Understanding Clinical Hemodynamics

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The physiologic paradigm that clinicians reference in their attempts to explain and understand the biology of both healthy and critically ill patients has been in evolution for more than 100 years. Interestingly, our understanding of the clinical circulation has always been thought of as ‘complete’, with creative clinicians invoking a variety of reasons to explain away apparent discrepancies between commonly used mental models and the realities of clinical medicine.

The most primitive formulation of the circulation entails simple conservation of matter:

\[ \text{Cardiac Output} = \text{Stroke Volume} \times \text{Heart Rate} = Q_t = SV \times HR \]

This statement, while obviously always true, offers sapient practitioners little insight into why the circulation in a particular patient might be unacceptable, and how they might rationally intervene. During the mid-20th century, a relatively complete paradigm for understanding the role of the venous return in controlling the cardiac output was refined by Guyton and his co-workers, and has been repetitively validated since it was first described (refs Jacobsohn, Magder, Guyton, Sylvester). Although not complete, this theory was powerful in the hands of those who understood it.

The balloon-tipped, flow directed, thermistor equipped pulmonary artery catheter heralded the subsequent era of the understanding of the clinical circulation. This device, coupled with a deep understanding of the mechanics of left ventricular function heralded the era in which the circulation and all of its pathology were understood from the perspective of the left-ventricle – which some now refer to as the LV centered view of the circulation (Sagawa). For those who trained in that paradigm, preload, afterload, and contractility were the determinants of cardiac output:

\[ \text{Cardiac Output} = CO = \frac{\text{MAP} - \text{RAP}}{\text{SVR}} \]

(Where MAP = Mean Arterial Pressure, RAP = Right Atrial Pressure and SVR = Systemic Vascular Resistance)

Some patients have a right heart limited circulation, which can be formulated using a very similar equation:

\[ CO = \frac{\text{PA} - \text{LAP}}{\text{PVR}} \]

(Where \( \text{PA} \) = Mean Pulmonary Artery Pressure, \( \text{LAP} \) = Left Atrial Pressure, and \( \text{PVR} \) = Pulmonary Vascular Resistance).

Nevertheless, the LV centered view of the circulation focused on preload, afterload, and contractility, and was frustrated by a variety of obstacles. The most important was the poor correlation between measured filling pressures and left ventricular end-diastolic volumes as assessed by echocardiography (refs Kumar, Hofer, Kramer). Echocardiography has documented that LV compliance is far more dynamic than anyone believed prior to its widespread clinical use (Coriat). The other, more insidious problem with the LV centered world-view is that adherents tend to regard RAP almost exclusively as an index of circulatory volume, forgetting that it is the downstream hydrostatic resistance to venous return in the model of Guyton:

\[ \text{VR} = \frac{\text{CO} \times \text{RVR}}{\text{Pms} - \text{RAP}} \]

(Where \( \text{VR} \) = Venous Return, \( \text{Pms} \) = Right Atrial Pressure, and \( \text{RVR} \) = Resistance to Venous Return)
The circulation in any patient at any moment in time is the product of the interaction of the venous circuit with the heart (the pump). The RAP is a product of that interaction.

All of this has produced the present understanding of clinical hemodynamics, which is predicated on a synthesis of venous return and cardiac physiology (Sylvester, Jacobsohn). This model can be used to generate a series of questions that can guide the assessment of a patient in shock.

**What is Shock?** Shock is globally inadequate perfusion of tissues sufficient to produce both tissue hypoxia and organ dysfunction. While shock is classically associated with hypotension, there is increasing acceptance of the contention that hypotension is a relatively ‘late’ indicator of shock, and that clinicians should be more attuned to organ system dysfunction as evidence of shock.

