Transperitoneal Oxygenation with Lactated Ringer's Solution

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This experimental study was performed on 5 rabbits to ascertain if oxygenated Ringer's lactate could be used in place of fluorocarbons through peritoneal administration. Oxygen was bubbled through solutions of Ringer's lactate at two different rates and the oxygen tension of each solution was determined. The solution used in vivo had oxygen delivered at a rate of 5 L/min.; the mean PO₂ and pH were 575.5 mmHg and 6.34 respectively, while the rate of oxygenation of the in vitro solution was 3 L/min. with a mean PO₂ and pH of 416.6 mmHg and 6.08. After peritoneal administration of the oxygenated solution the PaO₂ values were significantly increased from the control value. Other parameters such as pH, PaCO₂, HCO₃⁻, BE, SO₂ (oxygen saturation), Na and K were not shown to be statistically significant. Some degree of oxygenation could be obtained by the introduction of oxygenated Ringer's solution. This result suggested that this solution can be used for oxygenation via the transperitoneal administration, and that this method of oxygenation may possibly be used to treat some forms of respiratory failure.

Key Words: Transperitoneal oxygenation, Ringer's lactate

Intravenous injection of oxygen (Cole 1951) and intraperitoneal oxygenation with hydrogen peroxide (Awad et al. 1970) and fluorocarbons (Faithfull et al. 1984; Faithfull et al. 1985; Klein et al. 1986) have been studied experimentally. The method of extracorporeal membrane oxygenation for the support of patients with respiratory failure was applied clinically and revealed no benefit over conventional ventilation in a controlled study (Hall and Kaplan 1983).

The peritoneum which provides a surface area approximately equal to that of the skin had been considered as a gas exchangeable organ and investigated as an alternative route of oxygenation not requiring the use of the lungs (Lukas et al. 1970; Klein et al. 1986). Transperitoneal administration of hydrogen peroxide resulted in the formation of emboli in the lungs and coronary arteries with a moderate increase in blood oxygen tension. On the other hand, the introduction of fluorocarbons into the peritoneum proved to be an effective way of supplying oxygen without embolization (Faithfull et al. 1984).

Lactated Ringer's solution is an easily obtainable isotonic saline. Its electrolyte concentration is similar to that of the blood and its value in the treatment of metabolic acidosis syndrome is well known (Goldberger 1986). It was important for us to ascertain that lactated Ringer's solution could be employed as the oxygenated fluid instead of fluorocarbons before proceeding with more sophisticated studies. To this end, our preliminary work is completed and is reported here.

MATERIALS AND METHODS

Oxygen was bubbled through the in vitro study solutions at a rate of 3 L/min., while those solutions used in the in vivo animal studies received oxygen at a rate of 5 L/min. The gas tensions of these solutions were measured.

Adult rabbits, ranging in weight from 1.8 to 2.6 Kg, were used. Anesthesia was induced with intramuscular ketamine (10 mg/Kg) and maintained by continuous infusion of ketamine as needed (500 mg in 500 ml of 5% dextrose water). Animals were allowed to breathe spontaneously at room-air (Clifford 1984).

The femoral artery was exposed to cannulate and a 22-gauge catheter for measuring the arterial blood gas tension and electrolytes was inserted. Heparin was administered to prevent catheter clotting. Another
16-gauge catheter was inserted in the right paracolic area to allow the infusion of the oxygenated fluid. A small incision was made on the left paramedian of the abdomen and a silicone T-tube which served as a drain for the solution pumped into the peritonium was placed in it.

Following oxygenation of Ringer’s lactate at a rate of 5 L/min., the temperature of the fluid was maintained at 37–38°C by passing it through a FloTem-II blood/Fluid warmer (Dala Chem, Inc., Carmel, Indiana, USA). The warm solution was then infused into rabbit’s peritoneum using a diginfusa (Schoch electronics AG, Zürich, Switzerland) at rate of 99 drops (6.6 ml) per minute. Excess oxygen from the reservoir of Ringer’s solution was vented via a needle stucked in glass tube (Fig. 1). The constant oxygen bubble in the solution was trapped within the transfusion set.

The rabbit’s temperatures was maintained at 37°C and monitored rectally using Tele-thermometer (Yellow Springs Instrument Co., Inc, Ohio, USA). The electrocardiogram was recorded from a standard lead.

During perfusion, arterial blood gas tension and electrolyte were measured every 15 minutes for 1 hour. Control values were also measured prior to the administration of oxygenated Ringer’s lactate.

