

Coronary sinus venoarterial CO₂ difference in different hemodynamic states

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Summary : Myocardial metabolic rate and coronary flow are closely related limiting thus the diagnostic value of coronary sinus saturation monitoring as an indicator of flow. Regional venoarterial CO₂ gradient was found elevated during low flow in various clinical and experimental conditions, in animals and humans. This study was undertaken to examine the impact of the variations of cardiac mechanical work on veno-arterial CO₂ content and partial pressure difference (ΔPCO_2) of the coronary sinus blood.

Twenty-seven patients of either sex (m/f = 21/6), undergoing coronary artery bypass grafting under extracorporeal circulation, were studied. Monitoring included a Swan-Ganz catheter and a coronary sinus line. The correct position of the line was verified by the waveform displayed in the monitor. Immediately after cannulae placement, a hemodynamic profile was obtained and simultaneous arterial and coronary sinus sampling for blood gas analysis was done in an ABL 720 (Radiometer Copenhagen) analyzer. A second collection of the same data was obtained five minutes later with the patients in a slight "head-down" position. Conditions for exclusion was intersample variation of hemoglobin's concentration greater than 15% and sodium ion concentration difference greater than 10% of the greater value. Arteriovenous oxygen partial pressure difference ($\Delta\text{P(a-cs)O}_2$), veno-arterial carbon dioxide partial pressure difference ($\Delta\text{P(cs-a)CO}_2$), O₂ & CO₂ content difference and heart's respiratory quotient were calculated and correlated to cardiac output (CO) and the other hemodynamic parameters. Statistical analysis employed t-paired test and linear regression.

No ischemia was detected during sampling. "Head-down" position had a significant impact to all hemodynamic parameters except heart rate. In both data rows, although CO ranged widely and altered significantly, coronary sinus oxygen saturation and arteriovenous O₂ content difference were stable and showed insignificant correlations to all the hemodynamic parameters that were studied. Carbon dioxide content difference (coronary sinus-arterial) showed a trending of decrease with higher flow. $\Delta\text{P(cs-a)CO}_2$ appeared stable and independent of flow. Finally, respiratory quotient decreased significantly from 0.91 ± 0.4 to 0.86 ± 0.4 (mean \pm SD ; $p < 0.05$).

The heart's high basal oxygen consumption and the almost near hemoglobin's desaturation transcoronary extraction of oxygen limits the value of coronary sinus saturation monitoring as indicator of coronary flow. Heart's little extraction reserve is faced with coronary flow reserve. In the physiologic range and under the conditions of anesthesia, elevated CO₂ production is accompanied with increased coronary flow. Under these circumstances, $\Delta\text{P(cs-a)CO}_2$ appears stable and is not suitable for clinical decisions concerning heart's coronary flow.

Key words : Coronary sinus ; Venarterial CO₂ gradient ; Cardiac surgery.

INTRODUCTION

Local venous oxygen saturation monitoring provides information about the metabolic state of various organs, as increased demands or decreased local flow are early reflected on their decreased venous saturation due to the mobilization of oxygen from the extraction reserve. Concerning the heart, the high basal oxygen consumption and the almost near the limits of hemoglobin's dissociation transcoronary extraction of oxygen are characteristics of its metabolism. The cardiac muscle shows a decreased ability to compensate for increased oxygen consumption by increasing extraction. This little "extraction reserve" is faced *via* increasing coronary flow to meet increased metabolic demands (coronary "flow reserve"). Resting coronary flow is

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quite different from the flow in the coronaries during exercise, although the saturation of oxygen in the venous blood drained from the coronary sinus is almost unchanged in the above conditions. This tight correlation of heart's metabolic rate to coronary flow is a factor limiting the diagnostic value of coronary venous saturation monitoring.

