

Cardiac output estimation based on arterial and venous blood gas analysis: proposal of a monitoring method

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Abstract

Hemodynamic optimization is vital in high risk surgical patients or in high risk surgical procedures. The main objective of hemodynamic management is to maintain tissue perfusion and preserve aerobic metabolism through a cardiac output coupled with the metabolic demand. The technologies used for cardiac output monitoring use special techniques (e.g. lithium dilution or transpulmonary thermodilution) or implementation of dedicated devices with considerable rates of potential complications (pulmonary artery catheter). Thus, we propose a novel method to estimate cardiac output through the analysis of arteriovenous blood gases which could be an alternative to more expensive methods (minimally invasive devices, pulmonary artery catheter). A review of several formulas described in the literature to compute the variables needed to calculate cardiac output with the Fick principle was carried out. These formulas estimate the oxygen consumption using the O₂ sensor integrated in the anesthesia workstation. The other variables in the Fick equation are derived from the arterial and venous blood gas analysis and parameters obtained from mechanical ventilators. By integrating the data gathered from the publications found, the authors created a comprehensive formula for calculation of cardiac output and the cardiac index using the parameters obtained from blood gas analysis. The presented method provides a more accessible and affordable way to monitor cardiac output in surgical high-risk patients in an environment with limited resources.

Key words: hemodynamic monitoring, cardiac output, blood gas analysis, monitoring, intraoperative, anesthesiology, critical care.

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Hemodynamic optimization is vital in high risk surgical patients. The aim is to achieve an adequate coupling between oxygen (O₂) supply and metabolic demands in an organism under surgical stress, thus preserving aerobic metabolism. Tissue perfusion does not necessarily correlate with arterial blood pressure. Instead, cardiac output is the most important variable that determines tissue oxygen supply.

Monitoring based on the pulmonary artery catheter is considered the gold standard for cardiac output (CO) assessment. It is an expensive and invasive method with usefulness in various clinical conditions being a matter of debate [1, 2].

Nowadays, there are minimally invasive hemodynamic monitoring methods available. They are based on the estimation of cardiac output through the analysis of the pulse wave contour, such as the FloTrac system (Edwards Lifesciences, Irvine, CA,

USA) that requires arterial cannulation, providing information on blood pressure, cardiac function and probability of response to volume. More advanced examples are the VolumeView system (Edwards Lifesciences, Irvine, CA, USA), which requires cannulation of the femoral artery and a central venous catheter to provide information on cardiac function and volumetric data. Data are obtained from the transpulmonary thermodilution method. Other examples, using dilution methods, are the PiCCO system (Pulsion Medical System, Munich, Germany) and LiDCO system (LiDCO Ltd, London, United Kingdom). These systems can provide information on the hemodynamic status of critically ill patients and can also be used in the perioperative setting. Although abovementioned devices have made it possible to improve the treatment of critically ill patients and optimize goal-directed therapy, they are expensive, not commonly available, and have

shown limitations in providing reliable data in different contexts [3–5].

One of the areas of recent progress in monitoring is the increasing availability of non-invasive methods. There are several techniques, such as applanation tonometry, for example, the T-Line system (Tensys Medical, San Diego, CA, USA), which has shown similar accuracy in blood pressure measurement compared to the arterial catheter [6]. However, it does not correlate with the pressures obtained from an arterial catheter in critically ill patients, and the use of vasoactive agents and patient movements affect the result of measurements [6–8]. Volume clamp methods, such as the Clear-Sight system (Edwards Lifesciences, Irvine, CA, USA) and CNAP system (CNSystems Medizintechnik, Graz, Austria) have an adequate level of compliance for the measurement of cardiac output compared to the pulmonary artery catheter and good correlation with transthoracic Doppler ultrasound [9, 10]. Even though the risk of complications with these devices is low, the capacity to measure cardiac output based on the pulse wave contour analysis has, according to a meta-analysis of 16 studies, a weighted percentage of error of 46.4% [11].

The German doctor Adolf Eugen Fick proposed a mathematical method to calculate cardiac output using oxygen consumption and arterial and mixed-venous blood oxygen content. This method, known as the Fick principle, has been a reference standard to compute cardiac output for the last century [12]. It is possible to use this resource to estimate cardiac output with data obtained from O₂ and carbon dioxide (CO₂) sensors built into anesthesia machines and from arterial and mixed venous blood gas analysis.

