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Transcutaneous CO_2 tension measurement as an indicator of severity of hemorrhagic shock

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Abstract: This study was undertaken to evaluate whether transcutaneous CO_2 tension ($PtcCO_2$) could be used as an indicator of the global systemic severity of hemorrhagic shock. $PtcCO_2$ levels in ten anesthetized mongrel dogs were measured during hemorrhage and during volume restoration and were correlated with mixed venous CO_2 tension ($P\bar{v}CO_2$). After withdrawal of 30 ml·kg^{-1} blood, both $PtcCO_2$ and $P\bar{v}CO_2$ increased significantly (from 43 ± 7 to 70 ± 27 torr ($P < 0.05$) and from 48 ± 6 to 59 ± 12 torr ($P < 0.05$), respectively). Throughout the experiments, $PtcCO_2$ levels changed almost in parallel to $P\bar{v}CO_2$ levels. However, changes in $PtcCO_2$ exceeded those in $P\bar{v}CO_2$ from the end of hemorrhage, at which time cardiac output decreased to 35% of the baseline value, until the end of volume restoration, and the changes in $PtcCO_2$ showed a close logarithmic relationship with $P\bar{v}CO_2$ ($r = 0.78$, $n = 110$). Additionally, arterio-transcutaneous CO_2 tension gradients ($P[tc\text{-}\bar{v}]CO_2$) showed a close exponential correlation with cardiac output per body weight (CO/BW) during the shedding phase ($r = 0.85$, $n = 60$), although the correlation with CO/BW lessened during the retransfusion phase ($r = 0.55$, $n = 60$). $PtcCO_2$ was roughly correlated with $P\bar{v}CO_2$ during hemorrhagic shock, and levels of $PtcCO_2$ higher than $P\bar{v}CO_2$ reflected critical tissue perfusion.

Key words: Transcutaneous CO_2 tension, Mixed venous CO_2 tension, Hemorrhagic shock

Introduction

Venous and tissue hypercarbia is provoked during circulatory and other forms of shock, and has been related to reduced CO_2 clearance due to decreased pulmonary vascular flow, as well as to increased CO_2 generation due to the buffering of increased hydrogen ion production in the hypoxic cell [1]. It is now well recognized that mixed venous CO_2 tension ($P\bar{v}CO_2$) values indicate changes in tissue perfusion [2–5]. In recent years, several studies [6–9] have focused on increases in gastrointestinal mucosal CO_2 tension, which can be measured utilizing a tonometer or an ion-sensitive field-effect transistor (ISFET) sensor, during low-flow conditions. These studies have reported that PCO_2 values are reliable indicators of tissue hypoxia and a clinically useful predictor of prognosis. However, it is often difficult to obtain mixed venous blood from patients in shock or from pediatric patients, and the tonometric system cannot provide continuous information. Furthermore, the ISFET sensor system, in which a micro-sensor is surgically inserted into the submucosal tissue, is invasive and has not yet been clinically established. A noninvasive and continuous method of monitoring tissue CO_2 levels is needed to manage unstable patients during shock.

Transcutaneous CO_2 tension ($PtcCO_2$) monitoring has been widely accepted, particularly in neonatal intensive care, since it was developed by Huch et al. [10] in 1973. Animal experimental studies have shown that $PtcCO_2$ correlates directly with arterial CO_2 tension ($PaCO_2$) in normal circulation and correlates inversely with the cardiac index during low-flow shock [11]. These studies utilized very severe shock brought about by sudden withdrawal of blood, but the characteristics in moderate shock models have not yet been fully evaluated.

In the present study, we utilized gradient withdrawal of the blood to create the model and reconsidered whether $PtcCO_2$ could be an indicator of the global systemic severity of shock.

Materials and methods

Experimental preparation

The study protocol was reviewed and approved by the Institutional Animal Care and Use Committee. Ten

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adult mongrel dogs, weighing 7.6–13.4kg (mean, 9.7kg) were used. Anesthesia was induced with intramuscular ketamine ($10mg \cdot kg^{-1}$) and succinylcholine ($2mg \cdot kg^{-1}$), and was maintained with 1% isoflurane. After endotracheal intubation, ventilation was initiated with a Harvard animal respirator (Model B2; Igarashi, Tokyo, Japan), using 30% oxygen and a tidal volume of $10ml \cdot kg^{-1}$. The respiration rate was adjusted to 10–$20min^{-1}$ so that the end-tidal CO_2 tension was maintained at 35torr, measured with an expired CO_2 monitor (Respina IH31; San-ei, Tokyo, Japan). The settings of the respirator were not changed thereafter.