In the past decade, the regional absolute value of carbon dioxide's partial pressure and the regional veno-arterial difference ($P(v-a)CO_2$ or ΔPCO_2) were evaluated as bio-markers for early prediction of ischemia in various organs. In moderate states of ischemic dysoxia, increased production together with stagnation due to reduced flow increased CO_2 concentration in the local venous blood while $P(v-a)CO_2$ was also elevated. Regional veno-arterial carbon dioxide partial pressure difference ($P(v-a)CO_2$) was increased during the reduction of flow in an isolated innervated canine hindlimb controlled model (1). In other animal models of low-flow state with constant oxygen consumption and higher than critical oxygen delivery, $P(v-a)CO_2$ was also elevated (2). Finally, the clinical application of gastrointestinal tonometry in the management of the heavy ill patients is based on the same principles and the predictive value of these indices.

Presently, data concerning the myocardial veno-arterial CO_2 content or partial pressure difference during alterations of coronary flow or variations of cardiac mechanical work are unavailable. The aim of this study is to assess the impact of the variations of cardiac performance on CO_2 content and $P(v-a)CO_2$, by hypothesizing that increased work leads to increased $P(v-a)CO_2$ in the coronary sinus blood.

MATERIALS – METHODS

Patient population

After informed consent, 27 patients of either sex (m/f = 21/6), 65.3 ± 7.8 (mean \pm SD) yrs of age, 76.8 ± 12.8 Kg of weight and 165.2 ± 9.9 cm of height, who were undergoing elective coronary artery bypass grafting (CABG) under extracorporeal circulation (CPB), were studied. Patients with significantly compromised left ventricular function in the preoperative evaluation were not included. Ejection fraction (EF) was greater than 45% in all patients in the preoperative angioventriculography. Premedication consisted of diazepam 10 mg, orally given one hour before admission in the operating room. After arrival, the patients were

connected to the monitor (*Solar 8000, Marquette Medical Systems, Milwaukee, USA*) and a bispectral index sensor (*BIS/XP, Aspect Medical systems, USA*) was placed. Intraoperative electrocardiography consisted of continuous two-lead display (depending on the underlying pathology) chosen from seven in full monitor's view (I, II, III, avR, avL, avF, V_5), plus continuous 5-leads ST-segment analysis (II, III, avL, avF, V_5) with ST-trending displayed individually for every lead and in sum. After peripheral venous cannulation, a radial or brachial artery was cannulated under local anesthesia for measurement of arterial pressure and blood sampling. The anesthetic induction was achieved with the slow, single dose administration of midazolam (1-3 mg) plus fentanyl (100-250 μ g) plus etomidate (0.2 mg/kg) and neuromuscular blockade with pancuronium (0.12 mg/kg). After tracheal intubation, all patients were mechanically ventilated (*Julian, Draeger, Germany*) towards slight hypocapnia ($33 \text{ mmHg} < paCO_2 < 37 \text{ mmHg}$), based on repeated arterial blood sampling. For the maintenance of the hypnotic component of anesthesia, they received desflurane targeting to an end-tidal concentration of 4-5% and a BIS value less than 50. A continuous infusion of remifentanyl ($\approx 20 \mu\text{g/kg/h}$), facilitated by a pump (*Fresenius Vial, Brezins, France*), was also administered as a means of providing analgesia. After induction and *via* right internal jugular vein, the insertion of a triple-lumen central venous catheter was followed by the placement of a pulmonary artery catheter (*Oximetry TD catheter, Edwards Lifesciences, Germany*).

During the operative procedure and immediately after the placement of the aortic and venous cannulae and the pulmonary artery vent, a coronary sinus cardioplegia line was instituted. This line offers a side port for pressure measurement, which was connected to an independent transducer. Optionally, aortic root cannulation for administration of cardioplegic solution was also done, based on the preference of the surgical team. The correct placement of the coronary sinus line was verified by the waveform displayed in the monitor, consisting from an easy identifiable atrial contraction immediately followed by a ventricular contraction and from a diastolic period.

