Changes in P_{O_2} with Time in Human Blood Containing Anesthetic Agents

*Norman A. Bergman, M.D.**

DURING ANALYSIS of blood for respiratory gas tensions, observed values must be corrected for metabolic changes which occur between the time of sampling and completion of measurement. Correction factors customarily applied in this situation have been derived from blood obtained from unanesthetized subjects or prepared by equilibration of blood against gases of known composition in a tonometer, in the absence of anesthetic agents.[1] Anesthetic agents in clinically-applicable concentrations cause significant decreases in rates of oxygen utilization in various cell and tissue preparations *in vitro*.[2, 3] We questioned whether anesthetic drugs decreased oxygen utilization in whole blood enough to cause systematic overestimation of P_{O_2} if existing correction factors were used. In the present study it is shown that the presence of anesthetics does not significantly change the rate of decrease of oxygen tension of blood *in vitro*, stored at room temperature.

Methods

Eighteen ml of fresh, heparinized human blood were divided into three equal parts and tonometered for 30 minutes at 37 C using a common gas source. In ten such control determinations the gas consisted of 5 per cent carbon dioxide in oxygen. In ten further experiments, the 5 per cent CO_2 in O_2 was passed through a Fluotec vaporizer set to deliver 4 per cent halothane (Fluothane) before passage into the tonometer flasks. Effluent gas from the tonometer was monitored for halothane concentration by means of an ultraviolet halothane analyzer. In ten additional determinations blood containing 50 μg/ml thiopental (Pentothal) was prepared by addition of 0.01 ml 0.5 per cent thiopental/ml blood to the samples with a micrometer syringe, before equilibration with 5 per cent CO_2 in O_2.

The instant mechanical agitation of the tonometer ceased was designated as time zero. The blood samples were drawn into syringes anaerobically and rotated at room temperature until they were analyzed. The blood was then analyzed for P_{O_2} at predetermined intervals following termination of equilibration, using an Instrumentation Laboratory oxygen electrode with 1 mil polypropylene membrane, maintained at 37 C. The electrode was calibrated with oxygen before and after each series of measurements and the reading corrected for instrumental drift. Sterile syringes were used for collection and storage of the blood. All tonometer parts that came into contact with blood or gas were sterilized in an autoclave before use, and the cuvette of the P_{O_2} electrode was kept filled with aqueous benzalkonium chloride (Zepharin) when not in use.

The rate of decrease in P_{O_2} was determined by constructing a regression line as a function of the interval from the end of equilibration, using the method of least squares for each of the three experimental conditions. The significances of differences in slopes of the regression lines were established by calculating standard errors of the estimate for each regression line.

Results and Discussion

In the absence of anesthetics, P_{O_2} in blood decreased at an average rate of 1.79 torr/min

* Associate Professor of Anesthesiology, Division of Anesthesiology, University of Utah College of Medicine, and Anesthesia Service, Veterans Administration Hospital, Salt Lake City, Utah.

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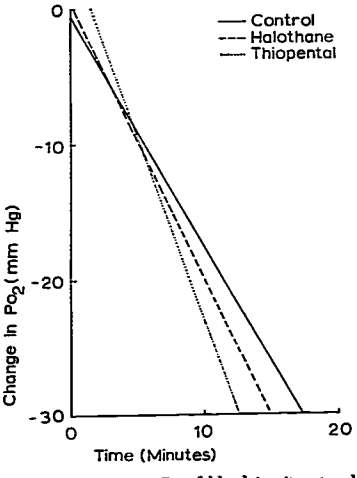

FIG. 1. Decrease in P_{O_2} of blood *in vitro* stored at room temperature following equilibration with approximately 95 per cent oxygen. Differences in slopes for blood containing no anesthetic, blood containing halothane, and blood containing thiopental are not significant.

(fig. 1). The corresponding value for blood equilibrated with 4 per cent halothane was 1.98 torr/min, and for blood containing 50 μg/ml thiopental, 2.75 torr. Scatter of individual points was large, and differences in rates of decline in P_{O_2} among the three experimental conditions were not significant.

The magnitude of decrease in P_{O_2} of blood *in vitro* owing to metabolic activity in the present study was in agreement with results of previous studies at comparable oxygen tensions.[4] Decrease in P_{O_2} of blood owing to metabolic activity is much less at lower P_{O_2} because of the shape of the oxyhemoglobin dissociation curve. Oxygen utilization in blood is attributable largely to metabolic activity of leukocytes and reticulocytes, varying with hematocrit, reticulocyte and leukocyte counts.[5]

Concentrations of anesthetics in the present study were quite high and rarely would be encountered in clinical practice. These agents did not significantly depress oxygen utilization by blood *in vitro* at high oxygen tensions. It is concluded, therefore, that correction factors for metabolic consumption of oxygen by blood *in vitro* need not be modified for use in blood containing anesthetics. The small increase in P_{CO_2} of blood *in vitro* (about 0.11 torr/min), is not the result of CO_2 production by blood elements,[6] and is unlikely to be influenced to any appreciable extent by anesthetics.

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