The Meaning of Central Venous Pressure (CVP) relative to Fluid Management and Blood Flow

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In modern clinical medicine, our understanding of Central Venous Pressure (CVP) has evolved from Guyton, Magder, Rothe, Levy, Jayant, Gelman, Brengelmann, Reddi and Carpenter, and Marik to the effect that many physicians question the clinical utility of CVP in patient care? This confusion and controversy stems from a lack of understanding of the source and regulation of CVP in human physiology. Where it comes from, why it exists, and what it does in the overall control of blood flow? The CVP controversy is related to a similar controversy related to the definition of PreLoad, Norton? Recent advances in noninvasive hemodynamic monitoring (VasoMap™) by NIVasc, Inc. have brought new insights and answers to these questions about CVP and Preload through the VasoMap™'s ability to map the residual volume vs pressure structure of the entire Peripheral Vascular Loop(PVL) in a slice of the upper limb. The VasoMap™ and its PVL structure allows us to now examine the relationships between Pressure, Residual Volumes, Resistance, Compliance, and Flow (Figure 1) along the pathway of blood flow.

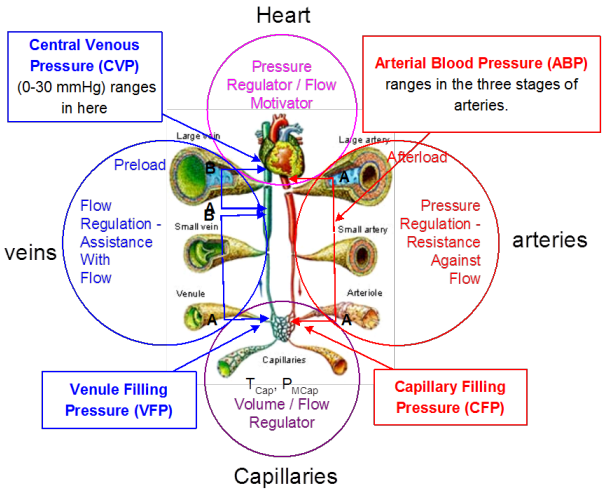

Figure 2

As seen in Figure 1 the PVL is a closed loop system comprised of serial vessels serving three different purposes and driven by a volume pump. It is important to understand the differences in purpose between the Arteries which "resist flow" and the Veins which "assist flow." Ohm's Law applied to the PVL demonstrates that CVP is nothing more than the pressure that remains (not dissipated by vascular resistance in the PVL segments) after the blood has taken its trip around the PVL along the pathway of flow. The Arteries make pressure while the Veins make flow via venous return. The VasoMap™ measures the Compliance or Elastance of these vessels and therefore can determine the Stressed Volume vs Unstressed Volume in each segment along the pathway of flow. It is the Stressed Venous Volume that determines Preload onto the Heart and Stressed Volume is a function of Volume and Vessel Elastance. Much confusion arises in fluid management in the absence of this information about the vascular status of the patient. It is clear from this model, that pressure is not the prime driver of the Frank-Starling mechanism but excess venous volume in combination with venous elastance. The definition of Mean Circulatory Filling Pressure (Pms) proves this since there is no flow with a Pms of 7 mmHg. Preload, in essence, is the 'Excess Volume' presented at the Tricuspid valve of the heart by the Venous Stressed Volume. Preload is not motivated unilaterally by the Blood Volume, but by the combination of Venous Elastance and Venous Blood Volume which is regulating

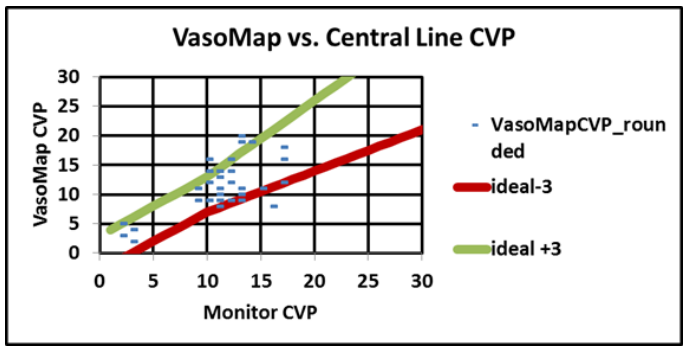

Figure 1

and optimizing Venous Return and Cardiac Output. Therefore, fluid management without the VasoMap™ and knowledge of the Venous Elastance and Stressed Venous Volume values in the patient's PVL, limits the clinician to only a fraction of the variables involved in managing blood flow. CVP remains an important clinical parameter as it indicates the overall vascular status of the patient but not necessarily in the way it is interpreted by many clinicians today. The VasoMap PVL model simplifies and clarifies these relationships for the clinician in order to improve the management of circulation in the patient.