Changes in PCO_2 with acute changes in cardiac index

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Purpose: *A direct relationship between cardiac index (CI) and end-tidal PCO_2 ($PETCO_2$) shortly after decreased CI was reported, but arterial PCO_2 was not measured. Our purpose was to supply the missing information on the immediate effects of alterations in CI on $PaCO_2$, $PETCO_2$ and thus on $Pa–PETCO_2$.*
Methods: *We measured CI, Pa and $PETCO_2$ and calculated the difference in 20 patients scheduled for elective heart surgery just before and immediately after the sternotomy. The measurements were made using standard methods: thermodilution for CI, infra-red and blood gas analysis for PET and $PaCO_2$ respectively. The results were analyzed by linear regression.*
Results: *Very significant, direct and immediate changes in PET and $PaCO_2$ with changes in CI were noted. The ratios were 3.8 and 4.2 mmHg L^{-1} respectively. The calculated values of r were 0.75 ($P < 0.001$) for $PETCO_2$ and 0.64 ($P < 0.005$) for $PaCO_2$. The magnitude of individual change in PCO_2 varied considerably such that the alterations in $Pa–PETCO_2$ were also variable, without any correlation with the direction or magnitude of change in CI.*
Conclusion: *Our results explain the reported wide variations in $Pa–PETCO_2$ that accompany perturbations of cardiac output. Our observations pertain to the unsteady state only. The results suggest that $PETCO_2$ can be used to estimate changes in CI with a reasonable degree of confidence.*

Objectif: *On a décrit une relation directe entre l'index cardiaque (IC) et la PCO_2 téléexpiratoire ($PETCO_2$) immédiatement après une baisse de l'IC, mais la PCO_2 artérielle n'avait pas été mesurée simultanément. L'objectif des auteurs était de combler ce manque d'information concernant les effets immédiats des altérations de l'IC sur la $PaCO_2$, la $PETCO_2$ et par conséquent de la différence $Pa–PETCO_2$.*
Méthodes: *Les auteurs ont mesurés l'IC, la Pa et $PETCO_2$ et en ont calculé la différence chez 20 patients programmés pour une chirurgie cardiaque non urgente avant et immédiatement après la sternotomie. Les mesures ont été réalisées selon les méthodes standards: la thermodilution pour l'IC, l'analyse aux infrarouges pour la $PETCO_2$ et des gaz artériels pour $PaCO_2$. Les résultats ont été analysés par régression linéaire.*
Résultats: *Des changements importants, directs et immédiats de PET et $PaCO_2$ ont été notés avec les changements de l'IC. Les ratios étaient respectivement de 3,8 et 4,2 mmHg L^{-1}. Les valeurs calculées de r étaient de 0,75 ($P < 0,001$) pour la $PETCO_2$ et de 0,74 ($P < 0,005$) pour la $PaCO_2$. L'ordre de grandeur des changements individuels de PCO_2 a varié considérablement de sorte que les altérations de la différence $Pa–PETCO_2$ ont aussi fluctué sans corrélation avec la direction ou l'importance des changements de l'IC.*
Conclusions: *Ces résultats expliquent la grande variabilité de $Pa–PETCO_2$ qui accompagnent les perturbations de l'IC. Ces observations ne s'appliquent qu'à un état d'instabilité. Les résultats suggèrent que la $PETCO_2$ peut être utilisée avec une degré de confiance raisonnable pour évaluer les changements de l'IC.*

Key words
ANAESTHESIA: monitoring, cardiac output (index), arterial PCO_2, end-tidal PCO_2.

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Continuous end-tidal CO_2 tension ($PETCO_2$) measurements during steady state anaesthesia reliably estimate arterial PCO_2 ($PaCO_2$). Thus the $Pa–PETCO_2$ gradient will not change when ventilation, cardiac output and CO_2 production are constant.[1] The considerable changes in cardiac index (CI) during major vascular surgery cause marked variations in the $Pa–PETCO_2$ difference.[2] A direct relationship between CI and $PETCO_2$ during aortic surgery has been reported recently.[3] The authors, however, did not measure the simultaneous changes in arterial PCO_2. The immediate influence of acute changes in CI on $PaCO_2$ and $Pa–PETCO_2$ is thus not known. The purpose of our study was to provide that information.

Methods

The study was approved by the Research and Ethics Committee of our hospital. The sample consisted of 20

ASA 3–4 adults undergoing elective coronary artery bypass grafting (CABG), some with valve replacement. The age range was from 44 to 79 yr and the weights and heights ranged from 64 to 90 kg and 156 to 175 cm respectively.

