

Title: PULMONARY SHUNTING WITH HYPERTHERMIA UNDER ANESTHESIA

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**Introduction:** Clinical signs of hypoxia are frequently encountered in patients with high fever, necessitating the administration of supplemental oxygen. Signs of arterial hypoxemia under anesthesia<sup>1</sup>, in spite of a high  $\text{FiO}_2$  and the addition of PEEP, have also been encountered during the anesthetic management of whole body hyperthermia for the treatment of metastatic cancer.<sup>2</sup> These clinical observations may be the reflection of increased oxygen consumption or increased pulmonary shunting with hyperthermia. To date this has not been investigated. The following study was therefore designed to determine the effect of hyperthermia on pulmonary shunting and oxygen consumption under anesthesia.

**Methods:** Twelve patients undergoing whole body hyperthermia were studied after obtaining informed consent to this institutionally approved protocol. Pretreatment pulmonary function tests were unremarkable, except for 4 patients with mild small airway disease. Prior to treatment a radial arterial line and Swan Ganz catheter were inserted percutaneously. Anesthesia was then established with a continuous infusion of thiopental ( $7 \pm 3$  mg/kg/hr) and fentanyl ( $3 \pm 1$  ug/kg/hr), allowing spontaneous ventilation on room air. Hyperthermia was induced by means of a high-flow, heated-water perfusion suit, which elevated the core temperature from 37 C to 41.5 C over a period of approximately 2 hours. At 37 C (after the induction of anesthesia, but before heating the patient) and at 41.5 C (under anesthesia), the following measurements and calculations were made:

Arterial and mixed venous blood gas tensions using an IL-713 blood gas analyzer, correcting for the effect of temperature using Severinghaus nomograms.

Oxygen saturation was measured directly by infrared absorption method.

Cardiac output was determined by the thermodilution method.

Hemoglobin concentration was determined by clinical hematology laboratory.

Shunt determinations were made while administering 100% oxygen for 15 minutes, using the formula:

$$\frac{Q_s}{Q_t} = \frac{C\bar{c}O_2 - CaO_2}{C\bar{c}O_2 - C\bar{v}O_2}$$

Oxygen consumption was calculated using the Fick equation:

$$\text{Cardiac Output} = \frac{\text{Oxygen Consumption}(\dot{V}O_2)}{CaO_2 - C\bar{v}O_2}$$

Data were analyzed using the Student's t test with significance defined as  $p < 0.05$ .

#### Results:

	37 C	41.5 C
PaO <sub>2</sub> (torr)	76.6±3.8	79.8±2.8
PaCO <sub>2</sub> (torr)	39.4±1.4	37.5±1.4
$\frac{Q_s}{Q_t}$ (%)	19.4±1.5	18.9±1.6
$\dot{V}O_2$ (ml/min)	282±20	450±43*

\*Significant at  $p < 0.05$  level.

**Discussion:** Our findings do not support the contention that pulmonary shunting increases with hyperthermia. Neither arterial oxygen tension on room air or shunt determinations on 100% oxygen were changed with hyperthermia. Shunt determinations on 100% oxygen primarily measure anatomical shunt, with the effect of ventilation-perfusion mismatches being evident only at lower inspired oxygen concentrations. While shunt determinations were not performed on room air, it is reasonable to assume that changes, if any, were so minimal as to be without effect on the arterial oxygen tension. The major difference between our anesthetic technique and that of previous investigators is our use of spontaneous ventilation. Mechanically controlled ventilation may both decrease cardiac output and aggravate already existent ventilation-perfusion mismatches. This effect coupled with the increased oxygen consumption that accompanies hyperthermia may be responsible for hypoxemia and clinical difficulties encountered by other investigators. There is no evidence that hyperthermia alone under conditions of spontaneous ventilation increases pulmonary shunting in anesthetized human subjects.

#### References:

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- Bull JM, Lees DE, Schuette W, et al.: Whole body hyperthermia: A phase I trial of a potential adjuvant to chemotherapy. *Ann Intern Med* 90:317-323, 1979