

Title: DOES PREOXYGENATION LOWER OXYGEN CONSUMPTION?
 Authors: R.D. Valley, M.D., W.J. Lucas, M.D. and E.A. Norfleet, M.D.
 Affiliation: Department of Anesthesiology, School of Medicine, University of North Carolina at Chapel Hill, Chapel Hill, North Carolina 27514

Introduction. Dynamic changes in $\dot{S}\bar{V}O_2$ occur during anesthesia as oxygen balance is altered by changes in both oxygen supply and demand. Preoxygenation and the induction of anesthesia are particularly important periods during which careful monitoring and its interpretation can be critical. Large changes in $\dot{S}\bar{V}O_2$ occur during this time. We studied these changes and the parameters that effect $\dot{S}\bar{V}O_2$ in patients scheduled for coronary artery bypass grafting (CABG).

Methods. Following informed consent, 10 patients about to undergo non-emergent CABG were studied. All patients were receiving calcium channel and Beta blocking drugs up until the day of operation. "In vivo" $\dot{S}\bar{V}O_2$ was measured continuously with a fiberoptic pulmonary artery thermodilution catheter (Oximetrix). Cardiovascular monitoring included arterial, pulmonary artery, central venous, and "wedge" pressures; cardiac outputs in triplicate; and an ECG. Arterial and mixed venous blood samples were obtained to measure blood gases, hemoglobin, and "in vitro" oxygen saturation (Co-oximeter). All measurements were made while breathing room air (PERIOD 1), after breathing at least 5 minutes of 100% O_2 (PERIOD 2), and following the induction of anesthesia with 50 μ g/kg of fentanyl and a metubine-pavulon mixture, intubation and mechanical ventilation with 100% O_2 (PERIOD 3). Arterial (CaO_2) and mixed venous ($C\bar{V}O_2$) oxygen contents were determined using standard formulas. Oxygen consumption ($\dot{V}O_2$) was calculated using the Fick principle. Paired Students t tests were used to analyze the data, a p value $<.05$ was considered significant. A correlation coefficient between in vivo (Oximetrix) and in vitro (Co-oximeter) $\dot{S}\bar{V}O_2$ measurements was also determined.

Results. (See table 1 and graph). CaO_2 increased significantly with the administration of 100% oxygen. $\dot{S}\bar{V}O_2$ and $C\bar{V}O_2$ increased significantly with oxygen administration and again with the induction of anesthesia. Cardiac Index (CI) decreased significantly with preoxygenation while SVR increased. PVR did not change. $\dot{V}O_2$ decreased significantly with oxygen administration and again with the induction of anesthesia. A correlation coefficient of 0.92 was found for the in vivo and in vitro $\dot{S}\bar{V}O_2$ measurements.

Discussion. Two consistent step-wise increments in $\dot{S}\bar{V}O_2$ were found, the first with preoxygenation (Period 2) and the second following induction of anesthesia (Period 3). The initial increase in $\dot{S}\bar{V}O_2$ was secondary to an increase in CaO_2 and a decrease in $\dot{V}O_2$, while the increase in $\dot{S}\bar{V}O_2$ from period 2 to period 3 was due to a further decrease in $\dot{V}O_2$ induced by anesthesia and muscle relaxation. The unexpected finding of a decrease in $\dot{V}O_2$ with preoxygenation appears to be a consistent and reproduceable phenomenon. The decrease in cardiac index with preoxygenation was

most likely in response to the observed increase in SVR in these B-blocked patients. Myocardial oxygen requirements as estimated by the rate-pressure product did not change, nor did the oxygen delivery index ($CI \times CaO_2$). Thus, it does not appear that the decrease in $\dot{V}O_2$ can be explained on the basis of a decrease in total systemic oxygen delivery or in myocardial work. To our knowledge, a decrease in $\dot{V}O_2$ in response to 100% oxygen administration has only been described once in the scientific literature (1). This fall in $\dot{V}O_2$ may represent an effect of hyperoxia on microvascular beds leading to a mismatch of supply to demand at the tissue level (i.e. "physiologic shunting"). This could limit oxygen consumption in affected tissues. Another possibility is a direct cellular response to hyperoxia leading to either a homeostatic down regulation of oxygen consumption or a toxic effect on the cellular respiratory pathway. The clinical implications are important not only in understanding changes in $\dot{S}\bar{V}O_2$ but also in assessing the potential benefits versus risks of oxygen administration in certain clinical settings.

References.

1. Becker L, et al.: Influence of FI_{O_2} on the Relationship between Oxygen Delivery and Uptake in the Dog. Abstract No. 2325. Federation Proceedings, 1985.

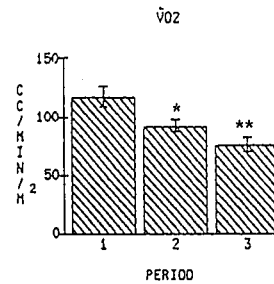


Table 1
(Results = Mean \pm SEM)

Period	n	CaO_2 (cc/100cc)	$S\bar{v}O_2$ (in vivo) (% Saturation)	$C\bar{v}O_2$ (cc/100 cc)	CI (L/min/m ²)	SVR (dynes-sec/cm ⁵)
1	10	17.1 \pm 0.9	69.0 \pm 1.7	12.5 \pm 0.8	2.67 \pm 0.24	1427 \pm 137
2	10	18.6 \pm 0.9*	80.0 \pm 1.4*	14.5 \pm 0.8*	2.39 \pm 0.24*	1652 \pm 187*
3	10	19.1 \pm 0.9*	88.3 \pm 1.0**	16.0 \pm 0.9**	2.56 \pm 0.19	1372 \pm 123**

* p $<.05$ - different from Period 1.
 ** p $<.05$ - different from Period 2.