Experimental protocol

An endovascular catheter was inserted into the jugular vein and the right carotid artery for blood shedding and blood sampling. A 5-Fr thermodilution catheter was also inserted via the right jugular vein. After systemic heparinization ($5mg \cdot kg^{-1}$ intravenously), blood was withdrawn at a rate of $6ml \cdot kg^{-1} \cdot 5min^{-1}$. Blood was withdrawn five times, at 5-min intervals, and a total of $30ml \cdot kg^{-1}$ was removed. Subsequently, the blood was restored at the same rate.

Measurements

Four parameters were measured after each cycle of hemorrhagic shock and volume restoration: transcutaneous gas tension, arterial gas tension, mixed venous gas tension, and cardiac output. Transcutaneous gas tension was measured with a transcutaneous gas monitor (Models 632 and 634; Kontron, Everett, MA, USA). The probes were warmed to 43°C. The oxygen probe was calibrated with air, and the CO_2 probe was calibrated with 5% and 10% CO_2. The probes were then placed on the shaved anterior chest wall. Gas tension in arterial blood and mixed venous blood was measured with an automatic blood gas analyzer (Stat Profile 5; Novametrix Medical System, Wallingford, CT, USA) immediately after blood sampling. Cardiac output was measured by the thermodilution method, using a cardiac output computer (9520A; Baxter Edward Laboratory, Irvine, CA, USA).

Statistical analysis

The measured variables were evaluated by analysis of variance (two-factor) with repeated measures, followed by Bonferroni's correction for multiple comparisons. Regression analysis and calculation of correlation coefficients were performed for $PtcCO_2$, $PaCO_2$, and $P\bar{v}CO_2$, as well as for cardiac output per body weight (CO/BW) and arterio-transcutaneous CO_2 tension gradients ($P[tc\text{-}a]CO_2$), at each point of measurement. Differences were considered significant at $P < 0.05$.

Results

Changes in CO/BW

CO/BW decreased to 35% of the baseline value by gradient hemorrhage of $30ml \cdot kg^{-1} \cdot 50min^{-1}$ and recovered to the baseline value on the retransfusion of 24 $ml \cdot kg^{-1} \cdot 40min^{-1}$. After the retransfusion of all shed blood, CO/BW increased to 112% of the baseline value (Table 1).

Changes in $PaCO_2$, $P\bar{v}CO_2$, and $PtcCO_2$ (Table 1, Fig. 1)

$PaCO_2$ remained relatively unchanged during hemorrhage and then rose slightly after volume restoration.

The mean baseline $P\bar{v}CO_2$, of 48 ± 6torr, began rising when CO/BW was approximately 50% of its baseline value. $P\bar{v}CO_2$ reached a peak 20min after the beginning of volume restoration, increasing to 59 ± 12torr ($P < 0.05$) and then decreasing to the pre-shock level.

$PtcCO_2$ changed almost in parallel to $P\bar{v}CO_2$. The mean baseline $PtcCO_2$ was 43 ± 7torr, and the value increased to 70 ± 27torr ($P < 0.05$) 10min after the

Table 1. Values of all variables measured during hemorrhage and retransfusion (means ± SD)

	Baseline	Hemorrhage					Retransfusion				
	0 min	10 min	20 min	30 min	40 min	50 min	60 min	70 min	80 min	90 min	100 min
CO/BW ($ml \cdot min^{-1} \cdot kg^{-1}$)	158 ± 31	135 ± 26	112 ± 25**	92 ± 25**	74 ± 18**	56 ± 21**	84 ± 28**	112 ± 36**	136 ± 34	159 ± 39	177 ± 46
PaO_2 (torr)	118 ± 17	115 ± 19	114 ± 20	112 ± 21	115 ± 19	117 ± 18	105 ± 22	102 ± 24	103 ± 27	103 ± 23	100 ± 20
$P\bar{v}O_2$ (torr)	49 ± 6	45 ± 7	41 ± 6*	37 ± 8**	33 ± 6**	28 ± 8**	34 ± 7**	41 ± 8*	45 ± 8	47 ± 8	50 ± 7
$PtcO_2$ (torr)	97 ± 16	91 ± 15	84 ± 16	72 ± 17**	53 ± 21**	36 ± 20**	46 ± 24**	58 ± 22**	70 ± 21**	79 ± 19	84 ± 17
$PaCO_2$ (torr)	39 ± 5	38 ± 6	38 ± 7	38 ± 7	38 ± 8	36 ± 8	42 ± 11	45 ± 11	44 ± 10	44 ± 10	43 ± 10
$P\bar{v}CO_2$ (torr)	48 ± 6	47 ± 5	47 ± 6	48 ± 9	51 ± 7	53 ± 7	58 ± 11*	59 ± 12*	55 ± 10	51 ± 11	50 ± 9
$PtcCO_2$ (torr)	43 ± 7	42 ± 8	43 ± 9	46 ± 12	51 ± 16	61 ± 23	70 ± 27*	69 ± 29*	64 ± 28	59 ± 23	54 ± 19
$P(\bar{v}\text{-}a)CO_2$ (torr)	8 ± 4	9 ± 3	9 ± 4	10 ± 5	14 ± 6	17 ± 8*	17 ± 7*	14 ± 8	11 ± 5	7 ± 6	7 ± 5
$P(tc\text{-}a)CO_2$ (torr)	3 ± 4	4 ± 4	5 ± 6	8 ± 8	14 ± 12*	25 ± 19**	28 ± 21**	25 ± 23*	20 ± 21	14 ± 17	11 ± 15

