Hemodynamic monitoring with two blood gases: “a tool that does not go out of style”

Monitoreo hemodinámico con dos gasometrías: “Una herramienta que no pasa de moda”


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Abstract

Hemodynamic monitoring of a critically ill patient is an indispensable tool both inside and outside intensive care; we currently have invasive, minimally invasive and non-invasive devices; however, no device has been shown to have a positive impact on the patient’s evolution; arterial and venous blood gases provide information on the patient’s actual microcirculatory and metabolic status and may be a hemodynamic monitoring tool. We aimed to carry out a non-systematic review of the literature of hemodynamic monitoring carried out through the variables obtained in arterial and venous blood gases. A non-systematic review of the literature was performed in the PubMed, OvidSP and ScienceDirect databases with selection of articles from 2000 to 2019. It was found that there are variables obtained in arterial and venous blood gases such as central venous oxygen saturation (Svo2), venous-to-arterial carbon dioxide pressure (Δpv-aCO2), venous-to-arterial carbon dioxide pressure/arteriovenous oxygen content difference (Δpv-aCO2/ΔCa-vO2) that are related to cellular oxygenation, cardiac output (CO), microcirculatory veno-arterial flow and anaerobic metabolism and allow to assess tissue perfusion status. In conclusion, the variables obtained by arterial and venous blood gases allow for non-invasive, accessible and affordable hemodynamic monitoring that can guide medical decision-making in critically ill patients.

Key words

Hemodynamic monitoring; blood gas; cardiac output; carbon dioxide; microcirculation.

Resumen

El monitoreo hemodinámico de un paciente en estado crítico es una herramienta indispensable tanto dentro como fuera de la terapia intensiva; actualmente se cuenta con dispositivos invasivos, mínimamente invasivos y no invasivos; sin embargo, ningún dispositivo ha demostrado tener impacto positivo en la evolución del paciente; la gasometría arterial y venosa proporcionan información del estado microcirculatorio y metabólico real del paciente pudiendo ser una herramienta de monitoreo hemodinámico. El objetivo de esta revisión fue realizar una revisión no sistemática de la literatura del monitoreo hemodinámico realizado mediante las variables obtenidas en la gasometría arterial y venosa. Se estudiaron las bases de datos de PubMed, OvidSP y ScienceDirect con selección de artículos del 2000 al 2019. Se encontró que hay variables obtenidas en la gasometría arterial y venosa como la saturación venosa central de oxígeno (Svo2), la diferencia de presión venoarterial de dióxido de carbono (Δpv-aCO2), la diferencia de presión venoarterial de dióxido de carbono/diferencia del contenido arteriovenoso de oxígeno (Δpv-aCO2/ΔCa-vO2) que están relacionadas con la oxigenación celular, con el gasto cardíaco (GC), con el flujo venoarterial microcirculatorio y con el metabolismo anaerobio que permiten realizar una valoración del estado de perfusión tisular. En conclusión, las variables obtenidas por gasometría arterial y venosa permiten realizar un monitoreo hemodinámico no invasivo, accesible y asequible que pueden guiar la toma de decisiones médicas en el paciente en estado crítico.

Palabras clave

Monitoreo hemodinámico; gasometría; gasto cardíaco; dióxido de carbono; microcirculación.

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INTRODUCTION

Monitoring, whether it be invasive, minimally invasive or non-invasive, supports clinical decision-making, hence its preeminent role in critical care. However, sufficient evidence on improved outcome data using one or another form of monitoring is still lacking (1). The most important thing will always be monere: warn or alert to prompt a decision before an event happens. The majority of the variables involved in hemodynamic monitoring hint at macrocirculation, because it appears to make sense that addressing macrohemodynamic variables should improve microcirculation (hemodynamic coherence); however, oftentimes this does not happen (2). Various techniques, methods and devices, ranging from clinical to invasive monitoring, are available to assess microcirculation abnormalities (3). In fact, across time, blood gases continue to prevail over lactate, base deficit (BD) or central venous oxygen saturation (SvO2), because we know today that venous-to-arterial carbon dioxide pressure difference (Δpv-aCO2) or venous-to-arterial carbon dioxide pressure arteriovenous oxygen content difference (Δpv-aCO2/ΔCa-vO2) reflect to cardiac output (CO), microcirculatory blood flow and anaerobic metabolism, which is perhaps the true target (4,5).