**Signs of Shock:**
- altered mentation
- oliguria
- decreased mixed venous or central venous saturation
- hypotension, abnormal heart rate
- lactic acidosis
- peripheral cyanosis (variable)

In both the critical care and trauma literature, the endpoints for resuscitation have also evolved. While traditional endpoints such as mean arterial pressure and central venous pressure are still regarded as important, increasing emphasis is being placed on the mixed/central venous oxygen saturation (Ladakis) and lactate levels in the blood. The combination of inexpensive and readily available serum lactates and increasing appreciation of the prevalence of hyperchloremic acidosis in the setting of large volume resuscitation has led to the near abandonment of the base excess/deficit as a guide to the adequacy of resuscitation. Several publications over the past several years have dampened enthusiasm for the use of central venous oxygenation (Chawla, Sander, Varpula), but it nevertheless remains a very useful indicator of the adequacy of oxygen delivery.

It is helpful to understand the modern incarnation of the Fick Equation of the relationship between oxygen consumption, cardiac output, arterial oxygen content, and mixed venous oxygen content. This algebraic rearrangement emphasizes that the mixed venous saturation is adequate only when the delivery of oxygen to the peripheral tissues is well matched to their needs:

\[
SvO2 (CvO2) = \frac{CaO2 – VO2}{Qt}
\]

(Where \(SvO2\) is the mixed venous oxygen saturation, \(CvO2\) is the mixed venous oxygen content, \(CaO2\) is the arterial oxygen content, \(VO2\) is the oxygen consumption, and \(Qt\) is the cardiac output)

Importantly, as oxygen delivery to the tissues falls, oxygen extraction rises, and continues until the tissues are no longer able to extract more oxygen. When this happens, crisis ensues.
In the above figure, oxygen extraction increases as oxygen delivery decreases. When the tissues reach the limits of their ability to extract oxygen (the critical extraction ratio $ER_c$), the critical oxygen delivery has been reached ($Q_{O2c}$), and further decreases in oxygen delivery will be associated with a decline in oxygen consumption.

Arterial hypoxemia, anemia, hyper-metabolism, and a low cardiac output all lower the mixed venous and central venous saturation. Increasingly, practitioners are utilizing protocols which include as one of their endpoints a central venous oxygen saturation above a certain level (Ladakis, Rivers). This strategy of ‘forward defense’ is in part based on the increasing recognition that hypotension is a relatively late indicator of shock, and that resuscitating a patient to a marginal blood pressure may leave them with an inadequate physiologic reserve.

From Physics: $V = I \times R$ Substituting produces: $BP - Pra = Qt \times SVR$

Hypoperfusion (shock) can arise from:
- low cardiac output
- low SVR
- the combination of a low cardiac output and high SVR

We can represent the circulation by superimposing the Starling curve and the venous return curves. Examples of this are below:
As demonstrated by the above figure, we can superimpose the Starling curve from above left upon the venous return curve from the above right and generate a graphical representation of the state of the circulation. The cardiac output is represented by the Y projection of the intersection of these curves, and the CVP we measure clinically is represented by the X projection of the intersection of these curves.

Diastolic dysfunction is a generally underappreciated and very important contributor or cause of shock states.

In animal models of hemorrhagic shock, even small reductions in pleural pressures from reduced levels of PEEP or reduced respiratory rates can produce dramatic improvements in survival (Herff). This data, coupled with similar data from animal models of CPR, are generating increased interest in ventilation strategies associated with the lowest possible airway pressures in patients with shock.

<table>
<thead>
<tr>
<th>Causes of Diastolic Dysfunction:</th>
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<tbody>
<tr>
<td><strong>Extrinsic</strong></td>
</tr>
<tr>
<td>PEEP, iPEEP</td>
</tr>
<tr>
<td>tPTX</td>
</tr>
<tr>
<td>Pericardial fluid</td>
</tr>
<tr>
<td>Massive ascites</td>
</tr>
</tbody>
</table>

In the above table, those causes listed in italics may be responsive to the infusion of volume. The other causes are minimally responsive to volume infusion, or not at all.

