Comparative value of peripheral and central venous pCO_2 in predicting normal $paCO_2$ during anaesthesia

A. d'Hollander MD PH D, V. Capouet MD,
R. Czerucki MD, J.P. Bomblet MD,
M.J.M. Govaerts MD

The ability of venous pCO_2 to predict arterial pCO_2 within the normal range was tested by measuring pCO_2 in blood sampled simultaneously from a large forearm vein (PER), from the superior vena cava (SVC), and from an artery in 35 anaesthetized patients.

The relationship between arterial and both venous pCO_2's were studied in a first series of 15 patients (ASA physical status class I–II) anaesthetized with methohexitone, fentanyl, pancuronium and nitrous oxide/oxygen, and in a second series of 20 patients scheduled for cardiac surgery anaesthetized with flunitrazepam, fentanyl, pancuronium and nitrous oxide. A marked correlation was found between arterial and both venous pCO_2's samples in the normal patients (a/PER: $r = 0.922$; a/SVC: $r = 0.940$); in the patients with abnormal cardiovascular status the correlation observed was less pronounced (a/PER: $r = 0.501$; a/SVC: $r = 0.507$). In view of the similar correlation coefficients observed from the PER or SVC blood sampling sites, we conclude that the degree of accuracy of the prediction of $paCO_2$ from the venous pCO_2's is not modified by the origin of the venous blood. The differences between the coefficients of correlation found in the normal patients and in those with abnormal cardiovascular function indicate that venous pCO_2 as estimate of $paCO_2$ appears useful only in subjects with normal haemodynamic status.

Key words

ANAESTHESIA; general; CARBON DIOXIDE TENSION; venous, arterial.

From the Free University of Brussels, Department of Anaesthesia of Brugmann Hospital, Brussels, Belgium.

Address correspondence to: Dr. A. d'Hollander, Dept. of Anaesthesia, place Van Gehuchten, 4, Brugmann Hospital, B-1020, Brussels, Belgium.

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During general anaesthesia, monitoring of normocapnic ventilation may be performed either directly, by measuring the $paCO_2$ or indirectly, by measuring the exhaled CO_2.[1-3] Routine placement of an arterial catheter or multiple arterial punctures carries some risk of arterial damage.[4,5] The widespread use of the non-invasive, indirect method is limited by the cost of CO_2 analysers. A simple alternative is the measurement of peripheral venous pCO_2 from the back of the hand, as a predictor of the arterial pCO_2 in anaesthetized healthy patients,[6,7] especially if cutaneous vasodilation is induced by halogenated agents.[8,9]

This study examined the degree of correlation of venous and arterial pCO_2's in the absence of administration of halogenated agents from blood sampled simultaneously from a forearm vein, from the superior vena cava and from an artery, in anaesthetized normal subjects. The validity of such correlations was also studied in patients scheduled for cardiac valvular and/or coronary artery surgery in order to determine the effects of abnormal cardiac function on veno-arterial pCO_2 differences. In the latter patients, the stability of the veno-arterial pCO_2 difference was also assessed for a twenty-minute period.

Methods

This study was approved by the Free University of Brussels Ethics Committee. Thirty-five adult patients were studied with their informed consent.

Group I included 15 patients (ASA physical status class I–II) without cardio-respiratory disease, scheduled for elective surgery. Group II included 20 patients undergoing surgery for valvular and/or coronary artery disease. The patients requiring valvular replacement (n = 8) had preoperative

