

TITLE: END-TIDAL PCO₂ REFLECTS CHANGES OF CARDIAC OUTPUT

AUTHORS: K. Shibutani, M.D., T. Komatsu, M.D., N. Kashiwagi, M.D., M. Bairamian, M.D., V. Kumar, M.D.

AFFILIATION: Anes. Dept., New York Medical College, Valhalla, NY 10595

End-tidal CO₂ (PetCO₂) tension often decreases during hypotension or hemorrhage and increases during surgical stimulation or blood transfusion. In this study, we examined the possible mechanisms of PetCO₂ changes during blood withdrawal both in human and in dogs.

In human studies (Table 1), ventilation was maintained constant and PetCO₂ 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 In animal studies (n=10), blood was withdrawn in two stages (Stage I and Stage II) to produce progressive decrease of oxygen delivery (DO₂) as described in Table 2.

During blood withdrawal, both in human and dog studies, PetCO₂ and cardiac output (CO) decreased significantly (P<0.05), while mixed venous pH (pHv) and PCO₂ (PvCO₂) were unchanged. Arterial PCO₂ (PaCO₂) decreased insignificantly in human studies but significantly decreased in dog studies at Stage I. Increases of arterial end-tidal PCO₂ difference (a-ePCO₂) were insignificant.

During transient changes of CO, content of CO₂

in mixed venous blood apparently did not change. Therefore, total amount of CO₂ returning to the heart depends primarily on CO₂. The cause of decreases of PetCO₂ during transient decrease of CO may be attributable to decreases of total amount of CO₂ return rather than due to ventilation perfusion abnormalities. PetCO₂ 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*
pH _a	7.46 ± 0.05	7.50 ± 0.09
PaCO ₂ : mmHg	32.5 ± 3.8	31.5 ± 2.4
pH _v	7.44 ± 0.04	7.43 ± 0.04
PvCO ₂ : mmHg	37.4 ± 9.4	37.1 ± 3.6
Δ pH	4.1 ± 1.3	6.3 ± 1.3*
Δ PCO ₂ : mmHg	6.6 ± 1.5	7.2 ± 1.7
a-ePCO ₂ : mmHg	5.1 ± 3.7	6.6 ± 2.8

Table 2. Dog Studies

	C	Stage I	Stage II
Δ pH = pH _a - pH _v			
Δ PCO ₂ = PaCO ₂ - PvCO ₂			
VECO ₂ = CO ₂ output			
DO ₂ : ml/min.kg			
pH _a	7.32 ± 0.08	7.33 ± 0.08	7.25 ± 0.11
PaCO ₂ : mmHg	44 ± 7.1	38.5 ± 5.4	40 ± 8.1
pH _v	7.29 ± 0.09	7.25 ± 0.11*	6.81 ± 0.48*
PvCO ₂ : mmHg	50 ± 9	52 ± 11	62 ± 14*
Δ pH	0.03 ± 0.02	0.08 ± 0.04*	0.09 ± 0.03*
Δ PCO ₂ : mmHg	-5.9 ± 4.7	-14 ± 9.0*	-22 ± 11*

* = P<0.05 as compared to C

A507

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

AUTHORS: A. DELLER, M.D., K. FORSTNER, R. STENZ, M.N. SCHREIBER, M.D., T. FÖSEL, M.D.

AFFILIATION: UNIVERSITÄTSKLINIK FÜR ANÄSTHESIOLOGIE, POSTFACH 3880, D-7900 ULM, W. GERMANY

Pulse oximetry currently in use determines the oxygen saturation of hemoglobin (S_{O₂}) 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) $SaO_{2,frac} = O_2Hb / (O_2Hb + Hb + COHb + MetHb) \times 100\%$ cannot be exactly determined - four wavelengths would be required for analysis of $SaO_{2,frac}$.

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_{O₂} 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 - COHb 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

	w(mn)*	1	2	3	4	5	6	7
D n=13 (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_{O₂}, 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