## A234

AFFILIATION:

RELATIONSHIP BETWEEN OXYGEN DELIVERY (DO2) AND MIXED VENOUS OXYGEN SATURATION TITLE:

(SVO2) DURING SEPTIC SHOCK **AUTHORS:** 

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INTRODUCTION: The aim of this study is to evaluate if continuous monitoring of SvO2 during septic shock, where oxygen uptake (VO2) is dependent on DO2, remains a reliable predictor of delivery.

METHOD: Institutional approval and informed consent were obtained prior to the study. 62 measurements (n=62) were performed on 5 patients (age 49 $\pm$ 11 years; mean $\pm$ SD) during septic shock confirmed by hemodynamic and bacteriological parameters. They were all mechanically ventilated (FiO<sub>2</sub> = 0.3 to 0.6) and sedated with continuous infusion of fentanyl and midazolam. SvO<sub>2</sub> monitoring was insured by a fiberoptic pulmonary artery catheter (Opticath- Oxymetrix®. Abbott) and VO2 by a metabolic monitor (Deltatrac®, Datex), Blood oxygen saturation and haemoglobin concentration were measured by spectrophotometry (Corning 2500- CO-oxymeter) and cardiac output by thermodilution.

DO<sub>2</sub> variations were induced by catecholamine infusion, blood transfusion or fluid loading depending upon therapeutic needs. Measurements of DO2, VO2 and SvO2 were performed at 2-hourly intervals for 24 hours.

Statistical analysis was performed using linear regression (least squares method). p<0.05 was considered as significative.

RESULTS: A relationship exists between the change in DO2 and VO2 (fig. 1) (n=57, r=0.71, p<0.0001) and also between the change in DO2 and SvO2 (fig. 2) (n=57, r=0.61, p<0.0005). This relationship is stronger in the patient who survived (n=11, r=0.87, p<0.0005) than in the patients who died (n=46, r=0.36, p<0.05).

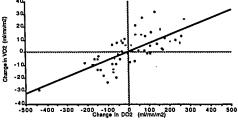
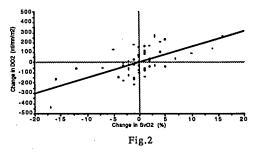


Fig. 1



DISCUSSION: As a relationship between DO2 and SvO2 persists during septic shock, oxygen extraction is still able to rise when DO2 decreases. This seems to be paradoxical in a situation of tissue oxygen debt confirmed by a dependence of VO2 on DO2. Septic shock is probably characterized by coexistence of hypoxic and non hypoxic tissues. The relationship DO2/VO2 refers to the hypoxic tissues whereas the relationship SvO2/DO2 is dependent on the non hypoxic areas. This could explain why the surviving patient in our study had a stronger relationship between DO2 and SvO2 than the patients who died.

## Reference

1. Crit Care Med 1: S70-S74, 1990

## A235

HYPOCAPNIA AND HYPERCAPNIA INCREASE VA/Q HETEROGENEITY IN TITLE:

CANINE OLEIC ACID LUNG INJURY.

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Hypocapnia may increase pulmonary shunting in anesthetized, obese patients(1). In contrast, increasing PaCO<sub>2</sub> by inspiration of CO<sub>2</sub> may improve arterial oxygenation in pneumonia(2). We studied the effects of changes in PaCO<sub>2</sub> on the matching of ventilation (VA) and perfusion (Q) in dogs with oleic acidinduced permeability edema, using the multiple inert gas elimination technique. We hypothesized that hypocapnia would impair and hypercapnia would enhance VA/Q matching. Methods: 8 pentobarbital-anesthetized, closed-chested dogs were administered 0.06 ml/kg of oleic acid at least 2 hours prior to study. They were ventilated with 90% O<sub>2</sub> at 35 breaths/min with a tidal volume of 20 ml/kg. Inspired CO<sub>2</sub> was added to change PaCO<sub>2</sub> to 25, 38, and 50 mmHg in a randomized order. A dilute solution of six inert gases (SF<sub>6</sub>, ethane, cyclopropane, halothane, diethyl ether, and acetone) was infused to assess gas exchange. Arterial and mixed venous blood gases and inert gases, mixed expired inert gases, and hemodynamics were measured. VA/Q mismatch (heterogeneity) was assessed by true shunt, dead space (VD/VT), log standard deviation of perfusion (log SDQ) and ventilation (log SDv) distributions and arterial-alveolar difference ([a-A]D) area. Data were analyzed by a two-factor within subjects analysis of variance and Duncan test. Results: QT, Ppa, true shunt, VD/VT and log SDO during hypocapnia and hypercapnia did not differ compared to normal PaCO<sub>2</sub> (see table). PaO<sub>2</sub> was lower with hypocapnia and higher with hypercapnia. Both hypocapnia and hypercapnia increased VA/Q heterogeneity, indicated by increases in log SD<sub>V</sub> and (a-A)D area compared to normal PaCO<sub>2</sub>.

## Table: Gas Exchange Measurements

	Normal PaCO2	Low PaCO2	High PaCO2
Mean Ppa(mmHg)	25 ± 1	24 ± 1	28 ± 2
Ċτ(l/min)	$4.1 \pm 0.4$	$3.8 \pm 0.3$	$4.2 \pm 0.4$
PaO <sub>2</sub> (mmHg)	221 ± 35	181 ± 33*	250 ± 36*
True shunt(%)	22 ± 3	23 ± 3	21 ± 3
VD/VT(%)	38 ± 4	37 ± 4	35 ± 4
Log SDo	$1.35 \pm 0.19$	$1.61 \pm 0.15$	$1.61 \pm 0.16$
Log SDŷ	$0.97 \pm 0.12$	$1.12 \pm 0.12*$	$1.13 \pm 0.10*$
(a-A)D area	$0.50 \pm 0.09$	$0.63 \pm 0.09$	$0.63 \pm 0.11*$

Means ± SE are presented. \*p < 0.05 versus normal PaCO<sub>2</sub>

Conclusions: Hypocapnia increased VA/Q mismatch and worsened arterial oxygenation in canine oleic acid-pulmonary edema. Inspiration of CO<sub>2</sub> to 50 mmHg also increased VA/Q heterogeneity, although PaO<sub>2</sub> was increased, due to the Bohr effect. The results suggest that hyperventilation to reduce PaCO<sub>2</sub> may increase arterial hypoxemia in patients with significant lung disease.

References: 1. Anesthesiology 63:A520, 1985; 2. Am Rev Respir Dis 137:A142, 1988. Supported by B.B. Sankey Anesthesia Advancement Award, NHLBI Clinical Investigator

Award (#HL02507), and HL12174.