Clinical findings in shock are sensitive but not very specific when it comes to assessing microcirculatory blood flow, and practically of no account when it comes to anaerobic metabolism. Information provided by blood gases is a closer reflection of the true microcirculatory and metabolic status of the patient, as they show dysoxia and hemodynamic incoherence (6,7).

OBJECTIVE

To conduct a non-systematic review of the literature on hemodynamic monitoring using variables derived from arterial and venous blood gases.

METHOD

Non-systematic review of articles published between 2000 and 2020 in PubMed, OvidSP and ScienceDirect databases, using the following search terms in Spanish and English: hemodynamic monitoring, microcirculation, central venous oxygen saturation, venous-to-arterial carbon dioxide difference, venous-to-arterial carbon dioxide difference over arteriovenous oxygen content delta. Articles were selected according to title and abstract, and observational, review and original articles were included, while those that did not provide relevant information for the objective or were outside search dates were excluded. The results of this review were consistent with the authors’ perspective regarding relevance, and were included if at least four authors were in agreement (Figure 1).

DEVELOPMENT

Central venous oxygen saturation (SvO2)

A central venous catheter (CVC) and an arterial line (AL) are part of the devices required for patients in an intensive care unit (ICU) and samples from each of those lines can provide crucial information (8). The monitor is enough to gain insight into the macrocirculatory status but, for microcirculation, central venous and arterial blood gases are an excellent choice.

In healthy individuals, SvO2 is lower than mixed venous oxygen saturation (SvMVO2) by approximately 3%. This is explained by lower oxygen extraction (O2ER) from the lower body as compared to the upper body. However, this SvO2/SvMVO2 ratio is inverted in shock. In patients with septic shock, SvO2 is greater than SvMVO2 by up to 8%, as a result of increased O2ER from the lower body (gastrointestinal tract) (9). Some studies have shown that SvO2 may be an excellent proxy for SvMVO2 values. In pathological conditions, SvO2 rises or falls, with 70% being the normal or reference value (10).

SvO2 is a VO2/DO2 (oxygen consumption/oxygen delivery) dependent variable: it drops when oxygen transport is low (high VO2/DO2) and increases when oxygen usage is low (low VO2/DO2). When DO2 drops, compensation occurs through O2ER increase but, without the right intervention, the compensation mechanism is not sufficient, the target point being VO2 dependence on DO2. Up to that point (cell dyoxia) SvO2 drops in tandem with DO2 reduction and, from then on, anaerobic metabolism may give rise to disproportionate changes due to tissue hypoxia secondary to delays in timely and adequate interventions. Consequently, SvO2 reliably reflects cell oxygenation status. In the context of low SvO2, DO2 increase (in dependent zone) involves an increase in VO2 and, despite adequate intervention, SvO2 remains low and will increase only after VO2 is no longer dependent on DO2 (independent zone). Low SvO2 is not equal to DO2 increase (fluids, inotropes, vasopressors, beta-blockers, transfusion, oxygen) because such an intervention may have undesirable consequences. Rather, the answer is to reduce VO2 (pain control, sedation, mechanical ventilation, fever control, treatment for agitation, tremor or shivering control) and, consequently, perhaps the best option is a personalized intervention. High SvO2 may be an indication of impro-
vement, but also of inadequate VO2. Therefore, elevated SvcO2 does not rule out the need for therapeutic interventions and, in any case, whether it is low, normal or high, the best option is to add Δpv-aCO2 and Δpv-aCO2/ΔCa-vO2. Although lactate can be used, it is not the best option because it does not necessarily reflect tissue hypoxia or anaerobic metabolism; it should not be interpreted as a single or isolated variable, considering that there are non-hypoxic mechanisms that determine a higher reading [11,12].