* $P < 0.05$; ** $P < 0.01$ compared with baseline values.
CO/BW, cardiac output per body weight; $P(\bar{v}\text{-}a)CO_2$, arteriovenous PCO_2 gradient; $P(tc\text{-}a)CO_2$, arterio-transcutaneous PCO_2 gradient.

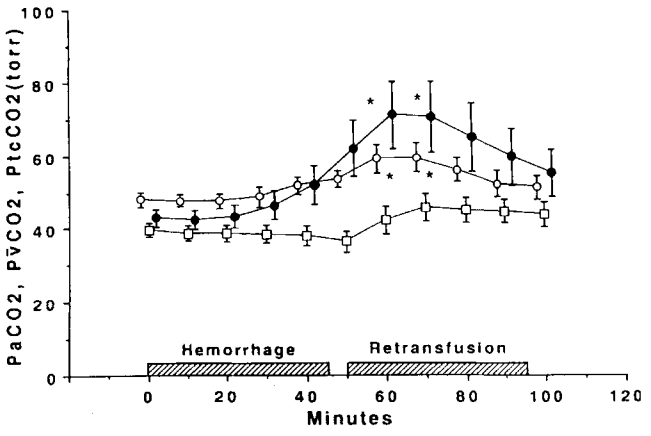

Fig. 1. Changes in $PaCO_2$ (*squares*), mixed venous CO_2 tension ($P\bar{v}CO_2$; *circles*) and transcutaneous CO_2 tension ($PtcCO_2$; *dots*) during hemorrhagic and retransfusion. *$P < 0.05$ compared with baseline values. Values are means ± SEM

beginning of volume restoration. Thus, in the early phase of hemorrhage, the $PtcCO_2$ value lay between the values for $PaCO_2$ and $P\bar{v}CO_2$, but it subsequently increased, exceeding $P\bar{v}CO_2$ and remaining higher until the end of volume restoration. $PtcCO_2$ did not return to control values until 20 min after the recovery of cardiac output.

Relationship of $PaCO_2$, $P\bar{v}CO_2$, and $PtcCO_2$

Throughout this experiment, $PtcCO_2$ showed a closer logarithmic correlation to $P\bar{v}CO_2$ ($r = 0.78$, $n = 110$) than to $PaCO_2$ ($r = 0.68$, $n = 110$) (Fig. 2). Additionally, in several cases, $PtcCO_2$ values increased to about two-fold over $P\bar{v}CO_2$ values from the end of hemorrhagic shock until the early phase of volume restoration, although there was no significant difference between $PtcCO_2$ and $P\bar{v}CO_2$ (Table 1, Fig. 2).

