

## REVERSIBLE DECREASE OF OXYGEN CONSUMPTION BY HYPEROXIA

K. Reinhart, M.D., F. Bloos, F. Koenig, D. Bredle, Ph.D., L. Hannemann, M.D.

Dept. of Anesthesiology, Klinikum Steglitz, Free University, Berlin, Germany

Dept. of Physiology and Biophysics, Univ. of Alabama at Birmingham, USA

**Introduction.** Preoxygenation is routinely used prior to intubation and trachea suctioning in critically ill patients to increase alveolar  $O_2$  in periods of hypoventilation. In preliminary work, however, we found with  $O_2$  breathing for 10 min a decrease in oxygen consumption ( $\dot{V}O_2$ ) resulting from decreases in both cardiac output and arteriovenous  $O_2$  content difference ( $avDO_2$ ). This study further explored this  $O_2$  supply/demand paradox, particularly the time course and reversibility of 100%  $O_2$  ventilation on  $O_2$  delivery and uptake. As an indication of how microcirculatory changes might be coupled to metabolic changes, we also measured tissue  $PO_2$  in leg muscle.

**Methods.** 20 critically ill, ventilated patients requiring monitoring with pulmonary artery catheters were studied.  $O_2$  content in arterial ( $CaO_2$ ) and mixed-venous blood was derived from Hb concentration,  $O_2$  saturation (IL-282 Co-oximeter) and  $PO_2$  ( $PaO_2$ ,  $PvO_2$ ). Cardiac output was measured in triplicate by thermodilution.  $O_2$  delivery ( $\dot{D}O_2$ ) was calculated as the product of cardiac index (CI) and  $CaO_2$ .  $\dot{V}O_2$  was calculated as the product of CI and  $avDO_2$ . Measurements were taken at 30 min intervals during hemodynamically stable periods in the following stages: 1) baseline,  $FI_{O_2} < 0.50$  but sufficient to maintain  $PaO_2 > 100$  mmHg; 2) 90 min at  $FI_{O_2} = 1.0$ , and 3) 30 min with  $FI_{O_2}$  at baseline values. Tissue  $PO_2$  values were obtained from the quadriceps femoris muscle using a fast responding polarographic-type hypodermic needle probe (response time 90-500 ms). 200 samples at each stage were displayed as a  $PO_2$  histogram. Data were analyzed with paired Student's t-test.

**Results.** Hemodynamic and  $O_2$  transport variables are shown in the table. With 100%  $O_2$  ventilation,  $PaO_2$  increased and CI was unchanged, thus  $\dot{D}O_2$  increased. However,  $O_2$  extraction ratio ( $O_{2e.r.}$ ) was reduced, particularly during the first 30 min. Thus  $\dot{V}O_2$  was decreased 12% at 30 min of hyperoxia. Mean tissue  $PO_2$  increased gradually in hyperoxia, but the difference from baseline did not reach significance until 90 min. Upon return to normoxia, CI and  $\dot{D}O_2$  increased,  $O_{2e.r.}$  remained constant, and  $\dot{V}O_2$  and tissue  $PO_2$  returned to baseline. Systemic vascular resistance (SVR) was decreased.

**Discussion.** The paradoxical decrease in  $\dot{V}O_2$  despite an increase in  $\dot{D}O_2$  suggests maldistribution of blood flow and functional  $O_2$  shunting. This is substantiated by an increase in  $PvO_2$  and a decrease in  $O_{2e.r.}$ . Similar findings have been found in an animal model.<sup>1</sup> Increased  $PO_2$  causes vasoconstriction and reduction of capillary density and flow in muscle preparations.<sup>2</sup> If microcirculatory control mechanisms protect tissue  $PO_2$  during hyperoxia, overshooting of such response might lead to areas of relative tissue hypoxia. This hypothesis is

supported by the finding that mean tissue  $PO_2$  did not increase after 30 min hyperoxia. This resulted from a less normal distribution of the tissue  $PO_2$  values: incidence of both low and high extremes increased during early hypoxia. This has been seen by others.<sup>3,4</sup> Some escape from this over-compensation to hyperoxia was evident by 60 min when  $\dot{V}O_2$ ,  $O_{2e.r.}$ , and tissue  $PO_2$  increased. A decrease in SVR and an increase in CI during return to normoxia indicate release of constriction of the resistance vessels whereas an increase in  $\dot{V}O_2$  and a decrease in  $PvO_2$  to baseline suggests improvement of blood flow at the distribution vessels. We conclude that in patients with  $PaO_2 > 100$  mmHg, ventilation with 100%  $O_2$  does not improve whole body oxygenation over a time span of 90 min. These findings do not imply, however, that brief preoxygenation prior to short periods of hypoventilation is useless or dangerous.

	Normoxia	Hyperoxia				Normoxia
Time (min)	0	30	60	90	120	
$PaO_2$ (mmHg)	113±25 *	398±95	395±101	386±103*	100±14	
$PvO_2$ (mmHg)	41±5 *	53±7	50±7	50±7 *	41±5	
$PO_2$ tissue (mmHg)	27±8	28±10	33±10	37±12	29±11	
CI ( $ml \cdot min^{-1} \cdot m^{-2}$ )	4.0±1.1	3.9±1.0	3.9±1.0	3.9±0.9*	4.4±0.7	
$\dot{D}O_2$ ( $ml \cdot min^{-1} \cdot m^{-2}$ )	573±165*	609±157	607±190	597±199	624±175	
$avDO_2$ (vol%)	4.2±1.1*	3.7±1.2	4.2±1.3	4.1±1.0	3.8±0.5	
$\dot{V}O_2$ ( $ml \cdot min^{-1} \cdot m^{-2}$ )	155±31 *	137±33	147±28	143±27 *	154±25	
$O_{2e.r.}$ ( $avDO_2/CaO_2$ )	.28±.07*	.23±.05	.26±.06	.26±.06	.26±.04	
SVR (dyne $\cdot cm^{-5} \cdot m^{-2}$ )	977±265	980±235	1017±195	1017±205*	843±145	

\*=p<0.05 between consecutive measurements  
all values are mean ± SD

**References**

1. Chapler CK, Cain SM, Stainsby, WN: The effects of hyperoxia on oxygen uptake during acute anemia. *Can J Physiol Pharm* 62:809-814, 1984
2. Lindbom L, Tuma RF, Arfors KE: Influence of oxygen on perfused capillary density and capillary red cell velocity in rabbit skeletal muscle. *Microvasc Res* 19:197-208, 1980
3. Lund N, Jorfeldt L, Lewis DH: Skeletal muscle oxygen pressure fields in healthy human volunteers. *Acta Anaesth Scand* 24:272-278, 1980
4. Kessler M, Hoper J, Krumme BA: Monitoring of tissue perfusion and cellular function. *Anesthesiology* 45:184-197, 1976