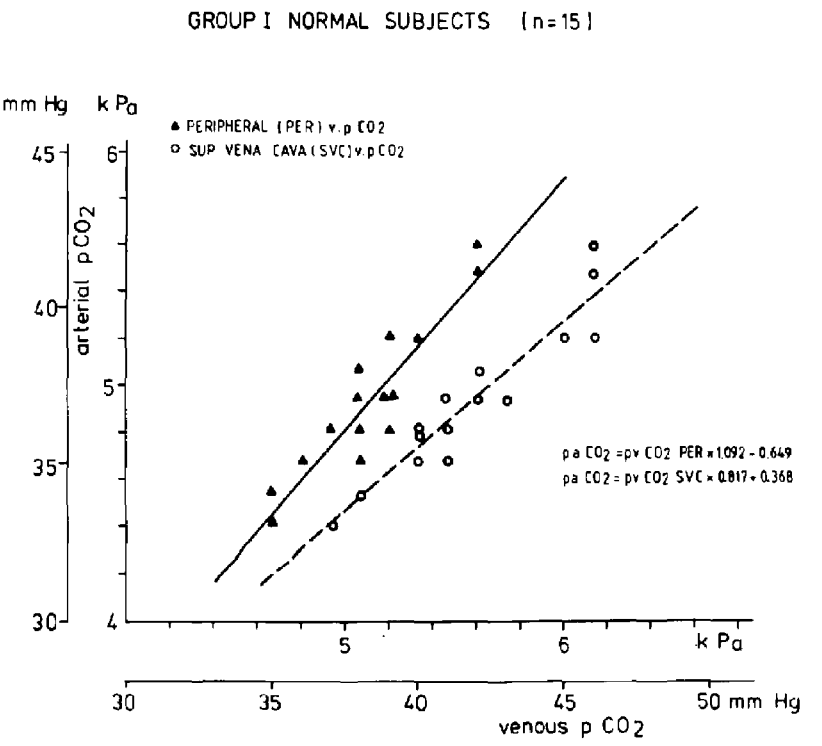

FIGURE 1 Plot of peripheral venous (▲) and superior vena cava (○) pCO_2's in 15 normal anaesthetized subjects. Regression coefficients: $paCO_2/pvCO_2$ PER: r = 0.922; $paCO_2/pvCO_2$ SVC: r = 0.940.

cardiac indices ranging from 1.5 to 2.17 l·min^{-1}·m^{-2} body surface area.

The patients received a standard oral premedication of diazepam 10 mg about 45 minutes before arrival to the OR. In group I patients, anaesthesia was induced with methohexitone (1 mg·kg^{-1}), pancuronium (0.08 mg·kg^{-1}), fentanyl (5 μg·kg^{-1} + supplementary doses of 0.75 μg·kg^{-1}), droperidol (0.33 mg·kg^{-1} + supplementary doses of 0.02 mg·kg^{-1}) and N20/02 2:1 v/v. In group II patients, anaesthesia was induced with flunitrazepam (0.03 mg·kg^{-1}), pancuronium (0.1 mg·kg^{-1}), fentanyl (initial dose 10 μg·kg^{-1} + supplementary doses of 0.75 μg·kg^{-1}), droperidol (as required, at doses of 0.02 mg·kg^{-1}), N_2O/02 1:1 v/v. All patients were ventilated at a frequency of 14 breaths/minute with a constant volume ventilator (Engström 300), with volume settings in accordance with Radford's nomogram.[10]

Fifteen minutes after orotracheal intubation and initiation of controlled ventilation, 2 ml samples of arterial blood, peripheral venous blood (PER) and blood from the superior vena cava (SVC) were taken simultaneously using heparinized syringes after discarding the first 20 ml of blood.

Venous blood samples were taken from an intrathoracic catheter (Intramedicut 16 G – total length: 30 cm; placed percutaneously into the right internal jugular vein by an antero-lateral approach – skin puncture 3 cm above the clavicle and 17.5 cm inserted) and from a peripheral venous catheter (Abbocath 18 G) in a large antecubital vein. The arm with the catheter was kept at the level of the patient's body. The arterial blood was obtained by direct puncture of the femoral artery (Group I patients – 23 G needle) or by percutaneous catheterization of the radial artery (Group II patients – Medicut 18 G).

