in the U.S. presenting with heart failure-like symptoms annually, a current challenge for physicians is to obtain a quick and accurate measure of a patient's central venous pressure in a manner that poses minimum discomfort.

A novel noninvasive method to estimate CVP is to use ultrasound imaging in conjunction with a surface pressure measurement. Ultrasound is first used to visualize the *internal jugular* (IJ) vein below the skin on a patient's neck, and a custom pressure transducer is then used to detect the surface pressure required to

collapse the IJ. The collapsing pressure is correlated to the central venous pressure and reported back to the operator. This proposed measurement procedure is suitable for emergency situations or primary care settings where rapid diagnosis of a patient's CVP is required, and mitigates the need for further invasive and costly procedures.

BACKGROUND

Central Venous Pressure is dependent on the ratio of venous blood volume (V_v) to venous compliance (C_v), as described in (1):

$$CVP = \frac{V_v}{C_v} \quad (1)$$

From this relationship, it is clear that an increase in blood volume within the thoracic *vena cava* will lead to greater pressure exerted on the vessel walls, and that a vessel with low compliance will experience greater pressure from an increase in blood volume than a vessel with high compliance. A variety of physiological conditions may influence CVP, due to changes in blood volume and/or compliance, and a few are presented here:

- **Hypervolemia:** Condition where overall blood volume is too high. Hypervolemia can arise from high salt intake or a failure in the kidney's ability to excrete salt and water from the body. Excess fluids seep into body tissue leading to edema (swelling), which can cause headaches and respiratory difficulties. The increase in blood volume increases central venous pressure.

- **Hypovolemia:** Condition where overall blood volume is too low. A common cause of hypovolemia is blood loss due to trauma, which leads to weakness and, in extreme cases, organ failure. The drop in venous blood volume decreases central venous pressure.
- **Heart Failure:** Condition where the heart is no longer able to maintain adequate blood circulation. Some causes of heart failure including coronary artery disease, valvular disease, and myocardial infarction. Low cardiac output leads to a backup of blood in the venous compartment, and increases central venous pressure.
- **Sympathetic Activation:** General increase in the activity of the sympathetic nervous system. Sympathetic activation, characterized as the “fight or flight” response, leads to increased heart rate, inhibition of the digestive system, and increased vascular tone. The latter decreases venous compliance, and increases central venous pressure.

Due to the high compliance of the thoracic *vena cava* and its inconvenient physiological location, direct measurement of CVP using a sphygmomanometer has long been considered impractical. Physicians have instead relied on direct measurements of intraluminal venous pressure, or crude noninvasive techniques for estimating CVP. This section explores both the traditional and more novel diagnostic approaches for measuring CVP, as a prelude to our discussion of the proposed measurement procedure and device.

Traditional Methods of Measuring Central Venous Pressure

- a. Lewis (1930) describes one of the oldest methods to estimate CVP using jugular vein distension. When hypervolemic or cardiac failure symptoms manifest as increased venous blood volume, the extra blood pools and increases the size of the patient’s jugular veins. The jugular vein’s proximity to the surface of the neck makes it possible for a clinician to visually identify the swelling of neck veins, but this technique is not a true measurement of CVP since it is quite subjective. For one, the jugular veins are not always visible, especially when obscured by layers of fat, and venous distension is only readily apparent in cases of extremely high CVP. Therefore, this

technique is useful for rapid diagnosis of a major increase in CVP, but not suitable for an objective measurement, rendering it largely inadequate for continuous monitoring.

- b. Another noninvasive measurement technique that attempts to quantify CVP readings involves treating the jugular veins as manometer tubes to the right atrium, implying that higher pressure leads to a greater column of blood in the jugular veins (Lipton, 2000). The selected zero-reference point is the angle of Louis, located at the junction of the *manubrium* and the *sternum* 5 cm above the right atrium, because venous pressure at this point changes very little with posture. The height of the blood column can be determined by visually identifying jugular vein pulsations, which are oscillations in pressure caused by the cardiac cycle, and visible where the vein transitions from the distended (filled with blood) to the collapsed (no blood) state. The hydrostatic pressure that corresponds to the height difference between the point of fluctuations and the angle of Louis is used as a measure of CVP. In practice, this quantitative approach is often impractical, because the venous pulsations are only visible when the top of the blood column is in an area of the neck where the jugular passes close to the skin surface. If not, the clinician must raise the patient slowly upward from a supine position until the pulsations become visible, a process that is both physically taxing and time-consuming. Furthermore, if CVP is too high, then the top of the blood column may lie above the mandible, even with the patient sitting upright, making the fluctuations invisible. As with the previous method, layers of fat may also hinder visualization of the venous pulsations on the neck.
- c. Numerous studies have shown that noninvasive methods fare poorly in quantifying CVP, because subjective measurements are difficult to standardize. Factors such as posture are hard to control in heart failure patients that must be lifted upright to observe jugular pulsations, which leads to highly variable and inaccurate measurements when compared to invasive recordings (McGee, 1998). Therefore, situations necessitating accurate CVP readings for prompt and correct treatment rely on invasive catheter-based measurements as the standard-of-care. Central venous access is the

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