**Bedside Assessment of the patient with shock**
The following questions constitute an orderly way to assess the patient with inadequate circulation:

1. Is the Cardiac Output Reduced?
2. Is the heart “too full”?
3. What doesn’t fit?

**Is the cardiac output reduced?**
- No → Vasodilated Shock
- Yes → Hypovolemic shock, Cardiogenic Shock, or Obstruction to Venous Return
The above figure demonstrates the sentinel feature of vasodilated or high cardiac output shock: the wide pulse pressure. Patients with vasodilated shock almost invariably have a pulse pressure which is greater than half of their systolic pressure, whereas patients with low cardiac output shock typically have a pulse pressure which is substantially lower than normal. A patient with a blood pressure of 80/30 almost certainly has vasodilated shock, whereas a patient with a blood pressure of 80/60 will have one of the causes of low cardiac output. On examination, patients with vasodilated shock will have brisk capillary refill while patients with low cardiac output shock will have delayed capillary refill.

**Differential Diagnosis of Vasodilated Shock:**
- Sepsis, Sepsis, Sepsis
- Systemic Inflammatory Response Syndrome (SIRS) (e.g. pancreatitis)
- Hepatic failure
- Anaphylaxis
- Adrenal insufficiency
- AV fistula
- Others

**Is the heart too full?**
If the cardiac output is low, the differentiation of hypovolemic and cardiogenic shock is accomplished through the review of pertinent historical, physical examination, and laboratory data. Historical information is often compelling in its support for the conclusion that hypovolemia is the cause of an unacceptable circulation.

<table>
<thead>
<tr>
<th>Causes of Hypovolemia:</th>
<th>Supportive of Cardiogenic Shock:</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Hemorrhage</td>
<td>- jugular venous distention</td>
</tr>
<tr>
<td>- insensible losses</td>
<td>- extra heart sounds</td>
</tr>
<tr>
<td>- redistribution to extravascular space</td>
<td>- pulmonary edema in association with narrow PP</td>
</tr>
<tr>
<td>- GI losses</td>
<td>- signs or symptoms of myocardial ischemia</td>
</tr>
<tr>
<td>- renal losses</td>
<td>- new heart murmurs</td>
</tr>
<tr>
<td>- vasodilation (venodilation)</td>
<td>- cardiomyopathy or myocarditis</td>
</tr>
</tbody>
</table>

Cardiogenic shock is most readily assessed with echocardiography. The differential diagnosis of cardiogenic shock includes acute LV infarction, acute on chronic LV failure, RV infarction, RV failure from some
cause of increased pulmonary vascular resistance, and previously undiagnosed valvular lesions such as aortic stenosis, mitral stenosis, and mitral regurgitation.

Echocardiography has supplanted the Swan-Ganz catheter as the method of choice for assessing the patient with suspected cardiogenic shock. Reasons for this include increasing recognition that practitioner understanding of how to utilize data from a Swan-Ganz catheter is generally poor (Iberti), difficulty demonstrating that these catheters improve outcomes (Sandham), and increasing acceptance that central venous gases correlate well with mixed venous gases. Perhaps most importantly, echocardiographic studies have documented surprisingly poor correlation between filling pressures as measured by invasive monitors and left ventricular end-diastolic volume (Osman). Evidence impeaching the use of central venous pressure measurements continues to accumulate, and is now being summarized in colorful review articles (Marik).

