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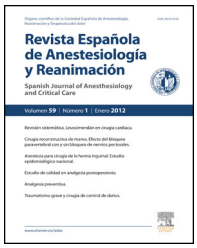

REVIEW

Prognostic value of gasometric parameters of carbon dioxide in resuscitation of septic patients. A bibliography review☆

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KEYWORDS
Septic shock;
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Lactic acid;
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Patient outcome assessment

Abstract The anaerobic metabolism is the cornerstone in physiopathology of septic shock. Nowadays we have both the central or mixed venous oxigen saturation and lactate levels to monitoring the metabolism in septic patients. Some studies have shown that normalisation of systemic haemodynamic and oxygen metabolism variables not prevent progression to multiorgan damage and death.

Recently has been proposed the venous-to-arterial carbon dioxide difference ($\Delta pvaCO_2$) as an alternative marker of tissue hypoperfusion, like Cardiac Index. High $\Delta pvaCO_2$ predicts adverse outcomes. Also has been proposed both, the ratio between the $\Delta pvaCO_2$ and arterial-to-venous oxygen content difference ($\Delta CavO_2$): $\Delta pvaCO_2/\Delta CavO_2$; and, the ratio between venous-to-arterial carbon dioxide difference ($\Delta CvaCO_2$) and $\Delta CavO_2$: $\Delta CvaCO_2/\Delta CavO_2$, as markers of anaerobic metabolism. Both of high ratios are related to high levels of lactate and worse prognosis. Therefore in patients with sepsis the combination of markers of resuscitation could be important to improve the outcomes.

PALABRAS CLAVE
Shock séptico;
Dióxido de carbono;
Ácido láctico;

Valor pronóstico de los parámetros gasométricos del dióxido de carbono en pacientes con sepsis. Una revisión bibliográfica

Resumen El metabolismo anaerobio es clave en la fisiopatología del shock séptico. Actualmente disponemos de la saturación venosa central y mixta, y del lactato para monitorizar estos

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Análisis de los gases de la sangre;
Consumo de oxígeno;
Evaluación del resultado de la atención al paciente

pacientes. Hay trabajos que han demostrado que la normalización de parámetros hemodinámicos y de oxigenación no previene la progresión del daño orgánico o de mayor mortalidad.

Recientemente se ha propuesto como marcador de hipoperfusión tisular la diferencia venoarterial de pCO_2 ($\Delta pvaCO_2$), equivalente al gasto cardiaco; un gradiente elevado se ha relacionado con resultados adversos. Así mismo se ha propuesto la ratio $\Delta pvaCO_2$ entre diferencia de contenido arteriovenoso de oxígeno ($\Delta CavO_2$): $\Delta pvaCO_2/\Delta CavO_2$, también la ratio diferencia de contenido venoarterial de CO_2 entre $\Delta CavO_2$: $\Delta CvaCO_2/\Delta CavO_2$, como marcadores de metabolismo anaerobio. Ambas ratios elevadas están relacionadas con mayores niveles de lactato y peor pronóstico. Por lo tanto, en pacientes con sepsis pudiera ser importante la combinación de objetivos de reanimación para mejorar los resultados.

Introduction

Mortality in sepsis and septic shock has fallen considerably in recent years. This, however, is probably due more to increased awareness of sepsis among clinicians and earlier diagnosis and treatment of the disease, than to the impact of the measures proposed in the clinical practice guidelines published by the Surviving Sepsis Campaign (SSC).[1] Recent multicentre trials (ProCESS,[2] ARISE,[3] ProMISE[4]) and a meta-analysis[5] of randomised clinical trials have challenged the findings of the classic study on early, time- and resource-intensive goal-directed treatment of sepsis published by Rivers.[6]

Today, the global perfusion status of sepsis patient is determined by measuring blood oxygen saturation (central venous saturation [$ScvO_2$] or mixed venous saturation [SvO_2]) and serum lactate levels. Several different guidelines[1,7] have recommended using these parameters as markers of tissue perfusion. There are, however, limitations to their usefulness, and $ScvO_2 \geq 70\%$ is associated with increased mortality.[8] Serum lactate levels are not without their limitations either,[9] because hyperlactaemia in septic shock is not always caused by tissue hypoxia.[10] For a more accurate picture, therefore, both markers must be supplemented with other determinations.[11]