Arterial line and central venous catheter placement are common procedures in high risk surgical patients and the widespread availability of blood gas analysis in tertiary care centers makes the proposed method of CO estimation a reasonable alternative in the vast majority of patients, without the necessity of additional or special devices.

If we consider that O₂ consumption is equal to the product of CO multiplied by the arterio-venous difference of O₂ content, it is possible to rearrange the equation as follows:

$$VO_2 = (CO \times CaO_2 - CvO_2) \quad (1)$$

$$CO = \frac{VO_2}{(CaO_2 - CvO_2)} \quad (2)$$

where: VO_2 is oxygen consumption, CO is cardiac output, CaO_2 is arterial oxygen content and CvO_2 is venous oxygen content.

Data from arterial and venous blood gas analysis allows us to determine their respective oxygen con-

tents, thus completing the equation's (2) denominator [13].

$$CaO_2 = \{(Hg \times 1.36 \times SaO_2) + (PaO_2 + 0.0031)\} \times 10 \quad (3)$$

$$CvO_2 = \{(Hg \times 1.36 \times SvO_2) + (PvO_2 + 0.0031)\} \times 10 \quad (4)$$

where: CaO_2 is the oxygen content of arterial blood measured in mL L⁻¹, CvO_2 is the oxygen content of venous blood measured in mL L⁻¹, Hg is hemoglobin expressed in g dL⁻¹, the value 1.36 corresponds to the oxygen-carrying capacity per g of Hg (1.36 mL of O₂ per 1 g of Hg), SaO_2 is the arterial O₂ saturation in decimals, PaO_2 is the arterial O₂ pressure expressed in mm Hg, SvO_2 is the venous O₂ saturation in decimals, PvO_2 is the venous O₂ pressure expressed in mm Hg, and the value of 0.0031 corresponds to the dissolved or unbound oxygen fraction per each 100 mL of blood. Then the value is multiplied by 10 in order to convert the units from mL dL⁻¹ to mL L⁻¹.

The pulmonary artery catheter allows the monitoring of mixed venous oxygen saturation (SvO_2), while a central venous line allows measurement of central venous oxygen saturation ($ScvO_2$). The first one reflects global O₂ extraction, and the second one the upper body's extraction degree. Both variables assess the relationship between oxygen supply and consumption and tissue perfusion, thus allowing them to be assumed as an indirect reference to cardiac output. Because the use of SvO_2 and $ScvO_2$ has a positive influence on results of treatments of critically ill patients, it is possible to use these parameters as objectives during goal-guided therapy, $ScvO_2 > 70\%$ or a $SvO_2 > 65\%$ in both critically ill septic and non-septic patients being the fixed threshold during resuscitation [14].

Venous oxygen saturation differs between individuals and relies on O₂ extraction, which varies according to cell requirements. Inferior vena cava oxygen saturation is commonly higher than that in the superior vena cava. In the pulmonary artery (PA) there is a mixture of blood coming from both the upper and lower body, so the oxygen saturation in PA blood is an intermediate between both inferior and superior cava veins. In the right atrium, mixed blood is a partial mixture, so oxygen saturation depends on the degree of venous return and catheter tip location.

Intraoperatively, $ScvO_2$ can reach values up to 6% higher than SvO_2 . This depends on the effect of inhaled anesthetics on blood flow and cerebral oxygen extraction [15]. $ScvO_2$ and SvO_2 are similar in healthy patients, the former being 2–3% lower, due to the contribution from several vascular networks to the inferior vena cava, in which oxidative phosphorylation is reduced under certain conditions (renal, portal, hepatic flow). During shock, the coefficient of variation can exceed $\pm 20\%$ [14, 16].

It is explained by differential changes in blood flow and O_2 extraction (higher in brain and splanchnic circulation) [17]. Considering that in the absence of anemia and hypoxemia (which means adequate CaO_2), low values of SvO_2 and $ScvO_2$ correlate with a reduced cardiac output, trends in their values become helpful during resuscitation and intraoperative management.

