TITLE: **END-TIDAL PCO_2 REFLECTS CHANGES OF CARDIAC OUTPUT**

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End-tidal CO_2 ($PetCO_2$) tension often decreases during hypotension or hemorrhage and increases during surgical stimulation or blood transfusion. In this study, we examined the possible mechanisms of $PetCO_2$ changes during blood withdrawal both in human and in dogs.

In human studies (Table 1), ventilation was maintained constant and $PetCO_2$ was monitored by Nellcor 1000 when 1000 ml of blood were harvested prior to cardio-pulmonary bypass in 10 patients undergoing coronary artery bypass graft surgery. In animal studies (n=10), blood was withdrawn in two stages (Stage I and Stage II) to produce progressive decrease of oxygen delivery ($\dot{D}O_2$) as described in Table 2.

During blood withdrawal, both in human and dog studies, $PetCO_2$ and cardiac output (CO) decreased significantly ($P<0.05$), while mixed venous pH (pHv) and PCO_2 ($P\bar{v}CO_2$) were unchanged. Arterial PCO_2 ($PaCO_2$) decreased insignificantly in human studies but significantly decreased in dog studies at Stage I. Increases of arterial end-tidal PCO_2 difference ($a\text{-}ePCO_2$) were insignificant.

During transient changes of CO, content of CO_2 in mixed venous blood apparently did not change. Therefore, total amount of CO_2 returning to the heart depends primarily on CO. The cause of decreases of $PetCO_2$ during transient decrease of CO may be attributable to decreases of total amount of CO_2 return rather than due to ventilation perfusion abnormalities. $PetCO_2$ monitoring can be a useful noninvasive method to assess changes of CO in the clinical states, where ventilation is maintained constant.

Table 1. Human Studies

	Prior to blood withdrawal	Following blood withdrawal
$PetCO_2$: mmHg	26.0 ± 2.7	24 ± 1.8*
CO : l	3.7 ± 0.9	2.7 ± 0.5*
pHa	7.46 ± 0.05	7.50 ± 0.09
$PaCO_2$: mmHg	32.5 ± 3.8	31.5 ± 2.4
$pH\bar{v}$	44 ± 0.04	7.43 ± 0.04
$P\bar{v}CO_2$: mmHg	37.4 ± 9.4	37.1 ± 3.6
ΔpH	4.1 ± 1.3	6.3 ± 1.3*
ΔPCO_2 : mmHg	6.6 ± 1.5	7.2 ± 1.7
$a\text{-}ePCO_2$: mmHg	5.1 ± 3.7	6.6 ± 2.8

Table 2. Dog Studies

$\Delta pH=pHa-p\bar{H}v$

$\Delta PCO_2=PaCO_2-P\bar{v}CO_2$

$\dot{V}ECO=CO_2$ output

*=P<0.05 as compared to C

	C	Stage I	Stage II
$\dot{V}ECO_2$: ml/min	127±22	106±23*	92±33*
CO : l	3.3±0.9	1.5±0.5*	0.99±0.38*
$\dot{D}O_2$: ml/min.kg	29±7	15.4±6*	9.6±3.8*
pHa	7.32±.08	7.33±.08	7.25±.11
$PaCO_2$: mmHg	44±7.1	38.5± 5.4	40±8.1
$pH\bar{v}$	7.29±.09	7.25±.11*	6.81±.48*
$P\bar{v}CO_2$: mmHg	50±9	52±11	62±14*
ΔpH	0.03±0.02	0.08±0.04*	0.09±0.03*
ΔPCO_2 : mmHg	-5.9±4.7	-14±9.0*	-22±11*

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TITLE: CO-Hb IN 1000 SMOKERS AND NONSMOKERS, THE BIOLOGICAL HALF LIFE OF COHb, AND CONSEQUENCES ON PULSOXIMETRY

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Pulse oximetry currently in use determines the oxygen saturation of hemoglobin (S_pO_2) on the basis of BEER's law using light of only two wavelengths: 660 and 940 nm. Thus, in the presence of carboxyhemoglobin (COHb) or methemoglobin (MetHb) in human blood the fractional hemoglobin saturation
(1) $SaO_{2frac} = O_2Hb:(O_2Hb + Hb + COHb + MetHb)x100\%$
cannot be exactly determined - four wavelengths would be required for analysis of SaO_{2frac} [1].

We investigated in the present study with informed consent and approval of the local research committee in which patients COHb and MetHb might complicate the interpretation of S_pO_2 and impair oxygen transport. The endpoints were the following:
A - distribution of COHb and MetHb in 1000 smoking (S) and nonsmoking (NS) out-patients,
B - COHb1 before (at arrival in the hospital) and COHb2 after preoperative stop smoking > 10 h (at introduction of anaesthesia) in 50 heavy S,
and the biological half life ($t_{1/2}$) of COHb in
C - 9 volunteer S overnight from two measurements, 1-after the last, and 2-before the first cigarette
D - 13 volunteer S during moderate daily activity, calculated from 11 hourly measurements - first sample drawn after stop smoking (COHb-kinetics).

Heparinized samples of 200 µl (as a minimum) of venous blood were measured immediately after withdrawal by the in-vitro CO-oximeter Corning 2500 (Ciba-Corning). Differences were tested in parametric data by t-test and in nonparametric data by Wilcoxon test.
Significance ($p<0.05$) S vs NS *, men (m) vs women (w) +.
Mean (mn) and/or single values (sv) are presented.

	total	w	m	ns	s	w/ns	w/s	m/ns	m/s
n	1000	381	619	630	370	288	93	342	277
A COHb(mn)	3.05	2.45	3.41	1.82	5.13*	1.78	4.52*	1.85	5.34*
MetHb(mn)	0.66	0.69	0.64	0.66	0.66	0.69	0.66	0.63	0.65

B n=50 (12w, 38m), COHb1(mn)=6.87±1.83, COHb2(mn)=3.77±1.11

	w	(sv)			m	(sv)			
C n=9	(mn)+	1	2	3	4	(mn)	5	6	7	8	9
(sleep)$t_{1/2}$	3.18	3.23	2.32	4.30	2.85	4.67	5.59	4.73	7.06	5.58	6.4

		(sv)						
D n=13	w(mn)+	1	2	3	4	5	6	7
(awake)$t_{1/2}$	2.61	3.20	2.65	2.18	2.35	2.72	2.71	2.44
	m(mn)	8	9	10	11	12	13	
	3.69	3.39	4.18	4.02	3.69	3.38	3.46	

We conclude from these data that
1. COHb in S (5.13±2.25), even when they stopped smoking (after 10 h: 3.77±1.11) may complicate the interpretation of S_pO_2, whereas COHb in NS (1.82±0.3) and MetHb (0.66±0.22) are constant. Thus, pulse oximetry by more than two wavelengths appears desirable.
2. As $t_{1/2}$ of COHb greatly varies, a more than 10 hour period of stop smoking preoperatively may be recommended. On behalf of the significantly shorter $t_{1/2}$ of COHb in women, women might be allowed to stop smoking closer before anaesthesia than men.

References: 1. Anesthesiology 70:98-108, 1989

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