Samples collection

All samples were drawn before the initiation of CPB. A short period for hemodynamic stabilization was permitted after the completion of coronary sinus line placement. Immediately after, a

hemodynamic profile was obtained with the thermodilution method. Measurements were performed in triplicate, using cold water. Pulmonary artery occlusion pressure (PAOP) and central venous pressure (CVP) were measured with the patient shortly disconnected from the ventilator. Next, ten milliliters of blood were drawn for "washing" the flushing solution from the arterial and the coronary sinus lines and commercially prepared syringes (Drihep™ Bencton Dickinson Vacutainer Systems U.K.) were attached to the ports for simultaneous sampling (art1 & cs1). Immediately after and if pulmonary artery occlusion pressure was less than 16 mmHg, the operating table was tilted in a slight "head-down" position (10 degree Trendelenburg manoeuvre). This manoeuvre took place under close monitoring (continuous ST-segment analysis, v-wave recognition), direct vision of the heart and, as already described, after a recent estimation of cardiac function with the thermodilution method and with the cannulae of the circuit already introduced. The later could allow immediate unloading to the venous reservoir or even initiation of CPB. Five minutes later and under stable conditions, the same sequence was held (hemodynamic profile and blood sampling; samples: art2 & cs2). Samples were analyzed in an ABL 720 (Radiometer Copenhagen) analyzer, accepting that $P_{50(st)}$ equals 26.85 mmHg and respiratory quotient (RQ) is 0.86, in 37°C, after identifying arterial or venous blood. All samples were drawn before any intraoperative blood transfusion.

Data conversion and statistical analysis

Results were entered, checked, processed, analyzed and stored in Excel. If the absolute difference of the hemoglobin's concentration in the arterial sample minus coronary sinus sample was less than 15% of the greater value, the patient was excluded. The patient was also excluded when the absolute sodium ion concentration difference of the two simultaneously drawn samples was greater than 10% of the greater value. These two conditions were held to diminish the error from flush-line dilution of the simultaneously drawn samples. Venous-arterial CO₂ partial pressure gradient was calculated as: $\Delta P(cs-a)CO_2 = P_{cs}CO_2 - P_{art}CO_2$ (cs & art denote arterial and coronary sinus respectively). Plasma total concentration of CO₂ ($ctCO_2(P)$) was calculated by the analyzer with the equation I (see appendix). Whole blood concentration of CO₂ ($ctCO_2(B)$) was calculated by the analyzer with the equations II (see appendix). Whole blood concen-

tration of CO₂ ($ctCO_2(B)$) was considered equal to carbon dioxide content ($ctCO_2$). The heart's respiratory quotient (RQ) was calculated as: $(cs\ ctCO_2 - art\ ctCO_2)/(art\ ctO_2 - cs\ ctO_2)$. Finally, patients presenting calculated RQ > 2 were also excluded. Data were expressed as *mean* ± *SD*. To test our primary hypothesis, three outcome variables were defined: mean systemic arterial pressure, pulmonary artery occlusion pressure and $\Delta P(cs-a)CO_2$, the first two for the differentiation of the hemodynamic state and the third expressing the experimental question. Correlation was analyzed with linear regression. Comparison was made with the t-paired test. A value of $p < 0.05$ was considered statistically significant.

RESULTS

Data obtained during the transition between the two hemodynamic states are presented in Table 1. As it is shown there, the only primary parameter that remained stable after tilting the table was practically the heart rate. In the patients of the study ($n = 27$), the hemoglobin's concentration (g/dL) of the arterial samples was 11.76 ± 2.1 and 11.70 ± 1.8 (sampling 1 & 2 resp; ns; see Table 2). Hemoglobin's concentration of the coronary sinus samples was 12.21 ± 1.9 and 12.12 ± 1.8 respectively, also not differing statistically. Mean temperature was $36.60 \pm 0.189^\circ C$. During sampling,

Table 1
Hemodynamic data during sampling in the 27 patients of the study

mean	Sampling period 1		Sampling period 2		p value
	SD		mean	SD	
HR	78.7	± 17.9	77.8	± 14.5	ns
sAP	106	± 12.3	123	± 14.6	< 0.001
dAP	65.1	± 10.8	70.0	± 9.0	< 0.001
mAP	77.8	± 10.6	87.9	± 8.6	< 0.001
sPAP	25.3	± 3.6	34.0	± 6.0	< 0.001
dPAP	13.0	± 2.6	15.8	± 2.4	< 0.001
mPAP	18.0	± 3.3	22.0	± 3.0	< 0.001
CO	4.4	± 1.2	4.6	± 1.1	< 0.01
SV	57.9	± 16.6	60.1	± 14.0	< 0.05
SVR	1309	± 359	1369	± 310	< 0.05
PVR	127	± 57.3	139	± 49.2	ns
PAOP	11.9	± 2.3	14.2	± 2.3	< 0.001
CVP	9.6	± 1.9	12.6	± 1.8	< 0.001