The samples were analysed immediately in a

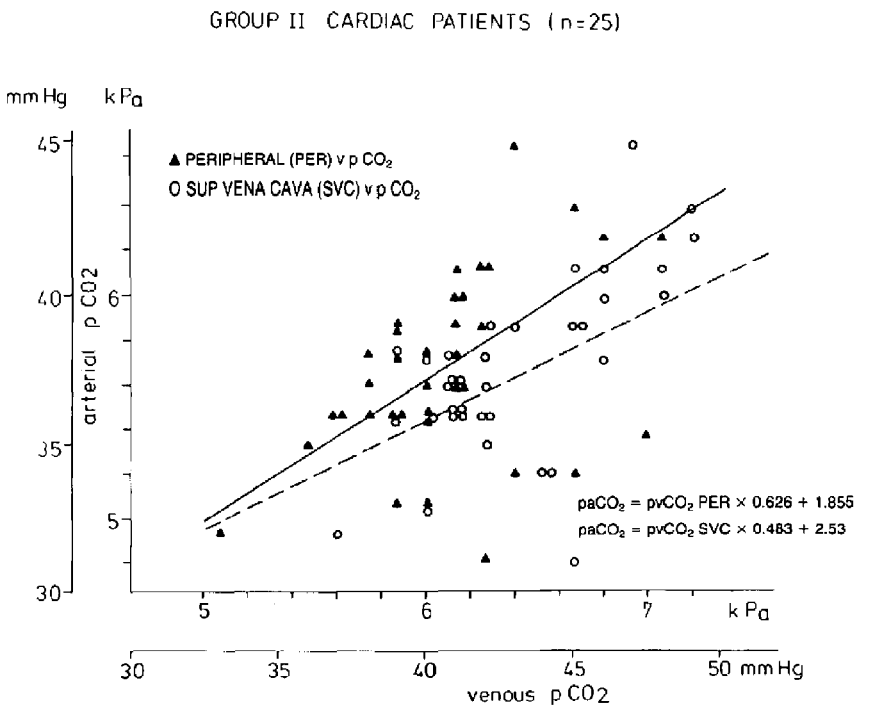

FIGURE 2 Plot of peripheral venous (▲) and superior vena cava (O) pCO_2's in 25 cardiac anaesthetized subjects. Regression coefficients: $paCO_2/pvCO_2$ PER: r = 0.501; $paCO_2/pvCO_2$: r = 0.507.

random order with an automatic blood gas analyser (Corning 175) calibrated before each group of measurements. Reproducibility of the analysis (sample repeated 15 times) was approximately 2.5 per cent.

The effect of the time of each analysis on successive determinations was assessed. Three successive determinations on the same sample showed a mean difference of 0.1 kPa (15 different samples analysed 3 times).

Each measurement was corrected for temperature (rectal temperature – YSI telethermometer). Statistical analysis of the data collected was performed using linear regression and the paired two-tailed Willcoxon signed rank test.

Results

The pCO_2 results are summarized in the Table. The results of the linear regressions performed between the values of $paCO_2$ and $pvCO_2$ PER, and the $paCO_2$ and the $pvCO_2$ SVC were:

Group I;

$$paCO_2 = pvCO_2\ PER \times 1.092 - 0.649, \quad r = 0.922 \quad p < 0.001$$

$$paCO_2 = pvCO_2\ SVC \times 0.187 + 0.368, \quad r = 0.940 \quad p < 0.001$$

Group II

$$paCO_2 = pvCO_2\ PER \times 0.626 + 1.855, \quad r = 0.501 \quad p < 0.001$$

$$paCO_2 = pvCO_2\ SVC \times 0.483 + 2.53, \quad r = 0.507 \quad p < 0.001$$

In group II patients the pCO_2 results 15 minutes after induction of anaesthesia (period I) and 20 minutes later (period II), were not significantly different (Willcoxon signed rank test): pCO_2 (per-a): 0.34 ± 0.10 kPa (period I) vs 0.39 ± 0.15 kPa (period II) and pCO_2 (SVC-a): 0.74 ± 0.10 kPa (period I) vs 0.82 ± 0.1 kPa (period II).

TABLE Values of simultaneous determinations of $paCO_2$, $pvCO_2$ PER and $pvCO_2$ SVC in anaesthetized normal patients (Group I) and cardiac patients (Group II) mean ± SEM

	$paCO_2$ kPa	$pvCO_2$ PER kPa	$pvCO_2$ SVC kPa	pCO_2 (PER-a) kPa	pCO_2 (SVC-a) kPa
Group I	4.93 ± 0.08	5.11 ±0.07	5.58 ±0.09	0.18 ±0.03	0.65 0.03
n = 15	Range 4.39–5.59	4.66–5.59	4.93–6.13	0.0–0.4	0.53–0.93
Group II	5.63 ±0.07	6.03 ±0.06	6.41 ±0.08	0.40 ±0.07	0.78 ±0.07
n = 35	Range 4.66–6.66	5.06–6.80	5.59–7.60	−0.27–1.47	0.13–2.53

$paCO_2$: arterial pCO_2; $pvCO_2$ PER: subcutaneous venous pCO_2; $pvCO_2$, SVC: superior vena cava pCO_2.

