Basic Critical Care Physiology and Its Applications in Therapy

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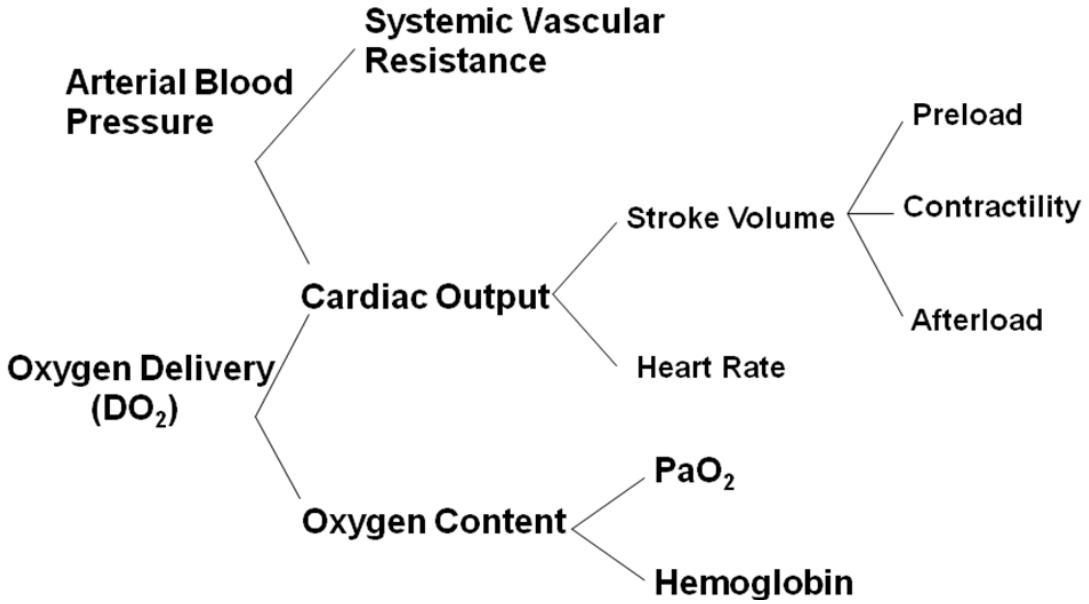
There are several basic physiologic concepts that are can be useful when applied to the practical management of critically ill patients. A basic understanding of these principles can provide a road marks to measure patient progress and offer hints for areas to improve treatment when evaluating your therapeutic plan. The following monograph offers a graphic overview of basic critical care physiology:



This lecture will look at five components of this physiologic monograph often seen in veterinary critical patient care.

1. Tissue perfusion. Oxygen delivery (DO2) is a product of Cardiac Output (CO) and Arterial Oxygen Content (CaO2). DO2 can be calculated using the following equation:

   DO2 = CO x CaO2 = CO x (1.34 x Hb x SaO2) x 10

   Philosophically, perhaps the end goal of critical patient care management is in doing what you can to improve oxygen delivery – or maximizing tissue perfusion. In order to do this, the clinician can first break this pursuit down into to two parts, cardiac output and arterial oxygen content. Efforts made to insure CO and CaO2 are in the normal to slightly supra-normal range will likewise insure DO2 is as best as it can be therapeutically driven.
2. Cardiac Output (CO). Cardiac Output is the product of Stroke Volume (SV) and Heart Rate

(HR). Certainly, this is a very basic physiologic concept, but it is also a useful one to revisit during you case management. Heart Rate should generally be kept above 60 bpm, and below an appropriate maximum given the size of your patient – practically less than ~200 bpm for larger patients and maybe 220 for smaller patients. Too fast a rate won't allow for adequate cardiac filling and too low won't allow for sufficient overall forward blood flow.

3. Stroke Volume (SV). Stroke Volume is controlled by Preload, Afterload, and Contractility. Generally, within its physiologic and anatomic limits, the heart will pump out whatever is taken in or returned from the body. Assuring adequate cardiac filling or improving cardiac filling within the individual heart's limits can enhance SV. Although not the same thing (ie volume does not equal pressure), Central Venous Pressure (CVP) can be used as a rough guide for SV. Afterload is the resistance that the heart pumps against when expelling blood from the ventricle. Resistances too high or too low are not beneficial. Blood pressure can be a practical guide or measure of systemic afterload. Contractility can be thought of as the overall "pump-ability" of the heart. Each heart has an inherent ability to perform as a pump. Helping the heart to work near its potential, without pushing it too far (ie beyond its abilities and thus into failure), is desirable for the critically ill patient.
4. Mean Arterial Blood Pressure (MAP). Mean arterial blood pressure can be thought of as the driving pressure, of force, of forward blood flow. Using Ohm's Law, MAP can also be thought of as the product of flow, or Cardiac Output, and resistance, typically Systemic Vascular Resistance (SVR). Keeping this relationship in mind, the clinician can take efforts to manipulate MAP by altering CO or SVR.
5. Arterial Oxygen Content (CaO2). The two major determinants of CaO2 are hemoglobin concentration [Hb] and the partial pressure of arterial oxygen (PaO2). CaO2 can be calculated using the following equation:

$$CaO2 = (1.34 \times [Hb] \times SaO2) + 0.003(PaO2)$$

SaO2 is the saturation of oxygen on the hemoglobin in arterial blood. The relationship between SaO2 and PaO2 is known as the oxygen dissociation curve.

Using these few physiologic concepts allows the clinician to address several commonly seen conditions in critically ill veterinary patients. By therapeutically adjusting the components of these concepts, as best as clinically possible, tissue perfusion and thereby oxygen delivery can be maximized. In the end, it is this optimization of oxygen delivery in the critical patient that is the primary goal of treatment in these patients.