$ScvO_2$ is clinically accessible and less invasive than SvO_2 , gaining more relevance. Studies carried out in critically ill patients have revealed that during parallel measurements of $ScvO_2$ and SvO_2 they correlate in 90% of cases, $ScvO_2$ being 7 ± 4 (%) higher than SvO_2 [18]. These findings suggest that $ScvO_2$ has the potential to represent changes in the O_2 supply/consumption relationship in critically ill patients; even when absolute numeric values of both $ScvO_2$ and SvO_2 are not the same, the trend of $ScvO_2$ values during serial measurements can serve as a guiding parameter.

It is also possible to obtain blood samples to measure atrial blood gases. Perez *et al.* [19] assessed the agreement between O_2 atrium blood saturation ($RAvO_2$) and SvO_2 in pediatric critically ill patients with catecholamine-resistant septic and cardiogenic shock. They found minimal differences (1–5% changes in SvO_2 represent changes in $RAvO_2$ in 79% of cases) with a concordance correlation coefficient of 0.90. Given that it is a venous mixture at the right atrium, global oxygen extraction could be better represented at this level than the one represented in the venous mixture sampled from the superior cava vein.

Given the abovementioned limitations and knowing the physiological differences between each type of venous blood and giving more relevance to trends than to absolute values, it is possible to skip the insertion of a pulmonary artery catheter and replace SvO_2 with $RAvO_2$ or $ScvO_2$ to estimate the arteriovenous O_2 difference through Fick's equation.

Although the estimation of O_2 consumption requires specialized measurement methods, such as indirect calorimetry, it is possible to approach it via information provided by an anesthesia workstation. Sykes suggested an equation to estimate O_2 consumption through the data from an oxygen sensor installed in an anesthesia breathing circuit when using low fresh gas flows [20]. The difference between the inspired oxygen fraction (FiO_2) and the expired oxygen fraction (ETO_2) corresponds to a proportion of minute ventilation, which allows oxygen consumption to be calculated:

$$VO_2 = (FiO_2 \times ETO_2) \times MV \quad (5)$$

where: VO_2 is "oxygen consumption", FiO_2 is the "inspired oxygen fraction", ETO_2 is "expired oxygen fraction" and MV is "minute ventilation".

Ritchie-Mclean and Shankar [21] suggested a modification to this formula by replacing minute ventilation with alveolar ventilation as minute ventilation includes dead space ventilation. This can enhance estimation of VO_2 with low tidal volumes.

$$VO_2 = (FiO_2 \times ETO_2) \times VA \quad (6)$$

where: VO_2 is in $mL \min^{-1}$, FiO_2 is in decimals, ETO_2 is in decimals, VA is alveolar ventilation in $mL \min^{-1}$.

The Bohr equation allows one to calculate alveolar volume using arterial CO_2 pressure ($PaCO_2$):

$$\frac{VD}{VT} = \frac{(PaCO_2 - ETCO_2)}{PaCO_2} \quad (7)$$

$$VD = VT \times \frac{(PaCO_2 - ETCO_2)}{PaCO_2} \quad (8)$$

where: VD is "dead space volume" in mL, VT is "tidal volume" in mL, $PaCO_2$ is in mm Hg, $ETCO_2$ is "end tidal CO_2 " in mm Hg.

The anesthesia workstation ventilator allows the control of tidal volume (VT). Considering that VT equals the sum of VD and VA , it is possible to estimate VA once "dead space ventilation" is known with equation 7.

$$VT = VD + VA \quad (9)$$

$$VA = VT - VD \quad (10)$$

Alveolar ventilation is calculated by multiplying VA by the respiratory rate (RR) set on the ventilator:

$$\dot{V}A = VA \times RR \quad (11)$$

where: $\dot{V}A$ is alveolar ventilation in $mL \min^{-1}$, VA is alveolar volume in ml, RR is the respiratory rate in breaths \min^{-1} .

Solving the equation by rearrangement of the variables results in equation 12:

$$\dot{V}A = RR \times \left\{ VT - \left[\frac{(PaCO_2 - ETCO_2)}{PaCO_2} \right] \right\} \quad (12)$$

Thus, using a modified Sykes equation (6) and arteriovenous blood gas values it is possible to calculate cardiac output:

$$CO = \frac{(VO_2)}{(CaO_2 - CvO_2)} \quad (2)$$

↓ replacing numerator by equation 6.

$$CO = \frac{[(FiO_2 \times ETO_2) \times VA]}{(CaO_2 - CvO_2)} \quad (13)$$

where,

$$\dot{V}A = RR \times \left\{ VT - \left[VT \times \left(\frac{(PaCO_2 - ETCO_2)}{PaCO_2} \right) \right] \right\} \quad (12)$$

Cardiac index is the result of the division between cardiac output and total body surface area. This value is more useful in pediatric patients.