O2ER represents the amount (%) of oxygen consumed (extracted) by cells from arterial blood. Non-extracted oxygen returns through the venous circulation (SvcO2) to the right cardiac cavities for reoxygenation in pulmonary circulation. In non-pathologic conditions, O2ER of 20-30% is considered normal. Any rise in O2ER (low SvcO2) reflects increased cell metabolism (hypoxia); in contrast, a drop in O2ER (high SvcO2) reflects diminished cell metabolism (hyperoxia) [13,14]. Defining hypodynamic or hyperdynamic requires looking beyond the clinical condition to include cardiac index < 2.5 L/min/m2, SvcO2 < 70% or O2ER > 30% (hypodynamic); or, conversely, cardiac index ≥ 4.0 L/min/m2, SvcO2 ≥ 80% or O2ER ≤ 20 % (hyperdynamic) [15,16] (Figure 2).

When a given SvcO2 value is analyzed, it involves the assessment of the interaction between all its determinants: 1) oxygen inflow, 2) transport 3) availability and 4) consumption [13] (Figure 3). To assess oxygen delivery and consumption in the tissues, knowledge of the following formulas is required [17]:

\[
DO2 = GC \times CaO2
\]
\[
VO2 = GC \times (CaO2 - CvO2)
\]
\[
EO2 = (CaO2 - CvO2) / (CaO2 - SwO2) / SwO2 / VO2 / DO2
\]
\[
EO2 = (1 - SvcO2)
\]
\[
CaO2 = (Hb \times 1,34 \times SaO2) + (PaO2 \times 0,003)
\]
\[
CvO2 = (Hb \times 1,34 \times SvcO2) + (PvO2 \times 0,003)
\]

Where: CaO2 is arterial oxygen content and CvO2 is venous oxygen content.

**FIGURA 2.** Central venous oxygen saturation and oxygen consumption and availability ratio.

**FIGURA 3.** Determinantes de la saturación venosa central de oxígeno.

**FIGURA 4.** Types of hypoxia.

\[ Δpv-aCO2: \text{carbon dioxide venous-to-arterial pressure delta, mmHg; millimeters of mercury.} \]

**SOURCE:** Authors.
Venous-to-arterial carbon dioxide pressure difference (Δp(v-a)CO2)

Carbon dioxide (CO2) provides even more valuable information on macro and microhemodynamics than oxygen (O2) variables. CO2 changes faster than lactate levels. It is the metabolic product of the Krebs cycle. In the context of aerobic metabolism, increased tissue CO2 reflects greater oxidative metabolism or a higher intake of dietary carbohydrates. On the other hand, this increase in CO2 may be due to greater anaerobic metabolism.

Δp(v-a)CO2 is obtained by means of one central venous and one arterial blood gas measurement and it is the difference between venous pCO2 and arterial pCO2. Its normal value ranges between 2 and 6 mmHg. Changes in Δp(v-a)CO2 are determined by the degree of blood flow (perfusion) and not by the degree of tissue hypoxia. Increases in Δp(v-a)CO2 are associated with a reduction in tissue blood flow (ischemic hypoxia) in controlled settings, as long as adequate oxygen delivery to the tissues is ensured. When Fick's equation is applied to CO2 metabolism, its clearance is found to depend on the difference between CO2 content in venous blood (CvCO2) and CO2 in arterial blood (CaCO2), multiplied by cardiac output or (CvCO2 - CaCO2) × CO. Consequently, the main determinant of changes in Δp(v-a)CO2 is CO and it is inversely proportional to it. Even at low CO, a patient can respond to hyperventilation with a normal or low Δp(v-a)CO2. Therefore, Δp(v-a)CO2 depends on CO, CvCO2 - CaCO2 difference, VCO2, alveolar ventilation and, to a lesser extent, on microcirculatory blood flow alterations. This means that Δp(v-a)CO2 is a bedside surrogate of CO and microcirculatory blood flow. Studies have shown that, during hypoxic hypoxia (normal blood flow and lowered O2 arterial pressure), Δp(v-a)CO2 is < 6 mmHg; in contrast, during ischemic hypoxia (diminished blood flow and normal O2 arterial pressure), Δp(v-a)CO2 is > 6 mmHg.

