VENOUS EXCESS: A DIFFERENT APPROACH TO MONITORING THE CIRCULATION OF BLOOD

Thomas Edward Woodcock MB.B.S, M.Phil, FFARCS Eng.
fluidphysiology.org , Hampshire, United Kingdom.

Central venous pressure (CVP) is the autoregulated pressure on the venous side of the circulation, and mean arterial pressure (MAP) on the arterial side. Measurement of CVP is invaluable in the differential diagnosis of acute low cardiac output shock; CVP below the autoregulatory range is an indication for fluid resuscitation, while CVP above the autoregulatory range raises the possibilities of tension pneumothorax or pulmonary embolism, with very different treatment pathways.

CVP monitoring within the autoregulatory range is useful in the management of chronic heart failure. There is a growing body of evidence that venous 'congestion' leads to deteriorating renal function, and a simple, repeatable, non-invasive technique for monitoring CVP would bring substantial outcome benefits.

As a guide to intravascular volume, or as a predictor of fluid responsiveness in the critical care setting, CVP is reported to be poor. Under fluid loading, CVP will only start to rise substantially once the upper regulatory range has been passed and fluid overload is established. Stroke volume response to fluid loading should be assessed by monitoring the stroke volume, not the CVP.

CVP is the outlet pressure of the interstitial fluid circulation. In the post-resuscitation treatment of fluid overload with edema, the target CVP should be the lowest compatible with adequate stroke volume. Researchers have pragmatically suggested 4 mmHg.

Expert physiologists of the laboratory bench and expert physicians at the bedside provide differing accounts about the source of cardiac output more than a century after Otto Frank and Ernest Starling developed a much-quoted "law"; that the stroke volume of the heart increases in response to an increase in the volume of blood filling the heart (the end-diastolic volume) when all other factors remain constant. "Guyton" diagrams illustrate a commonly taught but confusing narrative. A curve describing the relationship between venous gradient and venous return is extrapolated to the point at which venous return is zero, and is called the mean circulatory pressure (MCP). The pressure gradient for venous return when it is greater than zero is therefore MCP-CVP, and one could calculate a venous resistance. Notice that, if true, lowering CVP would increase venous return. J Rodney Levick boldly states in Introduction to Cardiovascular Physiology 5e that "CVP, not 'venous return', is the true regulator of stroke volume." Clinicians expect to be able to increase cardiac output by raising CVP by infusion of intravenous fluid. Hence we find many major critical care resuscitation trial protocols requiring fluid to achieve a nominal higher CVP. A typical example is 8 mmHg in spontaneous respiration, 12 in positive pressure ventilated patients.

Physiologist CF Rothe taught that "A decrease in CVP by 1 cmH_2O can reduce cardiac output by half." Perhaps it would have been better to interprete this as "CVP is autoregulated over a wide range of cardiac outputs". That fits with physician M. Pinsky's assertion that "data support the hypothesis that the normal human right ventricle fills at or below its unstressed volume, such that right ventricular end-diastolic volume changes occur without any change in diastolic wall stretch." In the real patients who occupy our critical care beds there is observed to be no useful relationship between transmural right atrial pressure and right ventricular end-diastolic volume / stroke volume. It seems that the role of the venous system (including the right atrium and ventricle) is to sustain an effective cardiac output by assisting flow (elastic recoil of venules and small veins) and minimising impedance (stiffness of the right ventricle) without limiting left ventricular filling. The right atrial pressure (let's call it CVP) is kept constant and as low as possible in face of varying venous return. It is vital to be aware that maximal cardiac output responses to increased demand will be limited if CVP is raised above normal, because the left ventricle's ability to fill becomes increasingly limited, in the extreme causing cardiogenic shock.

Reddi and Carpenter (2005) remind us that we need an error signal to create a negative feedback loop for autoregulation. For stabilizing cardiac output/ venous return, venous excess (VE) is the accumulated volume of blood at the input side of the heart that facilitates rapid ventricular filling and supports stroke volume (SV). This is an example of a classic direct negative feedback system, the error signal being accumulating VE from a discrepancy between what is being supplied to the heart and what it is actually pumping out. Reflect now on Figure 1.