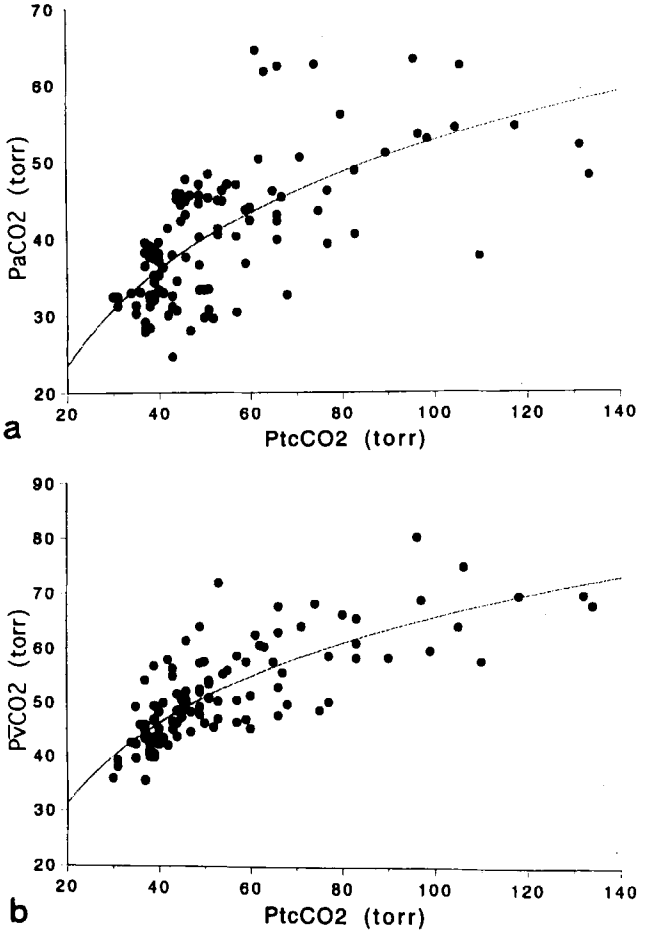

Fig. 2a,b. Relationship between $PtcCO_2$ and both **a** $PaCO_2$ and **b** $P\bar{v}CO_2$ during hemorrhage and retransfusion. **a**, $y = 18 * \ln(x) - 30.5$; $n = 110$; $r = 0.68$; **b**, $y = 21.4 * \ln(x) - 32.9$; $n = 110$; $r = 0.78$

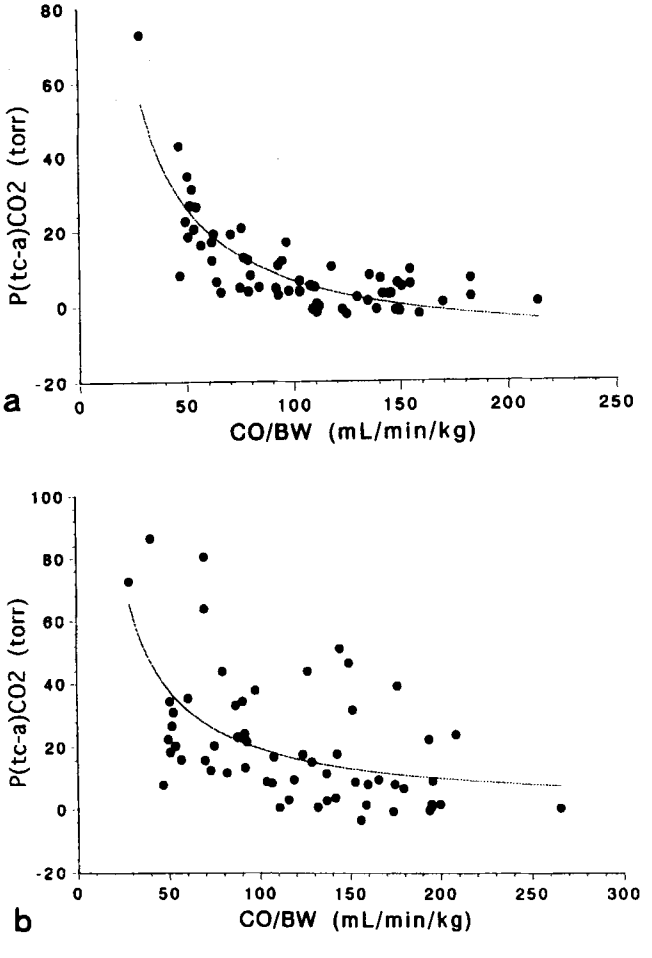

Fig. 3a,b. Relationship between cardiac output per body weight (*CO/BW*) and arterio-transcutaneous PCO_2 gradient ($P[tc\text{-}a]CO_2$) during **a** hemorrhage and **b** retransfusion. **a**, $y = 1401/x - 12.18$; $n = 60$; $r = 0.85$; **b**, $y = 1872/x + 0.8$; $n = 60$; $r = 0.55$

Relationship of P(tc-a)CO_2 and CO/BW

P(tc-a)CO_2 showed a close exponential correlation with CO/BW during the shedding phase ($r = 0.85$, $n = 60$). However, the correlation with CO/BW lessened during the retransfusion phase ($r = 0.55$, $n = 60$) (Fig. 3).

Discussion

A major finding of the present experimental study was the close logarithmic correlation between $PtcCO_2$ and $P\bar{v}CO_2$. This suggests that $PtcCO_2$, as well as $P\bar{v}CO_2$, reflects whole body hypercarbia fairly well during hemorrhagic shock. However, as cardiac output was reduced, values for $PtcCO_2$ differed from both $PaCO_2$ and $P\bar{v}CO_2$ values, remaining in excess of $P\bar{v}CO_2$ until the end of volume restoration. These changes may reflect two factors, the first being the disturbed distribution of blood flow to the various organs caused by the progression of shock. Previous studies indicate that, of all the vascular beds, the cutaneous vascular bed exhibits the greatest vasoconstrictive response and the most severe blood flow reduction during progressive blood loss, since this vascular bed possesses no local intrinsic blood flow regulation (i.e., autoregulation) and is under strong extrinsic (e.g., neural/humoral) control [12]. During circulatory shock, the skin is one of the first organs in which blood flow reduction occurs and the skin is also among the last organs to be restored to metabolic balance during resuscitation from shock [13–15]. Accordingly, the measurement of $PtcCO_2$ may be advantageous for the early diagnosis and assessment of shock, although this method may overestimate the extent of systemic injury.

The second factor that could explain the discrepancy between $PtcCO_2$ and $P\bar{v}CO_2$ values is a reduction in the washout of tissue gases into venous blood due to severe peripheral circulatory impairment. From this perspective, not only the conventional analysis of arterial gases but also the analysis of venous gases has limited value in the presence of severe shock and does not necessarily provide direct information about the global adequacy of oxygenation. In other words, only the monitoring of tissue gases can warn of critical conditions.

Johnson and Weil [1] found an exponential correlation between arteriovenous PCO_2 gradient ($P[\bar{v}\text{-}a]CO_2$) and cardiac output during hypoperfusion, using the Fick equation. This was in accord with the correlation between P(tc-a)CO_2 and CO/BW found in the present study. P(tc-a)CO_2 should provide an indication of the severity of inadequate cutaneous perfusion analogous to the arteriovenous PCO_2 gradient during global hypoperfusion [3–5]. As shown in Fig. 3, the curve increased steeply at approximately 50–60 $ml \cdot min^{-1} \cdot kg^{-1}$. According to the method described by Samsel and Schumacker [16], this point may be a critical value for skin, and seems to correspond to the point at which $PtcCO_2$ became higher than $P\bar{v}CO_2$ during the shedding phase.

Several other studies have focused on increases in PCO_2 in various tissues, such as the gastrointestinal (GI) tract [6–9], the myocardium [17], the brain [18], and the muscle [19] as an indicator of the adequacy of tissue oxygenation. Hypercarbia in various tissues appears to be the predominant phenomenon during hypoperfusion. PCO_2 in GI mucosa has been particularly well investigated during various types of shock, such as hemorrhagic [8,9], septic [20], cardiogenic [7], and anaphylactic [9], and this parameter has been reported to be an extremely sensitive indicator of systemic damage, since the GI mucosa is quite vulnerable to even mild hypoxia.

Tang and coworkers (Noc et al. [8] and Tang et al. [9]) monitored gastric intramural PCO_2 during hemorrhage and volume restoration, utilizing an ISFET sensor. The changes they reported in gastric intramural PCO_2 were in approximate agreement with those we found in $PtcCO_2$ in the present experiment. It appears that $PtcCO_2$ monitoring compares favorably with GI mucosal PCO_2 monitoring in terms of precision and reliability, at least during hemorrhage and volume restoration. Moreover, $PtcCO_2$ monitoring is a noninvasive and clinically well established method, this constituting an advantage over the ISFET sensor system. Further study is required to determine whether the present observations apply to other types of shock.

The monitoring of $PtcCO_2$ entails several problems for accurate interpretation of hypercarbia throughout the body. One problem arises from the disturbed distribution of blood flow among various tissues, as described above. This disturbed distribution is also seen among various skin regions [21]. An additional problem is related to measurement techniques. When the probe is heated, measurements can be altered by arterialization of the capillaries, by CO_2 production following metabolic enhancement, by changes in the CO_2 transporting capacity, and by diffusion resistance [10,22,23]. Hence, it should be considered that the values obtained while hypoperfusion is monitored may vary widely.

In conclusion, $PtcCO_2$ roughly correlated with $P\bar{v}CO_2$ during hemorrhagic shock, and $PtcCO_2$ levels higher than $P\bar{v}CO_2$ levels reflected critical tissue perfusion. $P\bar{v}CO_2$ monitoring provided more precise information about global systemic change than $P\bar{v}CO_2$ analysis, particularly in conditions of severe low blood flow.

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