HR for heart rate in *beats/min*; AP for arterial pressure; PAP for pulmonary artery pressure, (pressures in *mmHg*); s, d, m for systolic, diastolic, and mean respectively; CO for cardiac output in *L/min*; SV for stroke volume in *cm³*; SVR and PVR for systemic and pulmonary vascular resistance; PAOP for pulmonary artery occlusion pressure; CVP for central venous pressure.

Table 2
Results from simultaneous arterial and coronary sinus sampling (n = 27)

	Sampling period 1		Sampling period 2		p values			
	art mean \pm SD	cs mean \pm SD	art mean \pm SD	cs mean \pm SD	art(1/2)	cs(1/2)	art1/cs1	art2/cs2
Hb	11.76 \pm 2.1	12.21 \pm 1.9	11.70 \pm 1.8	12.12 \pm 1.8	ns	ns	< 0.05	< 0.05
PCO ₂	33.87 \pm 3.3	43.70 \pm 4.3	33.72 \pm 3.1	43.73 \pm 4.2	ns	ns	< 0.001	< 0.001
ctCO ₂ (P)	55.53 \pm 4.0	63.79 \pm 4.5	55.47 \pm 4.0	63.74 \pm 4.4	ns	ns	< 0.001	< 0.001
ctCO ₂ (B)	47.43 \pm 3.9	55.49 \pm 4.3	47.78 \pm 4.2	55.52 \pm 4.4	< 0.05	ns	< 0.001	< 0.001
PO ₂	344 \pm 100	24.87 \pm 3.1	341 \pm 102	24.05 \pm 2.5	ns	< 0.05	< 0.001	< 0.001
SO ₂	99.73 \pm 0.5	47.70 \pm 8.6	99.80 \pm 0.4	47.46 \pm 6.9	ns	ns	< 0.001	< 0.001
ctO ₂	17.40 \pm 2.6	8.19 \pm 2.2	17.26 \pm 2.2	8.03 \pm 1.8	ns	ns	< 0.001	< 0.001
ct(a-cs)O ₂	9.21 \pm 1.9		9.23 \pm 1.6		ns			
ct(cs-a)CO ₂	8.06 \pm 2.7		7.74 \pm 2.8		ns			
RQcs	0.91 \pm 0.4		0.86 \pm 0.4		< 0.05			
Δ P(cs-a)CO ₂	9.83 \pm 2.8		10.0 \pm 3.0		ns			

Samples analyzed in 37° C, partial pressures in mmHg,

ct denotes content in mL/dL, cs-a :veno-arterial difference, a-cs :arterial-venous difference, S for O₂ saturation in %, RQcs denotes respiratory quotient, calculated as : $ct(cs-a)CO_2 / ct(a-cs)O_2$

patients were free of myocardial ischemia, monitored by all available electrocardiographic leads, plus morphology of v-wave in the pulmonary artery occlusion pressure waveform. Values of metabolic status and of calculated parameters are also shown in Table 2. In general, differences of statistical importance were found only between arterial and coronary sinus samples, while values generated from blood of the same origin (arterial from sampling 1 & 2 or coronary sinus from sampling 1 & 2) were differing unimportantly. Table 2 also shows the mean values \pm SD of the calculated content differences (arterial minus coronary sinus for oxygen and coronary sinus minus arterial for carbon dioxide) and Δ PCO₂ from samples 1 and 2, which were not differing statistically. As it is shown there, the only parameter that was altered between state 1 and 2 was the respiratory quotient.

In both sampling periods 1 and 2, linear regression analysis of oxygen and carbon dioxide content differences and Δ PCO₂ to cardiac output, mean arterial pressure and systemic vascular resistance showed poor correlations with wide scattering, low correlation coefficients and statistical unimportance. Furthermore, the differences of content difference (for oxygen and carbon dioxide) and the difference of Δ PCO₂ were also poorly correlated to the differences of the above hemodynamic parameters, measured during sampling period 1 and 2. Details about the results of the analysis are shown in the Figures.