Venous-to-arterial carbon dioxide pressure difference/arteriovenous oxygen content delta (Δp(v-a)CO2/ΔC(a-v)O2)

The relationship between CO2 production (VCO2) and O2 consumption (VO2) is represented by the respiratory quotient (RQ = VCO2/VO2) which ranges between 0.6 and 1, depending on individual metabolic and energy conditions. In aerobic and resting conditions, VCO2 is not greater than VO2 and, therefore, the RQ is < 1; however, in anaerobic conditions, VCO2 is greater than VO2 resulting in a RQ > 1. Δp(v-a)CO2/ΔC(a-v)O2 is a useful surrogate of RQ = VCO2/VO2 since, according to Fick's equation, CO is present in the numerator and the denominator cancels out, removing the blood flow component and leaving the venous-to-arterial CO2 content difference (ΔCv-aCO2) and the venous-to-arterial O2 content difference (ΔCa-aO2) as the main determinant, providing the RQ result without the need for indirect calorimetry. Because it is less invasive and offers a good correlation, Δp(v-a)CO2 has replaced ΔCv-aCO2 in obtaining Δp(v-a)CO2/ΔC(a-v)O2 through one central venous and one arterial blood gas measurement, which allows to identify those patients in an anaerobic state.

Δp(v-a)CO2/ΔC(a-v)O2 > 1 is associated with microcirculatory abnormalities that lead to O2ER reduction which, added to the drop in CO, results in a lower DO2. Moreover, the increase in anaerobic CO2 favors cell dysoxia. If shock is reverted promptly Δp(v-a)CO2/ΔC(a-v)O2 can return to values < 1. Evidence suggests that improved microcirculatory blood flow distribution can revert anaerobic metabolism.

Elevated lactate does not always reflect tissue hypoxia or anaerobic metabolism and should, therefore, be used together with Δp(v-a)CO2/ΔC(a-v)O2 in order to provide more accurate information at any point during patient assessment.
Reduce VO₂

Increase DO₂?

Cardiac output > 6 mmHg

No intervention

Heart rate

Preload

Contractility

Afterload

Betablocker

Fluids

Inotrope

Vasopressor

Transfusión

PEEP

Oxygen source: Authors.

CO₂: carbon dioxide, VCO₂/VO₂: carbon dioxide production/oxygen consumption, Δpv-aCO₂/ΔCa-VO₂: venous-to-arterial carbon dioxide pressure delta/arteriovenous oxygen content delta.

SOURCE: Authors.

Hemodynamic monitoring using blood gas analysis has been and will continue to be a bedside diagnostic tool that enables optimum and timely intervention. Whether static or dynamic, the ideal hemodynamic monitoring method does not exist. Measurement interpretations and decision-making will always be operator-dependent, creating advantages and disadvantages. Blood gas analyzers are readily available in any hospital, unlike sophisticated monitors. Δpv-aCO₂ and Δpv-aCO₂/ΔCa-VO₂ are excellent markers or microcirculatory blood flow and anaerobic metabolism, respectively. Hemodynamic monitoring using two blood gases measurements is an option that allows to establish a diagnostic and therapeutic pathway and work on the premise of not more, not less, only what is needed.

**CONCLUSION**

**ETHICAL RESPONSIBILITIES**

**Human and animal protection**

The authors declare that no experiments were carried out in humans or animals for this research.

**Data confidentiality**

The authors declare having followed the protocols of their institution on patient data disclosure.

**Right to privacy and informed consent**

The authors declare that no patient data appear in this article.
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Appreciations

Conflict of interest

None declared.

None declared.

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References