Some authors have suggested using, in addition to the foregoing, venous and arterial carbon dioxide (CO_2) measurements as haemodynamic, metabolic and prognostic markers in septic shock.[6,12]

Carbon dioxide as a marker of blood flow and anaerobic metabolism

In physiological conditions, CO_2 is transported in the blood in 3 ways: (1) dissolved in plasma (8%); (2) buffered with water as HCO_3^- and H_2CO_3 (82%), in equilibrium with dissolved CO_2; and (3) bound to proteins, mainly haemoglobin (Hb) (10%). The relationship between CO_2 content (CCO_2) and PCO_2 is taken to be almost linear.[13] In tissue hypoxia, the proportion of CO_2 transported by Hb increases while pCO_2 remains the same. This is because hypoxia increases the affinity of Hb for CO_2 in a phenomenon called the Haldane effect.[13–15] CO_2 transport is also affected by Hb levels, acidosis, $SatO_2$, exercise, stress, etc.[16]

Carbon dioxide, which is an end product of cellular metabolism that does not accumulate in tissue, has a high diffusion capacity, does not metabolise in tissue, and is only excreted through the lungs, is a good marker of tissue hypoxia. Although some patients present CO_2 retention, this confounding factor can be neutralised. CO_2 level, therefore, is a promising indicator of anaerobic metabolism.[7,12,16,17]

Carbon dioxide as a marker of systemic blood flow

Fick's equation[13,14] shows that CO_2 production (VCO_2) (Fig. 1) equals the product of cardiac output (Q) by the difference between CCO_2 in venous and arterial blood ($C_vCO_2 - C_aCO_2 = \Delta CvaCO_2$): $VCO_2 = Q \times (\Delta CvaCO_2)$. The relationship between pCO_2 and CCO_2 is assumed to be linear in physiological conditions, which suggests that pCO_2, can be taken as a surrogate measure of CCO_2, thus: $\Delta CvaO_2 = \Delta PCO_2$.[13,16] Therefore, $VCO_2 = Q \times \Delta PCO_2$, $\Delta PCO_2 = VCO_2/Q$; thus ΔPCO_2 is directly proportional to

$CaO_2 = (1.34 x SatO_2 x [Hb]) + 0.003 x paO_2$ (mL dL-1)

$CvO_2 = (1.34 x SvtO_2 x [Hb]) + 0.003 x pvO_2$ (mL dL-1)

$\Delta CavO_2 = (CaO_2 - CvO_2)$ (mL dL-1)

$DO_2 = 10 x CI x CaO_2$ (mL min-1 m-2)

$VO_2 = 10 x CI x \Delta CavO_2$ (mL min-1 m-2)

$O_2ER = \Delta CavO_2/CaO_2$ (%)

$\Delta pvaCO_2 = (pvCO_2 - paCO_2)$ (mmHg o kPas)

$\Delta pvaCO_2/\Delta CavO_2 = (pvCO_2 - paCO_2)/(CaO_2 - CvO_2)$ (mmHg dL mL O_2-1)

$\Delta CvaCO_2/\Delta CavOv = (CvCO_2 - CaCO_2)/(CaO_2 - CvO_2)$

Figure 1 Blood gas equations and measurements (units). CaO_2: arterial oxygen content; $CaCO_2$: arterial CO_2 content; $CvCO_2$: venous CO_2 content; CvO_2: venous oxygen content; DO_2: oxygen delivery; CI: cardiac index; O_2ER: oxygen extraction ratio; $paCO_2$: partial pressure CO_2; paO_2: partial pressure of oxygen; $pvCO_2$: venous pressure of CO_2; VO_2: oxygen uptake, $\Delta CvaO_2$: venous-arterial oxygen content difference; $\Delta pvaCO_2$: venous-arterial pCO_2 difference.