DISCUSSION

Applying Fick's principal in a steady state, carbon dioxide production equals the product of flow by the venoarterial CO₂ content difference. Over the usual physiologic range of carbon dioxide contents, the relationship between PCO₂ and blood CO₂ content (ctCO₂(B)) is linear and, by substituting, the equation (1) can be obtained :

$$\Delta PCO_2 = k(VCO_2/Q)(1)$$

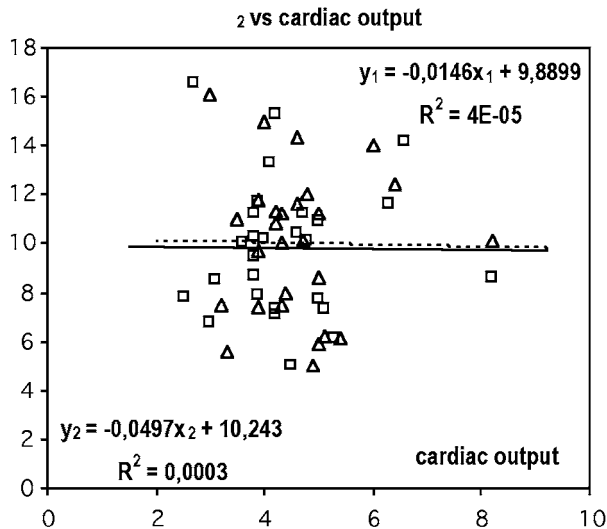
where :

ΔPCO_2 is the gradient of carbon dioxide's partial pressures or the difference between PCO₂ in the venous and arterial blood,

k is assumed to be a constant

Q is the blood flow

Therefore, Δ PCO₂ would be linearly related to CO₂ production and inversely related to flow, in separate organs or in the level of total CO₂ production (when flow equals cardiac output). From these considerations, two different models of clinical use of Δ PCO₂ have been proposed (3) : either Δ PCO₂ used as a marker of flow in relatively stable patients, or used as a marker of tissue hypoxia in severely ill patients. In septic animals with low Q but O₂ supply-independence, Δ PCO₂ was found significantly higher when systematic flow was decreased (4). In critically ill but hemodynamically stable patients, Δ PCO₂ was higher in those with the lower cardiac output (5). Pharmaceutically



y axis : ΔPCO_2 ($\text{PcsCO}_2 - \text{PaCO}_2$) in mmHg, x axis : cardiac output in L/min

rectangles, equation 1, filled line : sampling 1
triangles equation 2, dotted line : sampling 2

Fig. 1. — Heart's $\Delta\text{P}(\text{cs-a})\text{CO}_2$ and cardiac output.

In both sampling periods, the correlation was statistically insignificant. Transition to the second hemodynamic state, although accompanied by an increased in CO, had no effect in ΔPCO_2 . Between the two states, the difference of ΔPCO_2 ($\Delta\{\Delta\text{P}(\text{cs-a})\text{CO}_2\} = \Delta\text{P}(\text{cs-a})\text{CO}_2^{(2)} - \Delta\text{P}(\text{cs-a})\text{CO}_2^{(1)}$) showed an insignificant negative correlation to the increased CO ($\Delta\{\Delta\text{P}(\text{cs-a})\text{CO}_2\}$ to ΔCO : $y = -1.538x + 0.49$; $R^2 = 0.100$; $\Delta\text{CO} = \text{CO}^{(2)} - \text{CO}^{(1)}$; 1 & 2 denoting the two hemodynamic states). In the conditions of our study, increased systemic flow practically, if not lowering, leaves ΔPCO_2 unaffected. Our findings concerning ΔPCO_2 and mAP were similar.

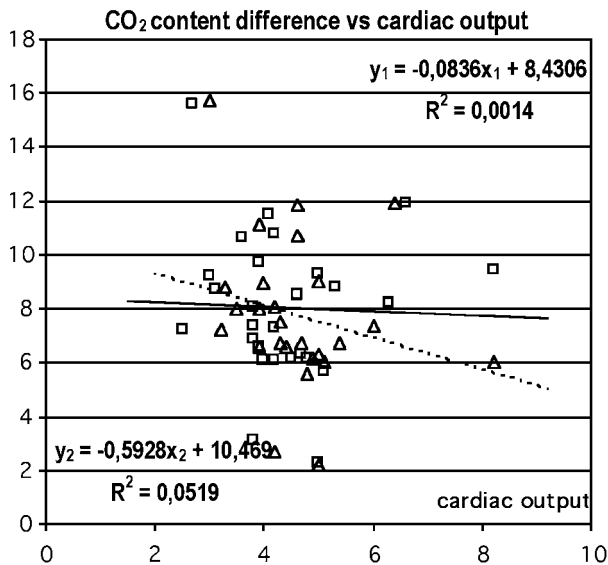
increased systemic flow with dobutamine in cardiac patients was associated with parallel decrease in ΔPCO_2 (6). In conditions of low flow state and tissue hypoxia, striking increases in ΔPCO_2 were reported, as in cardiac arrest-resuscitated animals or patients (7, 8). Venoarterial CO₂ gradient (ΔPCO_2) was reported as greater in patients with circulatory failure than in those without, even with relatively low cardiac output (8). In a model of cardiac tamponade in anesthetized dogs, by the relationship between ΔPCO_2 and Q was concluded that ΔPCO_2 can reliably represent a parameter of tissue hypoxia (9). When abdominal aorta is clamped for surgical repair of aortic aneurysms or occlusive lesions, regional ΔPCO_2 (sampling from the lower inferior vena cava) is related to the extent of the collateral circulation (10).

In our study, we tested whether heart's ΔPCO_2 is altered between different hemodynamic states. The findings of the study do not support our primary hypothesis. The systemic flow in our anesthetized patients showed wide ranges and intersampling significant variations, but correlation to

ΔPCO_2 was absent. Cardiac output together with systemic vascular resistance can be considered as mechanical work, which is produced through metabolic transformations with concurrent oxygen consumption. In our patients, increased oxygen consumption was not related to further desaturation of heart's venous blood, fact leading to the conclusion that heart was consuming oxygen from extra coronary flow, in other words from the coronary "flow reserve". It is easy therefore to conclude that coronary flow in our patients was related to the observed variations of mechanical work. Nevertheless, the hemodynamic parameters that were studied were not significantly correlated to ΔPCO_2 in any case.

Equation (1) provides also some explanation for our observations. Generally, ΔPCO_2 is related in a negative linear fashion to flow when VCO_2 is stable. In conditions or in organs performing differently, this relation is absent. The heart's narrow "extraction reserve" and the almost coupled oxygen consumption/coronary flow means that carbon's dioxide production is also coupled to coronary flow. Concerning our patients and in both conditions (sampling 1 & 2), although in the relations of ΔPCO_2 to the hemodynamic parameters that were studied linearity is negative, practically it is stable and the relation is not accompanied by a short of statistical significance (*see scattering and R² values in Figure 1*). For the mechanical work produced under the conditions of cardiac anesthesia, the heart's VCO_2/Q ratio seems stable.

Data obtained from our patients include $\text{ctCO}_2(\text{B})$, which is the total amount of CO₂ in the blood as measured by modern analyzers (11). Respiratory quotient (RQ) is the ratio of CO₂ production to O₂ consumption. Normally, and with inflow equal to outflow, its value becomes the ratio of CO₂ venoarterial content difference to O₂ arterial-venous difference and it may vary with respect to the predominant energy source. By rearranging equation (1), the ratio VCO_2/Q becomes $(\text{VO}_2 \times \text{RQ})/\text{Q}$ and ΔPCO_2 becomes a function of respiratory quotient multiplied by VO_2/Q . Concerning the heart that is working with a more or less stable ratio of VO_2/Q , minor alterations of ΔPCO_2 can be attributed in some degree to the alterations of RQ. In Figure 3, it is demonstrated that the arteriovenous oxygen content difference is increased with the systemic pressure. As it is reported in Figure 2, the venoarterial carbon dioxide content difference is decreased with the systemic pressure and flow. Furthermore, this relation is augmented in higher flows. From those observations it is concluded that

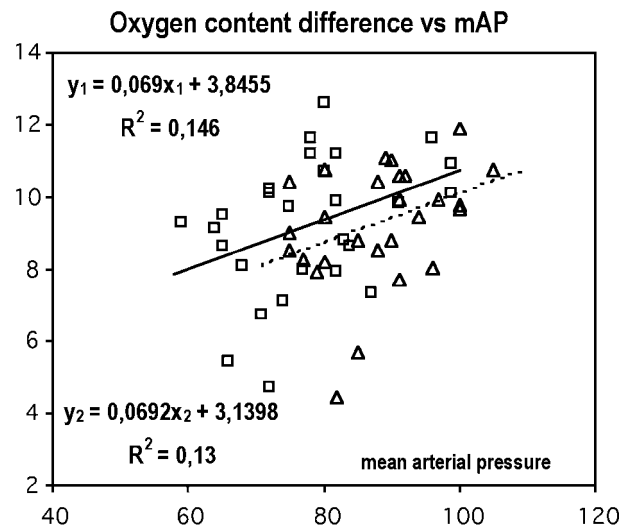


y axis : carbon dioxide content difference ($ct(cs-a)CO_2$)
 rectangles, equation 1, filled line : sampling 1
 triangles equation 2, dotted line : sampling 2
 cardiac output in L/min

Fig. 2. — Heart's venoarterial carbon dioxide content difference and cardiac output.

In both sampling periods, there was a negative, yet statistically insignificant, correlation. In both the hemodynamic states, higher systemic flows were not related to elevated $ct(cs-a)CO_2$. In fact, increasing CO by manoeuvring was followed by a decrease in $ct(cs-a)CO_2$. This decrease was positively related to ΔCO ($y = 0.804x - 0.47$; $R^2 = 0.076$; $\Delta CO = CO^{(2)} - CO^{(1)}$). Results concerning $ct(cs-a)CO_2$ and mAP were similar ($y_1 = -0.012x_1 + 9.0$ and $R^2 = 0.002$; $y_2 = -0.040x_2 + 11.2$ and $R^2 = 0.015$; 1 & 2 denoting the two hemodynamic states).

the metabolic pattern of the heart is changed when the flow or the opposing resistance to flow is increased. The heart is producing less CO_2 for the more O_2 that it consumes. It must be reminded that no patient developed clinically apparent signs of ischemia. As it is reported in Table 2, respiratory quotient was decreased statistically between sampling 1 and 2. It seems that the heart can modulate, between certain limits, the way by which mechanical work is produced, by modifying its metabolic pathways. Resting hearts, working towards an open periphery, exhibit a high, almost 1, respiratory quotient. On the other hand, addition of resistance may attenuate this "easy" and effective metabolism of carbohydrates, towards a decreased respiratory quotient. Increased production of lactic acid because of less clearance of pyruvate by the Krebs cycle and hydrolysis of adenosine triphosphate and diphosphate or decarboxylation of some substrates produced by intermediate metabolism such as cetoglutarate or oxaloacetate may account for the



y axis : oxygen content difference ($ct(a-cs)O_2$)
 rectangles, equation 1, filled line : sampling 1
 triangles equation 2, dotted line : sampling 2
 mAP : mean arterial pressure in mmHg

Fig. 3. — Heart's arteriovenous oxygen content difference and mean arterial pressure.

In both sampling periods, a positive, yet statistically insignificant, correlation was found. In the conditions of our study, a manoeuvre that augments venous return while minor impacts SVR increased mAP without causing extra desaturation of the coronary sinus blood. As it is shown, transition to the second hemodynamic state was accompanied by a right translocation of the (dotted) line. Between the two states, the difference of O_2 content difference was almost stable to the increased mAP ($\Delta ct(a-cs)O_2$ to ΔmAP : $y = 0.0023x - 0.003$; $R^2 = 0.002$; $\Delta ct(a-cs)O_2 = ct(a-cs)O_2^{(2)} - ct(a-cs)O_2^{(1)}$; $\Delta mAP = \text{mean AP}^{(2)} - \text{mean AP}^{(1)}$). Our findings concerning O_2 content difference and systemic flow were similar.

observed modification of respiratory quotient (2). These minor but potential sources of less aerobic CO_2 production may play a role in the independency of ΔPCO_2 from Q, which was observed in our patients. Furthermore, ΔPCO_2 goes as the equation (1) states when CO_2 production is aerobic. In dysoxic conditions, anaerobic CO_2 generation is not unlikely. Two possible sources of CO_2 have been identified : buffering of excess of H^+ ions by bicarbonates and decarboxylation of metabolic intermediates. The excessive production of lactic acid due to an acceleration of anaerobic glycolysis is a suitable example for the heart. These metabolic pathways are also carbon dioxide productive, although heart's flow is lowered. Obviously, the ratio VCO_2/Q and consequently ΔPCO_2 are altered in a different manner than the increase in ΔPCO_2 due to CO_2 stagnation. Finally, in patients with coronary artery disease, intercoronary variations of local flow / demand ratios cannot be considered as absent. These variations may produce normalization of

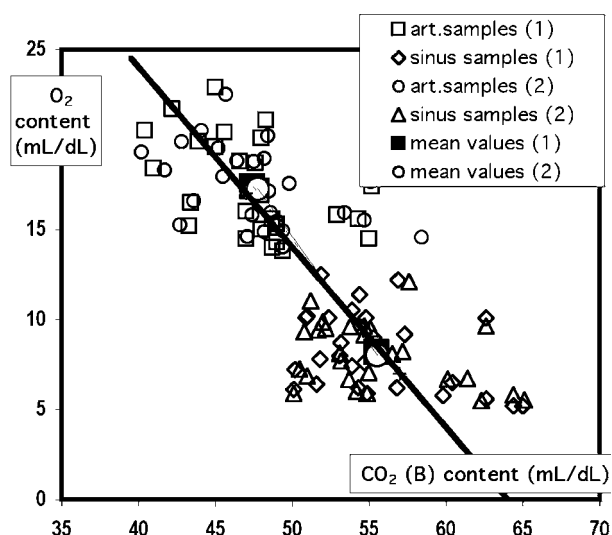


Fig. 4. — Heart's respiratory quotient during the transition of hemodynamic state.

The points displayed are formed from pairs of oxygen and carbon dioxide content, projected in axes of ctO_2 (y-axis) & $ctCO_2$ (x-axis). The arterial blood from each patient is representing a point ($artctO_2$ & $artctCO_2$), the coronary sinus blood another point ($cscctO_2$ & $cscctCO_2$) and the line connecting those two points was considered as an expression of the respiratory quotient. Both sampling periods are represented as well as the mean values. Thick line is an analog for $RQ = 1$; sampling 1 : $RQ = 0.91$ (dotted line); sampling 2 : $RO = 0.86$ (filled line).

findings in the coronary sinus blood in respect to ΔPCO_2 (12).

In conclusion, not only monitoring of coronary sinus oxygen saturation, but monitoring of carbon's dioxide venoarterial tension difference, as well, is not suitable for clinical decisions concerning heart's coronary flow. Factors limiting the diagnostic value of coronary sinus blood gases include the coupling to coronary flow and, as we are reporting, the variations of heart's respiratory quotient. Our study demonstrates that heart's ΔPCO_2 cannot be used as a marker of heart's flow in relatively stable patients. This mode of use should not be really recommended because of complex interrelations between heart's metabolism and coronary flow reserve as well as the ratio of aerobic and less aerobic carbon dioxide production, which may often exist in the working heart. This makes the interpretation of heart's ΔPCO_2 and of its changes particularly misleading in concern to the coronary flow.

APPENDIX

Eq.(I)

$$ctCO_2(P) = 0.23pCO_2 + cHCO_3(P)$$

Eq.(II)

$$ctCO_2(B) = 9.286 \times 10^{-3} \times pCO_2 \times ctHb(1+10^{(pHEry - pKEry)}) + ctCO_2(P)(1-(ctHb/21.0))$$

where : $pHEry = 7.19 + 0.77(pH-7.40) + 0.035(1-SO_2)$

$$pKEry = 6.125 - \log(1+10^{(pHEry - 7.84 - 0.06SO_2)})$$

ct for concentration

c for calculated

p for partial pressure

S for saturation

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