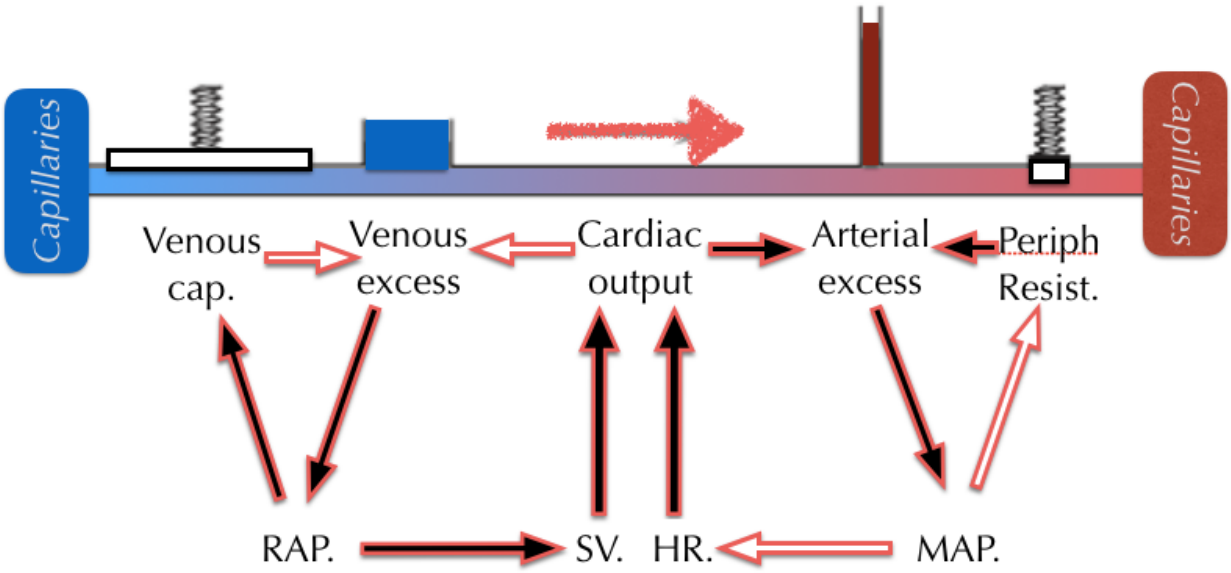

Figure 1: An illustration of the interplays between cardiovascular parameters according to Reddi & Carpenter.

The cardiac output exists to supply the metabolic needs of the systemic tissues. The tap that regulates flow of oxygenated and nutrient-rich arterial blood is peripheral resistance (PR), and it requires mean arterial pressure to be autoregulated so it can be physiologically responsive to metabolic demands. The tendency for MAP to fall with falling PR is countered by increase in heart rate and increase in VE which will protect or even increase SV. Right ventricular stroke volume increases as impedance to ejection falls (pulmonary circulation interaction) and venous excess increases to create the additional venous flow. Reddi & Carpenter do not include an appreciation of right – left ventricular interactions which are important in critical care practice. I see no reason why they could not be described in terms including pulmonary arterial excess and resistance, and pulmonary venous excess and capacitance. Systemic and pulmonary inflammation are characterised by hyper-filtration & increased lymph flow, with pulmonary hypertension and systemic hypotension. The effect of positive pressure ventilation on pulmonary arterial resistance is dependent on the distribution of blood flow between the West Zones 1, 2 and 3.

What are the anatomic correlates of the venous excess? After being extruded from the constricting environment of a capillary, blood cells find themselves moving with plasma along venules. Though each venule is of very small diameter, there are very many of them in the pressure range 20-30 mmHg and they provide elastic recoil at the top of their sigmoid pressure – volume curve. They drain to a fewer number of small veins, each of greater diameter than a venule but in their typical pressure range of 10-20 mmHg they exert less elastic recoil to assist flow. The small veins can, however, regulate venous capacitance by constriction/ relaxation. Large vein pressure is typically less than 10 mmHg, in which pressure range substantial changes in volume are possible with very little change in pressure. Anyone who has used 2D ultrasound to inspect a large vein knows that in health it may be almost collapsed while cardiac output is quite normal. Reddi and Carpenter claim that the adequacy of venous excess volume can be assessed by visualization of the jugular venous pulse. Guarracino *et al* have found that ultrasound visualization of jugular vein distensibility predicts fluid responsiveness in septic patients.

Consider now the relaxing right ventricle. As the ventricular capacity increases, a stroke volume's worth of blood is drawn from the central venous (intrathoracic) volume of the atrium and vena cava. CVP does not fall during diastole so long as there is sufficient venous excess to replace the central volume which has become right ventricular stroke volume and been ejected into the pulmonary artery behind a closed tricuspid valve. The large veins are in turn replenished by blood from the small veins, and the small veins are replenished from the venules. The venules are replenished by the open capillaries. Capillaries can be open ("flow" condition, containing moving red blood cells) or closed ("stop" condition) according to local tissue metabolic needs, but at any time the open capillaries are distended by the intraluminal capillary hydrostatic pressure. Such pressure-volume mapping of the venous circulation is possible with co-extensive bioimpedance plethysmography, but the technology is not yet commercially available.

You may notice some similarity here with the Guytonian concept of mean circulatory pressure (MCP), or mean systemic pressure (MSP) or some such, being the notional pressure within the not-yet-ischaemic circulation during asystole and no flow. MCP has been estimated to be around 18 mmHg in post-cardiac surgical patients. I do accept that MCP should go up and down with the venule - CVP gradient, but see no reason to invent it if our narrative works using real and measurable dynamic parameters.

The rapidity of elastic recoil inherent in healthy venules and veins is attributable to collagen and elastin fibres within the basement membrane, a perivascular condensation of the interstitial biomatrix. This rapid recoil keeps the intravascular pressure needed to assist flow from one venous compartment to the next to a minimum. With age, in diabetes, and in systemic inflammation including sepsis this elasticity is diminished. One contributory factor seems to be the deposition of advanced glycation end-products (AGEs). The process may be one of the causes of heart failure with preserved ejection fraction, which was previously called diastolic heart failure, and is an early feature of cardiovascular dysfunction in sepsis.

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Keywords

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