The premedication and the anaesthetic agents used were at the individual anaesthetist's discretion but consisted of opioids (fentanyl or sufentanil) by *iv* infusion or intermittent injection and isoflurane in O_2 enriched air, supplemented by major tranquilizers and muscle relaxants to facilitate controlled ventilation. The lungs were ventilated at 10 $ml \cdot kg^{-1}$ at 10 $b \cdot min^{-1}$. Venous, arterial and pulmonary artery catheters were inserted percutaneously.

The measurements were made at two specific points: just before and immediately after sternotomy because of the expected intensity of the surgical stimulus. Cardiac index was measured by thermodilution in triplicate and arterial and mixed venous blood were sampled for immediate analysis of PCO_2 by a GEM "premier" (Mallinckrodt®) analyzer. This analyzer is calibrated every morning for CO_2 tensions in the range of 35 to 79 mmHg and is then recalibrated automatically every 20 min. End-tidal PCO_2 was measured by a Hewlett-Packard® infra red CO_2 analyzer attached to the proximal end of the tracheal tube. The analyzer is calibrated daily in the range of 0 to 55 mmHg. During the withdrawal of the blood samples, the mean of three consecutive $P_{ET}CO_2$s was calculated and recorded.

The data were analyzed by least squares linear regression using a commercial statistical package (Instat 2). A *P* value <0.05 was considered significant.

Results

Cardiac index increased in seven patients, decreased in 11, and was unchanged in two patients. In almost all instances, Pa and $P_{ET}CO_2$ changed in the same direction as CI. The magnitude of PCO_2 change was variable. The range of change (Δ in CI was −1.0 to +1.0 L. Figure 1 shows the individual changes in $P_{ET}CO_2$ and in $PaCO_2$ versus ΔCI. The lines of best fit are also shown. A significant relationship was found in the case of Pa and $P_{ET}CO_2$ but not in the case of mixed venous PCO_2. The slopes of arterial and end-tidal PCO_2 were close (3.83 vs 4.24 $L \cdot mmHg^{-1}$), but not parallel. There was marked individual variation in Pa and $P_{ET}CO_2$, such that the difference also changed variably (Figure 2). There were no negative Pa–$P_{ET}CO_2$s.

The results of the regression analysis are shown in the Table.

Discussion

We report direct and immediate changes in P_{ET} and $PaCO_2$ with changes in CI (Figure 1), without significant changes in mixed venous PCO_2. These observations were made during increases and decreases in CI. We also found that the changes in Pa–$P_{ET}CO_2$ difference were variable in direction and magnitude and that the direction was not related to the direction of change in CI (Figure 2).

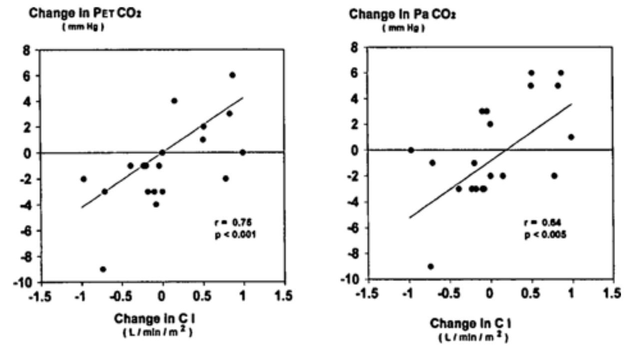

FIGURE 1 Change in Pa and $P_{ET}CO_2$ with change in Cardiac Index.

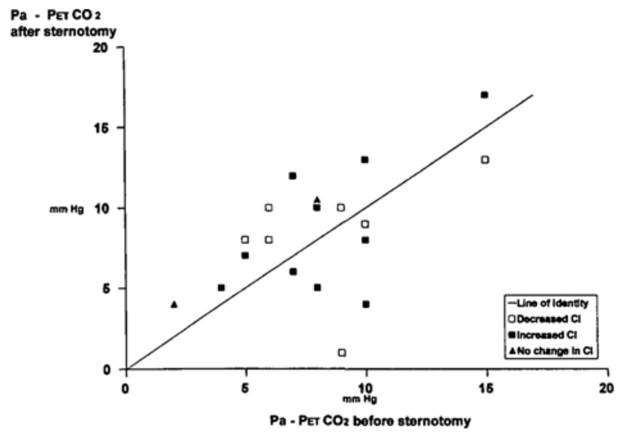

FIGURE 2 Arterial: end-tidal PCO_2 gradient.

We anticipated that sternotomy would increase CI. The fact that CI decreased in 11 patients suggests that anaesthesia was deeper than the usual clinical signs, blood pressure and heart rate, indicated. It is also possible that in some instances the greatly increased afterload may have prevented the expected increase in CI from occurring.

