Background:
Optimisation of circulation has proven outcomes benefit in sepsis, surgery, pediatrics, pregnancy and electrophysiology. Optimisation of preload, to optimize CO and oxygen delivery, is a critical component of circulatory optimisation and a common and critical clinical challenge often requiring central venous catheterization to measure venous pressure (CVP) or oxygenation (Scv02). In addition to being invasive, measurement of CVP is unreliable for determining ventricular preload fluid responsiveness or guiding fluid management. Sub-optimal circulation due to under filling or over filling may have critical clinical outcomes, and an improved non-invasive method of determining appropriate fluid management will contribute significantly to clinical practice.

Stroke volume (SV) is an important component of cardiac output (CO), and is critically dependent on the preload – the ventricular filling volume. The relationship between the ventricular filling volume and SV on any individual is described by a Frank-Starling curve and is valid at any fixed afterload. Preload is not only dependent on the intravascular blood volume but also the blood distribution. During erect standing, orthostasis, blood will pool in the dependent vessels of the circulation, thereby diminishing the preload, and subsequently the SV. Normally the circulation is maintained by the neurohumeral system upregulating both HR and SV. The capacity for the ventricle to upregulate SV in response to an increased preload is defined by the SV reserve. SV reserve can be defined as the change in SV associated with a fluid challenge and indicates the capacity for the ventricle to increase SV in response to increased preload. A positive SV reserve indicates the ventricular...
capability to increase SV and suggests value in administration of fluid. A negative SV reserve, defined as an unchanged or decreased SV in response to a fluid challenge, indicates no SV benefit to fluid administration. The legs are a significant fluid pool and can be emptied by simple leg lifting. This rapidly floods the atrium with approximately 200cm³ of blood; a significant fluid challenge, which results in increased preload and an increased SV in the preload deficient circulation.

Continuous Wave Doppler reliably and sensitively measures small changes in SV, and USCOM is a specialized non-invasive Doppler device for measurement of small serial changes in right and left sided haemodynamics, particularly SV and CO. This single observation identifies a simple method for determining SV reserve by changes in posture, and is a potentially important clinical maneuver for non-invasive determination of ventricular response to preload.

Method:
Normal adult male (53yrs) with no recent history of transfusion, haemorrhage or fluid imbalance, had an aortic haemodynamic assessment using USCOM after still standing for 5 minutes, with re-examination after supination, and supination with leg lift. Haemodynamic responses were observed. Each USCOM measure was the average of approximately 8 consecutive stroke cycles.

Results:
USCOM Examinations -
Stroke Volume and Posture – Case Study

Figure 1. USCOM measures after 5 minutes erect standing demonstrating an average SV for on screen measures of 61cm³.

Figure 2. USCOM measures after 5 minutes supine demonstrating an average SV for on screen measures of 84cm³.

Figure 3. USCOM measures after 5 minutes supine and a rapid leg lift demonstrating an average SV for on screen measures of 104cm³.
USCOM Doppler flow profiles standing, supine and supine with leg lift are demonstrated in figures 1, 2, and 3, demonstrating a progressive increase in SV. The changes in central haemodynamics measured by USCOM during the changes in body position are summarised in table 1 and figure 4.

<table>
<thead>
<tr>
<th></th>
<th>CO (L/min)</th>
<th>SV (cm³)</th>
<th>HR (bpm)</th>
<th>Vpk (cm/s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Supine</td>
<td>4.85</td>
<td>82</td>
<td>58</td>
<td>91</td>
</tr>
<tr>
<td>Standing</td>
<td>4.06</td>
<td>64</td>
<td>63</td>
<td>85</td>
</tr>
<tr>
<td>% Change</td>
<td>-16</td>
<td>-22</td>
<td>+9</td>
<td>-7</td>
</tr>
<tr>
<td>Supine+Lift</td>
<td>7.52</td>
<td>103</td>
<td>73</td>
<td>1.12</td>
</tr>
<tr>
<td>% Change</td>
<td>+55</td>
<td>+26</td>
<td>+26</td>
<td>+23</td>
</tr>
</tbody>
</table>

Table 1. lifting with % change referenced from supine values.

Figure 4. % changes of USCOM measured haemodynamic parameters after leg flexing referenced from standing values.
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In summary SVR decreased, while CO increased by component increases in both SV and HR.

Discussion:
These observations demonstrate that cardiovascular physiology is dynamic and variable, even during inactivity, as may occur in hospitalized subjects. The observed decrease in SV during standing is secondary to blood pooling in dependent extremities and diminishing the preload. The blood pooling results in increased peripheral venous pressures and SVR, and progressively reduced venous return, reducing preload and ultimately reduced SV. In the absence of an active pump support, such as leg motion, this process will continue. In this example simply flexing the lower limbs was sufficient to shift the pooled blood back into circulation, filling the atria and increasing the SV.

Conclusion:
CO is a complex and variable entity affected by subject position and the interaction of multiple complex entities, including autonomic function, and vascular pressure, a function of vascular compliance and blood distribution. The interplay of vascular pressure and cardiac performance is reflected in beat to beat changes in CO, HR and SV, all of which make the definition of meaningful population defined “normal” haemodynamic values challenging.

TeachingPoints:
Low or high CO may be real but not abnormal, particularly in subjects after prolonged standing or sitting as occurs in hospitalized patients. Quantitative determination of cardiovascular function is important for the reliable assessment and management of cardiovascular physiology and pathophysiology.
References: