Review Article

Regional capnometry to evaluate the adequacy of tissue perfusion

Stéphane Bar[1], Marc-Olivier Fischer[2]

[1]Anesthesiology and Critical Care Department, Amiens University Hospital, Amiens, France; [2]Normandie Univ, UNICAEN, CHU de Caen Normandie, Service d'Anesthésie Réanimation, Caen, France

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Correspondence to: Dr. Stéphane Bar. Anesthesiology and Critical Care Department, Amiens University Hospital, Rond-point Fernand Leger, F-80054 Amiens, France. Email: stephane.bar.sb@gmail.com.

Abstract: Tissue hypoperfusion is a major cause of morbidity and mortality in critically ill patients but cannot always be detected by measuring standard whole-body hemodynamic and oxygen-related parameters (e.g., blood pressure, cardiac output, and central venous oxygen saturation). Preclinical and clinical studies have demonstrated that low-flow states are consistently associated with large increases in venous and tissue PCO_2. Monitoring regional PCO_2 with gastric tonometry ($PgCO_2$) is known to have independent prognostic value for predicting postoperative complications and mortality. The $PgCO_2$ gap might also be of value as a treatment target (endpoint) in critically ill patients. However, this tool has several limitations and has not yet been developed commercially, thus restricting its use. Regional capnography with sublingual and transcutaneous sensors might be an alternative noninvasive option for evaluating the adequacy of tissue perfusion in critically ill patients. However, further studies are needed to determine whether or not this monitoring technique is of value—particularly as an endpoint for guiding resuscitation. Bladder PCO_2, has only been evaluated in animal studies, and so remains to be validated in patients.

Keywords: Regional capnography; anaerobic metabolism; hypoperfusion

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Introduction

In clinical practice, it is difficult to find relevant hemodynamic and oxygenation parameters that can serve as titration endpoints for hemodynamic interventions. In patients with shock or having undergone major surgery associated with high incidence of postoperative complications, the accepted goal for hemodynamic optimization is to increase O_2 delivery (DO_2) and thus O_2 consumption (VO_2) (1). However, recent studies have failed to confirm that hemodynamic optimization reduces morbidity and mortality (2-4). Even when macrocirculatory targets are met, microcirculatory disturbances can persist and lead to organ dysfunction (5). Hence, improved hemodynamic management might only be achieved by detecting VO_2's responsiveness to an increase in DO_2 (i.e., in patients with anaerobic metabolism) (6). To date, several variables have been described as markers of tissue perfusion: oxygen venous saturation (SvO_2) (7), the veno-arterial PCO_2 gradient (PCO_2 gap) (8), the arterial lactate level, and the ratio of the veno-arterial PCO_2 gradient to the arteriovenous content difference in O_2 (i.e., the PCO_2 gap/$DavO_2$ ratio) (9-12). Although these variables have been studied in intensive care units (ICUs) and operating theaters, they do have some limitations (described in other chapter of this publication). Moreover, these conventional variables are markers of systemic hypoperfusion, and so are not able to detect regional hypoperfusion (5).

In the 1990s, Nakagawa *et al.* and Tang *et al.* suggested that an increase in tissue PCO_2 ("stagnant hypercapnia") was a marker of inadequate tissue perfusion (13,14). Indeed, the difference between regional tissue PCO_2 and $PaCO_2$