Described method is based on monitoring devices routinely used in high risk surgical patients. Blood gas analyzers can be found in most perioperative units. Hence it would not be strictly necessary to have special equipment such as invasive monitoring devices with a pulmonary artery catheter or pulse contour analysis device, which are not generally available in all anesthesia services.

Arteriovenous gas analysis allows the clinician, in addition to cardiac output estimation, to assess perfusion parameters such as ΔCO_2 , O_2 extraction rate, oxygen supply (DO_2), and acid-base analysis (Δ hydrogen ion concentration, strong ion difference, serum lactate). Gomez-Duque *et al.* [22] proposed a formula to calculate pulmonary shunt:

$$Q_s/Q_t = \left[\frac{(1 - \text{SaO}_2)}{(1 - \text{SvO}_2)} \right] + (\text{FiO}_2 \times 0.13) \quad (14)$$

where: Q_s/Q_t is the relationship between shunt flow and total flow, SaO_2 is O_2 saturation in arterial blood gases, SvO_2 is oxygen saturation in central or atrium venous blood, FiO_2 is the inspired fraction of O_2 .

With central venous pressure (CVP), mean arterial pressure (MAP) and the previous calculation of cardiac output, the clinician can calculate systemic vascular resistance and the systemic vascular resistance index:

$$\text{SVR} = \frac{(\text{MAP} - \text{CVP})}{\text{CO}} \quad (15)$$

$$\text{SVRI} = \frac{(\text{MAP} - \text{CVP})}{\text{CI}} \quad (16)$$

In the Bohr equation, the CO_2 value corresponds to average CO_2 , not ETCO_2 . Using ETCO_2 to calculate dead space can lead to an underestimation of average CO_2 [23]. Using the Sykes equation without modifications could avoid variability since tidal volume (VT) is known and constant according to ventilatory parameters. Some anesthesia machines include volumetric CO_2 monitors that allow average CO_2 quantification with an adequate agreement compared to metabolic analyzers [24]. In case of having access to these machines, average CO_2 should replace the ETCO_2 value to provide higher accuracy in estimation.

In our opinion, the most relevant limitation of the proposed method is the impossibility to get continuous or real-time measurements, so we suggest an initial measurement before incision, followed by scheduled measurements to identify trends and determine changes in management. We propose hourly measurements in unstable or high-risk patients. The other patients could be tested every 2–3 hours or, depending on the clinical

criteria, according to the decision of the anesthesiologist in charge of the patient.

In neonates and infants, several difficulties in mechanical ventilation and higher respiratory rate can lead to inaccuracy in ETCO_2 values. With reduced blood volume, frequent blood sampling can lead to anemia and hemodynamic instability. That is why we recommend this method in patients weighing over 20 kg. If used in smaller patients, we propose sampling the minimal blood volume needed for processing blood gas analysis according to the equipment specifications of each institution, and also to distance as much as possible the time of sampling.

The method is subject to indirect estimations of some physiological variables. Estimating oxygen consumption using the Sykes equation has not been validated, so its use could be the principal source of error. Another problem is the impossibility to extract mixed venous blood with a central venous catheter. If possible, the anesthesiologist should position the tip of the catheter in the right atrium in order to get a percentage of blood mixture from both cava veins, getting closer to the values in the pulmonary artery. It is possible to achieve catheter placement by coupling a pressure transducer to the distal port of the central line looking for the ventricular waveform, then retracting the catheter until the waveform changes back to atrium characteristics. Another alternative to waveform analysis in catheter tip placement is the use of fluoroscopy during catheter insertion, but it implies additional resources, time and radiation exposure to the patient and medical staff.

According to the previous discussion, we consider trends analysis more important than relying on absolute and single measurements. Interpretation always must be subject to clinical information of the case. Further evaluations should be undertaken to validate this method compared to the gold standard (pulmonary artery catheter) or equivalents such as minimally invasive cardiac output monitoring devices.

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