Discussion

The data show that, in healthy anaesthetized patients, the pCO_2 of venous blood samples taken peripherally or from the superior vena cava can be used to estimate the $paCO_2$, with an average error about 0.27 kPa.

We noted very similar values of the correlation coefficients obtained for the regressions calculated between $pvCO_2$ and $paCO_2$, in central and peripheral blood (0.92 vs 0.94). Thus the choice between the two venous sampling sites depends on the ability to sample through the venous lines in place.

The correlations found, in Group II, although statistically significant, are much less pronounced than those in Group I. This means that prediction of the $paCO_2$ from the pvCO's is much less satisfactory than in Group I. In Group II patients the error of individual predictions for 95 per cent of the $paCO_2$ values estimated from the $pvCO_2$ is, at best, 0.80 kPa; about three times the error noted for the Group I patients.

The lesser degree of correlation seen in the Group II patients is probably due to marked interindividual alteration of cardiac function among these subjects. The role played by cardiac output in determining the value of arterio-venous CO_2 and O_2 contents is well known.[12,13] Schematically the relationships between tissue CO_2 production and tissue blood flow are:

For a given tissue t:

$$V_tCO_2 = Q_t \times (C_{CO_2}V_t - C_{CO_2}a) \text{ cf ref 13}$$

For the whole body:

$$V_{tot}CO_2 = Q_{c'} \times (Cv_{CO_2}V - C'c_{CO_2}) \text{ cf ref 13}$$

For a given CO_2 production, the difference between venous and arterial CO_2 contents is directly related to the tissue blood flow (Q_t) or, for the whole body, to the pulmonary capillary blood flow (Qc') – approximately the cardiac output – in normal patients.

Within the narrow range of variation of the pCO_2 observed here – about 1 kPa – one may consider that CO_2 blood content is quasilinearly related to its pCO_2.[13] Skin, subcutaneous fat, muscle and bone have low metabolic rates in anaesthetized subjects. Thus the CO_2 content of peripheral venous blood leaving these types of tissues approximates that of arterial blood, if peripheral blood flow is maintained. Such a narrow veno-arterial CO_2 gradient is not expected from more centrally sampled venous blood which, for SVC, also contains the venous flow from the brain and thyroid i.e., tissues with higher metabolic rates than limb tissues.

This interpretation conforms with the results observed in both groups of patients: $paCO_2 < pvCO_2$ PER $< pvCO_2$SVC.

The present results also confirm, as can be predicted from equations 1 or 2, that the difference between the venous and arterial CO_2 contents – or between veno-arterial CO_2 partial pressure – is increased in patients with reduced cardiac function, such as the patients in Group II.

In conclusion, the pCO_2 in venous samples can be used in anaesthetized normal patients to predict $paCO_2$ when $paCO_2$ or end tidal CO_2 measurement are not obtainable. In view of our observations, the use of halogenated agents such as enflurane, isoflurane or halothane to produce peripheral vasodilatation, or manoeuvres to "arterialize" the capillary blood[14] are not required in anaesthetized

normal patients. Although severe hypo- and hypercapnic conditions induce marked changes in tissue metabolism and on cardio-circulatory dynamics, the reported correlations between venous pCO_2 and $paCO_2$ were excellent – above 0.90.[7] Nevertheless, even if high degrees of veno-arterial pCO_2 correlations are generally observed, the regression equations may be influenced by environmental factors, such as the type of anaesthesia, nature and dose of the drugs used, temperature of the patient, location of venous sampling, etc. Consequently, a single equation system is not universal for all clinical situations, or, inversely, the best fit of the measurements performed with a given experimental design will be obtained with a regression equation specific to the conditions present.

References

1 *Nunn JF, Hill W.* Respiratory dead space and arterial to end-tidal CO_2 tension difference in anesthetized man. J Appl Physiol 1960; 15: 383–9.