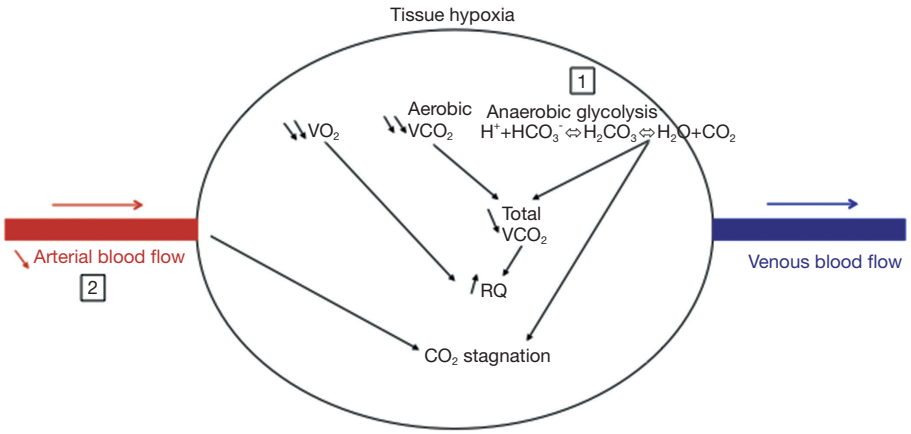

Figure 1 Mechanisms of the CO_2 stagnation in the tissues. [1] During tissue hypoxia, aerobic CO_2 production (VCO_2) falls as a result of a decrease in oxygen consumption (VO_2). However, anaerobic glycolysis increases, leading to the excessive production of lactic acid and thus the generation of H+ ions. The latter are buffered by bicarbonates, and so anaerobic VCO_2 increases in the cell. This results in a smaller reduction in total VCO_2 than in VO_2 and thus an increase in the respiratory quotient (RQ) (VCO_2/VO_2 ratio). [2] Tissue stagnation of CO_2 can occur only when blood flow is abnormally low.

(the tissue-arterial PCO_2 gap) is an earlier, more accurate marker of regional tissue hypoperfusion than whole-body parameters are (15,16). This concept has been validated in many animal models and clinical studies (13,17-20). From a physiological point of view, an increase in tissue PCO_2 results from two mechanisms that must both be present to produce "stagnant hypercapnia". Firstly, an increase in tissue CO_2 which can results of rises in aerobic metabolism with greater CO_2 generation by the cells or results of tissue hypoxia with an increase in anaerobic glycolysis and excessive production of lactic acid (17). Secondly, the maintenance of blood flow easily removes CO_2 into the venous circulation via the "washout phenomenon" (21). Thus, stagnant hypercapnia can occur only when blood flow is abnormally low. Thus, PCO_2 gap increases as cardiac output falls in case of tissue hypoxia or without tissue hypoxia (22,23) (*Figure 1*).

These findings have been confirmed by some clinical studies (18,24,25)—most notably by Vallet *et al.*'s study of limb-PCO_2 gap (26). These researchers demonstrated that the PCO_2 gap increased when the DO_2 fell after a reduction in blood flow (ischemic hypoxia) but not when DO_2 fell with maintenance of blood flow (hypoxic hypoxia). These results have been confirmed in animal studies of the tissue-arterial PCO_2 gap (19,20); the latter increased during ischemic hypoxia but not during non-ischemic hypoxia.

Hence, these findings suggest that the tissue-arterial PCO_2 gap is a marker of tissue hypoperfusion in general, and not just in cases of hypoxia. The normal reference range for the tissue PCO_2 gap is 8 to 10 mmHg (20,27).

The objectives of the present review were to describe the sites at which regional PCO_2 and tissue-arterial PCO_2 gap have been measured (gastric, sublingual, transcutaneous and bladder sites), assess this parameter's prognostic value, and evaluate its utility in goal-directed therapy.

Gastric intramucosal PCO_2 ($PgCO_2$)

The tonometric measurement of regional CO_2 pressures is based on equilibration of a gas's partial pressure between two compartments separated by a semi-permeable membrane. Using air or saline as an equilibration medium enables the gas analyzer to automatically measure the PCO_2 at a balloon located at the end of a gastric tube (*Figure 2*). The PCO_2 in the collected air is measured using infrared spectrometry. The stomach is easy to access, and is known to be highly sensitive to tissue hypoperfusion (28). Furthermore, $PgCO_2$ measurements have been used to detect early splanchnic ischemia (29).

In the event of tissue hypoxia and low VO_2, CO_2 production by the gastric mucosa increases. Thus, it has been suggested that $PgCO_2$ is a marker of tissue hypoxia (30) and can predict morbidity and mortality in critically ill patients (31). However, as mentioned in the introduction, the $PgCO_2$ – $PaCO_2$ gradient ($PgCO_2$ gap) might be more valuable because it reflects the adequacy

of gastric mucosal blood flow. In critically ill patients, the $PgCO_2$ gap values measured on admission to the ICU and 24 h later constituted an independent prognostic factor for 28-day mortality, with a cut-off of >20 mmHg (16). In a perioperative setting, this index was predictive of postoperative complications (27,32).

