

Title: VENO-ARTERIAL CARBON DIOXIDE TENSION DIFFERENCE: CORRELATION WITH HEMODYNAMICS IN CRITICALLY ILL PATIENTS

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Introduction. Increased veno-arterial PCO₂ difference, or P(v-a)CO₂, has been documented in cardiopulmonary resuscitation (CPR) [1], and in canine models of endotoxemia [2] and hemorrhagic shock [3]. We have empirically observed high P(v-a)CO₂ in critically ill patients with low cardiac output (Q_T), and low P(v-a)CO₂ in hyperdynamic states. Furthermore, we have noticed that changes of Q_T occurring in the same patient, are usually accompanied by inverse changes of P(v-a)CO₂. The present study was therefore designed to test the hypothesis that, in the critically ill patients, there exists an inverse correlation between cardiac output and P(v-a)CO₂, and that monitoring P(v-a)CO₂ may provide a qualitative assessment of the blood flow status.

Materials and Methods. The study was approved by the Mount Sinai School of Medicine Institutional Review Board. All consecutive patients admitted to the surgical intensive care unit between January 15, 1988 and March 31, 1988 were entered in the study if their clinical condition necessitated the insertion of a pulmonary artery catheter. When a cardiac output determination was mandated for the patient's optimum management, both arterial and pulmonary arterial blood gases were also measured. Thermodilution cardiac outputs were determined in triplicate using a Hewlett-Packard cardiac output computer, while the arterial and mixed venous blood gases were immediately measured, using a blood gas analyzer (Instrument Laboratory IL 1302). Data were analyzed with multiple regression (PROC REG, STEPWISE) and correlation (PROC CORR) procedures, using the SAS statistical package for microcomputers. Two sets of statistical analysis were performed: one on the aggregate number of data points collected for all patients, and the other on the admission data only. P value < 0.05 was accepted as significant.

Results. Sixty patients were eligible for the study. They presented with a variety of major pathological conditions, as listed in Table 1. The patients' ventilatory status varied from spontaneous breathing on room air, to controlled ventilation with FiO₂ 1.0 and positive end expiratory pressure (PEEP) 22 cm H₂O. Three hundred and sixty six data points were collected. There was a wide variety of hemodynamic and blood gas data. (See Table 2). The study shows a strong negative correlation between P(v-a)CO₂ and Q_T (r = -0.69, p < 0.0001). Other significant correlations are listed in Table 3. As a dependent variable, P(v-a)CO₂ was best predicted by Q_T (r² = 0.47, p < 0.0001), then by venous pH, PvO₂, PaO₂, PvCO₂, and PaCO₂. Further, the correlation between P(v-a)CO₂ and Q_T appears stronger than that between PvO₂ and Q_T. The analysis performed on the admission data point confirmed these results.

Discussion. This study in unselected surgical critically ill patients demonstrates that P(v-a)CO₂ has a strong negative correlation with cardiac output. Thus, monitoring P(v-a)CO₂ may provide a qualitative assessment of the perfusion status. This may be useful both during anesthesia or in the ICU when, for example, cardiac output determination is not practical in a patient who has indwelling arterial and pulmonary artery catheters. The importance of venous blood gas monitoring has been increasingly recognized. Most applications, however, concern the mixed venous oxygen (PvO₂ or SvO₂) and its relationship with tissue perfusion. Mixed venous oxygen, in addition to cardiac output, is also a reflection of other factors that affect either the oxygen delivery (such as hemoglobin concentration), or the O₂ uptake and utilization (microcirculatory homogeneity, efficiency of oxidative phosphorylation). By contrast, P(v-a)CO₂ may be more affected by Q_T than by other factors. As such, it may provide a better qualitative assessment of Q_T than PvO₂ or SvO₂. Further studies are needed to clarify the usefulness and limitations of P(v-a)CO₂ monitoring, and to determine whether, in this application, central venous blood can be substituted for pulmonary blood, thereby decreasing the invasiveness of this monitoring.

References.

1. Weil MH, et al: N Engl J Med 1986; 315:153-156.
2. Paluch TA, et al: Anesthesiology 1985; 63:A158.
3. Benjamin E, et al: Crit Care Med 1987; 15:516-518

TABLE 1
PATHOLOGICAL CONDITIONS

CHF and cardiogenic shock	15
Sepsis/septic shock	17
S/p abdominal aortic aneurysmectomy	13
S/p major non vascular surgery	17
Peritonitis	8
ARDS or severe pneumonitis	9
Diabetes mellitus	9
COPD	5
S/p cardiopulmonary resuscitation	1
Hemorrhagic shock	3
Cirrhosis of the liver	5

TABLE 2
HEMODYNAMIC AND BLOOD GAS DATA

Variables	Mean	S.D.	Min	Max
Q _T (L·min ⁻¹)	4.9 ±	1.9	1.3	11.8
SV (mL·beat ⁻¹)	51.8 ±	21.5	10.0	141.1
PaO ₂ (mmHg)	136.9 ±	91.0	41.0	526.0
PvO ₂ (mmHg)	36.4 ±	6.0	18.0	55.0
PaCO ₂ (mmHg)	34.4 ±	6.1	16.1	54.5
PvCO ₂ (mmHg)	39.9 ±	5.8	23.8	60.4
P(v-a)CO ₂ (mmHg)	5.5 ±	2.4	0.7	16.2

TABLE 3
CORRELATION COEFFICIENTS

P(v-a)CO ₂	Q _T	SV	PvO ₂	PvCO ₂	PaCO ₂
P(v-a)CO ₂	-0.69	-0.63	-0.48	0.06	-0.33
Q _T		0.83	0.47	0.10	0.36
SV			0.44	0.18	0.41
PvO ₂				0.17	0.34
PvCO ₂					0.92
PaCO ₂					