We report a 3.8 mmHg change in $P_{ET}CO_2$ per litre change in CI. This ratio is similar to the reported 3–4 mmHg per litre change with reduced CI during abdominal aortic aneurysmectomy (AAA).[3] and CABG.[4] In both reports, the direct relationship was also statistically significant (r = 0.82 and 0.87 respectively). Thus a knowledge of changes in $P_{ET}CO_2$ alone can give a use-

TABLE

Variable examined					Confidence limits			
Independant	*Dependant*	*Intercept*	*Slope*	*SE*	*Upper*	*Lower*	*r*	*P*
ΔCI	$\Delta P_{ET}CO_2$	−0.06	3.83	0.78	5.48	2.17	0.75	<0.001
ΔCI	$\Delta PaCO_2$	0.02	4.24	1.29	6.97	1.29	0.64	<0.005
ΔCI	$\Delta PvCO_2$	−2.59	2.79	1.55	−0.48	6.08	0.41	<0.09

ful estimate of changes in cardiac index, assuming that metabolism and alveolar ventilation are unchanged.

We are unaware of studies presenting data on Pa *and* $P_{ET}CO_2$. The fact that there was variability in the Pa–$P_{ET}CO_2$ difference and that mixed venous PCO_2 did not change are probably due to the unsteady state at the time of our measurements. The variability in Pa–$P_{ET}CO_2$ coincident with changes in CI explains the variability in that difference reported by others.[3] Indeed, when CI is constant during surgery, the Pa–$P_{ET}CO_2$ difference also remains stable.[1]

The temporal effects of altered CI on Pa and $P_{ET}CO_2$ in our patients are unknown, but information from the canine model of shock has been published. The inflation and deflation of a balloon placed in the venae cava caused acute decreases and increases in cardiac output respectively and the percent changes in Pa and $P_{ET}CO_2$ were directly related to the percent change in cardiac output.[6] The maximum change was reached after six to eight breaths (approximately 45 sec). The major difference between our results and those of the canine study is the Pa–$P_{ET}CO_2$. In our study, the change in Pa–$P_{ET}CO_2$ varied in direction and magnitude, whereas the Pa–$P_{ET}CO_2$ difference in the dogs was inversely related with CI. In the canine study, measurements of PCO_2 were made in a quasi-steady state, that is when the changes in $P_{ET}CO_2$ were at their maximum. This was attained within one minute. We can only assume that the changes in dead space ventilation [(V_D/V_T = $PaCO_2$–$P_{ET}CO_2/PaCO_2$], expected with alterations in cardiac output occur shortly after but not coincidently with CI changes. It has been demonstrated, in dogs, that during a maintained low cardiac output state, the initial reduction in Pa and $P_{ET}CO_2$ is followed by a progressive increase starting less than ten minutes after the acute reduction in cardiac output.[6,4] This raises the question of whether the effects of alterations of CI on shunt are also biphasic.

In summary, our results indicate that (1) the magnitude of alterations in CI can be estimated from a knowledge of the accompanying change in $P_{ET}CO_2$ and (2) that the immediate effect of alterations of CI on Pa–$P_{ET}CO_2$, and hence on V_D/V_T are variable in magnitude and direction.

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References

1 *Sharma SK, McGuire GP, Cruise CJE.* Stability of the arterial to end-tidal carbon dioxide difference during anaesthesia for prolonged neuro-surgical procedures. Can J Anaesth 1995; 42: 498–503.

2 *Raemer DB, Francis D, Philip JH, Gabel RA.* Variations in PCO_2 between arterial blood and peak expired gas during anesthesia. Anesth Analg 1983; 62: 1065–9.

3 *Shibutani K, Muraoka M, Shirasaki S, Kubal K, Sanchala VT, Gupte P.* Do changes in end-tidal PCO_2 quantitatively reflect changes in cardiac output? Anesth Analg 1994; 79: 829–33.

4 *Deriaz H, Song Q, Delva E, Lienhart A.* Relationship between cardiac output and end-tidal carbon dioxide tension in anesthetized patients. Anesthesiology 1993; 79: A509.

5 *Isserles SA, Breen P.* Can changes in end-tidal PCO_2 measure changes in cardiac output? Anesth Analg 1991; 73: 808–14.

6 *Morimoto Y, Kemmotsu O, Murakami F, Yamamura T, Mayumi T.* End-tidal CO_2 changes under constant cardiac output during cardiopulmonary resuscitation. Crit Care Med 1993; 21: 1572–6.