When used as a prognostic tool in critically ill patients, the $PgCO_2$ gap decreases upon fluid challenge. The change is related to the baseline $PgCO_2$: PCO_2 gap responders were defined by a decrease of more than 3 mmHg. Even more interestingly, whole-body indexes of oxygenation (SvO_2, VO_2 and PCO_2 gap) remained unchanged after fluid challenge, when the $PgCO_2$ gap decreased (33).

The main limitations of this method are the need for concomitant H_2-blocker use and the discontinuation of enteral feeding (34). Moreover, this type of device has not been developed commercially, thus limiting its availability.

Esophageal tonometry has been proposed as a convenient alternative to gastric tonometry in animal models of shock (35,36). Good inter-variable correlations were found at this measurement site. However, this site is more difficult to access, which explains its scarce use in clinical practice.

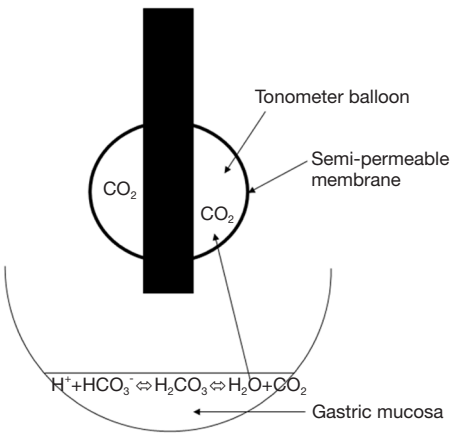

Figure 2 Gastric intramucosal PCO_2.

Sublingual PCO_2 ($PslCO_2$)

The most intensively developed and well-studied sublingual CO_2 sensor is the CapnoProbe® CO_2-sensing optode (Nellcor, Pleasanton, CA, USA) (31,37-40). The optode contains a CO_2-sensing fluorescent dye that is excited by light conducted through an optical fiber. The emitted fluorescence is then transmitted back to the instrument (*Figure 3*) (31).

Interest in the sublingual region has been stimulated by orthogonal polarization spectral imaging studies that have evidenced a decrease in sublingual capillary density in the event of septic shock (41,42). A study performed in the 1980s found that $PslCO_2$ was elevated in a model of hemorrhagic shock (43). In animal studies of hemorrhagic and septic shock, sublingual CO_2 measurements are well correlated with $PgCO_2$ and whole-body markers of tissue hypoperfusion (13,43,44). The main advantages of this technique relate to its noninvasive nature, the absence of a requirement for withdrawing enteral feeding, and the correlation with the splanchnic region.

The basal value of $PslCO_2$ was found to be predictive of mortality in acute circulatory failure and was associated with arterial lactate levels. When $PslCO_2$ exceeded a threshold of 70 mmHg, its positive predictive value was excellent. Conversely, $PslCO_2$ fell more quickly than arterial lactate during resuscitation (45).

However, the most interesting marker appears to be the $PslCO_2$ – $PaCO_2$ gradient (the $PslCO_2$ gap). It is reportedly a better prognostic factor than whole-body markers (SvO_2, cardiac index, DO_2, and arterial lactate), and the best cut-off value was 25 mmHg (15). The $PslCO_2$ gap may serve as an index of tissue dysoxia and the severity of tissue

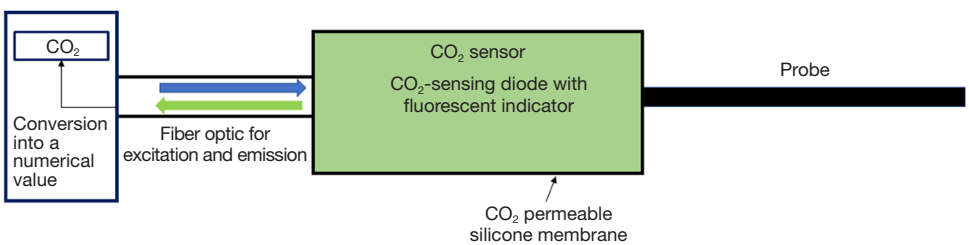

Figure 3 Sublingual PCO_2 measurement with the CapnoProbe®. The optode contains a CO_2-sensing fluorescent dye that is excited by light conducted through an optical fiber. The emitted fluorescence is then transmitted back to the instrument.

hypoperfusion in critically ill patients (38,39).

