

Chapter 3.19

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 re-

turn 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

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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

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