As a consequence of these insights, experts are increasingly advocating the use of arterial pulse pressure variation as a guide to administering fluid, with a difference of >10-15% with respiration strongly associated with a favorable response to fluid administration (Michard, 2005). The two most commonly used metrics are Systolic Pressure Variation (SPV) and Delta Pulse Pressure (ΔPP). Systolic Pressure Variation is easier to estimate from conventional monitors, but is slightly inferior to delta Pulse Pressure (also referred to as Pulse Pressure Variation – PPV). SPV and/or PPV outperform both CVP and Pcwp as predictors of volume responsiveness in septic patients and cardiac patients, including patients undergoing OPCAB and post-op CABGs (Auler, Hofer, Kramer). Newer monitors intended for use in either the ICU or the OR incorporate software that facilitates the evaluation of these parameters. Other technologies, including Stroke Volume Variation (SVV)(Lahnert, Machare-Delgado), and the PICCO derived Intrathoracic Blood Volume Index (ITBV) are being explored as alternatives to the CVP in predicting volume responsiveness (Mulier), but do not yet match the performance of either PPV or SPV. There is a growing literature regarding the use of pulse-oximeter derived plethysmography as a less-invasive alternative to SPV or PPV (e.g. Pizov).

Systolic pressure variation is useful as a guide to the management of the patient in shock in another way: patients with minimal or no variation in the blood pressure and pulse pressure are very unlikely to respond to volume administration. The initial efforts to resuscitate such patients should therefore be directed at pharmacologic or mechanical interventions, which are much more likely to be effective. Because this strategy minimizes the unnecessary administration of fluid to critically ill patients, it may improve outcomes.

What doesn’t fit?

Most patients with hypovolemic shock, LV shock, and sepsis respond to appropriate therapy. Failure to respond should raise red flags, and drive an evaluation for obstructive shock. Obstructive shock is shock caused by an obstruction to venous return. Obstructions to venous return are often insidious. While volume resuscitation and therapy with vasoactives might produce a transient minor improvement in the circulation, the definitive treatment consists of relieving the obstruction if this is possible.

Interestingly, as a group, obstructions to venous return produce the kinds of variations in pulse pressure described above (Magder 2004, 2005). More recent clinical studies have reported that right ventricular shock can also produce an increase in SPV or PPV that is not responsive to fluid administration (Mahjoud). Hence, when a patient with significant pulse pressure variation (and a higher CVP) fails to respond to fluid administration, the sapient practitioner should entertain the possibility of right ventricular shock or an obstruction to venous return as the explanation. The expeditious evaluation of such patients includes a physical exam with careful attention to the character of the heart tones, chest wall symmetry and excursion, and abdominal wall tension. In patients with tense or distended abdominal walls, transducing a bladder pressure is very helpful in completing the evaluation for abdominal tamponade. In mechanically ventilated patients, the expiratory flow waveform should be evaluated for auto-PEEP. In most instances, a stat portable chest radiograph, trans-thoracic echocardiogram, and the measurement of a bladder pressure will be sufficient to complete the evaluation of a patient with refractory shock.
Causes of Obstructive Shock (Obstructions to Venous Return)
- pericardial effusion
- restrictive pericardium
- tension pneumothorax
- high levels of PEEP or intrinsic PEEP
- massive pleural effusion
- abdominal tamponade
- venous occlusion (clot, air, tumor, pregnancy)
- atrial occlusion (clot, air, tumor)

Reconciliation of Central Venous Pressures and Dynamic Indicators

The following 2x2 table is intended to aid practitioners in their assessment of patients with hypotension:

<table>
<thead>
<tr>
<th>PPV</th>
<th>CVP</th>
<th>Low</th>
<th>High</th>
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<tbody>
<tr>
<td>Low</td>
<td>???</td>
<td>Pump Shock</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>LV vs RV</td>
<td></td>
</tr>
<tr>
<td>High</td>
<td>Hypovolemic Shock</td>
<td>Hypovolemic vs RV shock vs Obstructive Shock</td>
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</tbody>
</table>

Studies using strategies similar to this are beginning to be reported in the literature (Benes).

References:
- Machare-Delgado E, Decaro M, Marik PE: Inferior Vena Cava Variation Compared to Pulse Contour Analysis as Predictors of Fluid Responsiveness: A Prospective Cohort Study Journal of Intensive Care Medicine 26(2) 116-124
- Michard F: Changes in Arterial Pressure during Mechanical Ventilation Anesthesiology 2005; 103: 419-28
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