With regard to the $PslCO_2$ gap's potential as a treatment target (endpoint), it has been showed that the reperfusion of the damaged sublingual microcirculation (assessing using orthogonal polarization spectral imaging) was associated with the normalization of the $PslCO_2$ gap during the resuscitation of patients with septic shock (37). However, the $PslCO_2$ gap's potential value as an endpoint during resuscitation has not yet been evaluated in critically ill patients. Although, this technique has been tested at the bedside, the CapnoProbe® is no longer commercially available.

Transcutaneous PCO_2 ($PcCO_2$)

Transcutaneous measurement of tissue CO_2 is a simple, noninvasive technique. Interest in this method has been stimulated by studies of capnometry at the earlobe (46). The system includes a Severinghaus heated PCO_2 electrode and a pulse oximetry sensor clipped to the earlobe (TOSCA® 500 monitor, Linde Medical Sensors, Basel, Switzerland). In a study of patients in the ICU, the transcutaneous PCO_2 ($PcCO_2$) was well correlated with $PaCO_2$ (47-49). Other devices based on the same technology (SenTec AG, Basel, Switzerland) have yielded the same accuracy (50). However, a recent study found that measurement repeatability was poor (51).

Another study focused on the gradient between $PcCO_2$ and $PaCO_2$ (the $PcCO_2$ gap) (52). The baseline $PcCO_2$ gap levels were significantly higher in patients with septic shock, and the decrease after resuscitation was significantly greater in survivors than in non-survivors. Interestingly, survivors and non-survivors did not differ with regard to the change over time in whole-body parameters (cardiac output and $ScVO_2$). A $PcCO_2$ gap above 16 mmHg on day one was associated with a poor outcome. Interestingly, the variations in the $PcCO_2$ gap during fluid challenge were inversely correlated with changes in microcirculatory skin blood flow.

Even though a large number of studies have investigated transcutaneous PCO_2 in the field of neonatology, a Cochrane Collaboration review concluded that there was no evidence to recommend the use of transcutaneous CO_2 monitoring in neonates (53).

In critically ill patients, the $PcCO_2$ gap might be of value as an additional resuscitation endpoint when other parameters (e.g., arterial lactate and $ScVO_2$) are in the normal range despite persistent tissue hypoperfusion (54). However, well-designed, adequately powered, randomized, controlled studies of the efficacy and safety of transcutaneous CO_2 monitoring are now required.

Bladder PCO_2 ($PbCO_2$)

The results of several animal studies have suggested that monitoring the intramucosal PCO_2 in the bladder ($PbCO_2$) may be a minimally invasive technique for monitoring perfusion (55-57). This technique measures $PbCO_2$ via the gas analysis of saline samples collected from the balloon of a Foley catheter inserted into the bladder. The $PbCO_2$ value is well correlated with DO_2 and $PgCO_2$ (55,56). However, these results were not confirmed by another group of researchers (57). Clinical studies of the accuracy of this device are required.

Conclusions

Conventional whole-body hemodynamic markers cannot always predict tissue hypoperfusion. By analogy with measurement of the whole-body PCO_2 gap, the tissue PCO_2 gap has been described as a marker of blood flow adequacy and can be used to detect tissue hypoperfusion. Monitoring $PgCO_2$ gap has given good results, although several technical limitations and failure to develop this tool commercially has prevented the wider use of this technique. Further studies are needed to assess the efficacy and safety of $PslCO_2$ gap and $PcCO_2$ measurements. Although measurement in the bladder are promising, $PbCO_2$ must be now studied in patients. Lastly, the blood flow distribution across the various organs cannot yet be assessed.

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Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

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