2 *Tulou P, Walsh PM.* Measurement of alveolar carbon dioxide tension at maximal expiration as an estimate of arterial carbon dioxide tension in patients with airway obstruction. Am J Respir Dis 1970; 102: 921–6.

3 *Franckel DZN, Sandham G, Rebuck AS.* A new method for measuring pCO_2 during anaesthesia. Br J Anaesth 1979; 51: 215–20.

4 *Gardner RM, Schwartz R, Wong HC, Burcke JP.* Percutaneous indwelling catheters for monitoring cardiovascular function. N Engl J Med 1974; 290: 1229–32.

5 *Kim JM, Arakawa K, Briss J.* Arterial cannulation: factors in the development of occlusion. Anesth Analg 1975; 54: 856–41.

6 *France CJ, Eger EI, Bendixen HH.* The use of peripheral venous blood for pH and carbon dioxide tension determinations during general anesthesia. Anesthesiology 1974; 40: 311–16.

7 *Williamson DC III, Munson ES.* Correlation of peripheral venous and arterial blood gas values during general anesthesia. Anesthesiology 1982; 61: 950–2.

8 *Stevens WC, Cromwell TH, Halsey MJ, Eger EI II, Shakespeare TF, Bahlman SH.* The cardiovascular effects of a new inhalation anesthetic, Forane, in human volunteers at constant arterial carbon dioxide tension. Anesthesiology 1971; 35: 8–16.

9 *Eger EI II, Smith NT, Cullen DJ, Cullen BF, Gregory GA.* Comparison of the cardiovascular effects of halothane, fluroxene, ether and cyclopropane in man: a résumé. Anesthesiology 1971; 34: 25–41.

10 *Radford EP.* Ventilation standards for use in artificial respiration. J Appl Physiol 1955; 7: 451–60.

11 *Kelmann GR, Nunn JF.* Nomograms for correction of blood pO_2, pCO_2, pH and base excess for time and temperature. J Appl Physiol 1966; 21: 1484–90.

12 *Powles AC, Campbell EJ.* How to be less invasive. Am J Med 1979; 67: 98–104.

13 *Comroe JH, Foster RE, Dubois AB, Briscoe WA, Carlsen E.* The lung – clinical and pulmonary function tests, 2nd ed., pp. 154 and 342, Chicago, Year-Book Medical Publishers, 1962.

14 *Gambino SR.* Collection of capillary blood for simultaneous determinations of arterial pH, CO_2 content, pCO_2, and oxygen saturation. Am J Clin Pathol 1961; 35: 175–83.

Résumé

Les corrélations entre pCO_2 artérielle ($paCO_2$) et pCO_2 veineuses – d'origine périphérique ($pvCO_2$ PER) ou veine cave supérieure ($pvCO_2$ SVC) – prélevées simultanément furent testées auprès de 35 patients anesthésiés sans agent halogéné et normoventilés. Ces mesures effectuées auprès de 15 patients (ASA Class I–II) anesthésiés ont apporté les résultats suivants: $paCO_2 = pvCO_2$ PER $\times$ 1.092 − 0.649, $r = 0.922$; $paCO_2 = pvCO_2$ SVC $\times$ 0.717 + 0.368, $r = 0.940$. Les mêmes mesures déterminées auprès d'un groupe de 25 patients devant subir une intervention sous circulation extracorporelle et comprenant des patients avec une fonction cardiovasculaire anormale ont donné les résultats suivants: $paCO_2 = pvCO_2$ PER $\times$ 0.626 + 1.855, $r = 0.561$; $paCO_2 = pvCO_2$ SVC $\times$ 0.483 + 0.530, $r = 0.507$. L'ensemble des résultats montre que la prédiction de la $paCO_2$ ne peut être faite qu'avec une précision acceptable – ± 0.27 kPa – qu'auprès de groupes de patients normaux. Dans ce cas, la faible différence enregistrée entre les coefficients de corrélation trouvés pour du sang d'origine soit PER soit SVC indiquent que ces deux localisations de prélèvement veineux sont également exploitables pour la prédiction de la $paCO_2$ de patients anesthésiés et normoventilés.