Data were analyzed by the one-way analysis of variance (ANOVA) with repeated measures and the

![Fig. 1. Schematic presentation of experimental design.](image)

| Table 1. Comparison between the perfusate used in animals (5 L/min. oxygen supply) and the in vitro solution (3 L/min. oxygen supply) (Mean±S.D.) |
|----------------------------------------|----------------|----------------|----------------|----------------|----------------|
| pH                                     | PCO₂           | PO₂            | HCO₃⁻          | BE             |
| Perfusates used (n=5)                  | 6.34±0.43      | 8.0±1.41       | 575.5±32.67    | 0.5±1.0        | -46.75±11.96  |
| In vitro solutions (n=6)               | 6.08±0.48      | 7.8±1.3        | 416.6*±10.71   | 0.4±0.55       | -48.0±11.7    |

| Table 2. Blood gas tension and electrolyte in rabbits ventilated at 0.2 of FiO₂ before (control) and during intraperitoneal perfusion (n=5) (Mean±S.D.) |
|-----------------------------------------|----------------|----------------|----------------|----------------|----------------|
| Time of Perfusion                       | pH             | PaCO₂          | PaO₂           | HCO₃⁻          | BE             | SaO₂           | Na             | K              |
| Control                                 | 7.35±0.05      | 42.6±6.1       | 93.6±12.38     | -3.0±2.74      | 21.8±4.76      | 95.8±1.92      | 135.6±4.93    | 3.6±0.12       |
| 15 min.                                 | 7.38±0.06      | 40.4±5.77      | 106.2*±16.0    | -2.2±3.27      | 23.8±3.63      | 96.8±1.79      | 137.6±7.5     | 3.67±0.23      |
| 30 min.                                 | 7.38±0.05      | 41.2±5.06      | 106.6*±12.18   | -1.8±2.8       | 24.6±2.89      | 97.0±1.22      | 135.0±7.0     | 3.5±0.17       |
| 45 min.                                 | 7.42±0.05      | 41.6±6.62      | 110.8*±10.03   | -0.6±3.36      | 23.8±4.55      | 97.6±1.14      | 135.0±4.36    | 3.27±0.06      |
| 60 min.                                 | 7.39±0.06      | 41.0±4.83      | 108.5*±7.77    | -1.2±2.5       | 22.2±4.5      | 97.25±0.96    | 135.5±7.78    | 3.3±0.0        |

* Different from control (P<0.05)
PCO₂, PO₂ (mmHg)
HCO₃⁻, BE (mmol/L)
SaO₂, oxygen saturation (%)
Na, K (mEq/L)
unpaired Student's t-test where appropriate for the equality of mean values between each measure. Differences between specific values were tested with the Scheffe's test for multiple comparisons (Auh 1984; Norusis 1986). Probability values of less than 0.05 were considered statistically significant. All data are presented as Mean±S.D. (standard deviation).

RESULTS

The animals in this experiment remained normothermic ranges throughout the procedure; also, arrhythmia was not observed. The mean PO₃ of the oxygenated perfusate used in animal study was 575.5±32.67 mmHg and that of the in vitro solution was 416.6±10.71 mmHg, while the pH was 6.34±0.43 and 6.08±0.48, respectively (Table 1).

Table 2 shows the result of PaO₂ prior to and during peritoneal perfusion. As can be seen, PaO₂ values throughout the experimental period were significantly higher than the control value. Changes from the control value of 93.6±12.38 mmHg to 106.2±16.0, 106.6±12.18, 110.8±10.03 and 108.5±7.77 mmHg were recorded at 15, 30, 45, and 60 minutes.

However, no significant changes were found among the PaO₂ values of the perfusing period.

The results shown in Table 2 which are similar to the those of the control value are seen in the PaCO₂, BE and pH of the arterial blood. Also no significant changes were seen with each of the electrolytes studied.

DISCUSSION

The two solutions examined were somewhat different in PO₂ due to the amount of oxygen received per minute. A rate of 5 L per minute of oxygen was administered to the perfusion fluid employed in the animal study, and a rate of 3 L per minute in the experimental in vitro solution. Both fluids had similar pH's, 6.3 for the animal perfusate and 6.1 for the experimental solution. The solution employed in vivo was more acidic than the fluorocarbon emulsions (Moore et al. 1985; Klein et al. 1986). This might lead to a lower rate of oxygen uptake as it has been shown that a decrease in portal blood pH causes a decrease in portal blood flow. Oxygen uptake from the portal circulation mainly relies upon the difference between the oxygen tension of the perfused solution and the peritoneum (Gelman and Ernst 1977). Under normotensive conditions, the latter value is known to be about 42±1 mmHg (Klossner et al. 1974). The difference in oxygen tension in this study was 534 mmHg (576-42 mmHg).

Full saturation of portal blood by the intraperitoneal perfusate would deliver: 0.28×11.9×1.39=4.6 ml of oxygen per minute. 0.28 is the saturable ratio from 72% to 100% of hemoglobin (10-72) at the intraperitoneal oxygen tension of 42 mmHg (Altman and Dittmer 1970). The mean hemoglobin concentration of the rabbit is 11.9g/100ml, and 1.39ml/g is the oxygen-combining power (Albritton 1952; Faithfull et al. 1984). This amount of oxygen uptake is insufficient when compared to the requirement of 9.3ml/kg/min. But at hypoxic conditions, the difference (e.g. 534 mmHg) might be wider and somewhat more oxygen can be taken up into the portal circulation. Faithfull et al. (1985) demonstrated the changes in PaO₂ following intraperitoneal administration of Fluosol. At 0.2 of FiO₂ changes in PaO₂ against the control were 15.6±mmHg. These results concur with ours. On the other hand, the PaCO₂ decreased by 3 or more mmHg in cases where fluorocarbons were used, but changes were not observed in our experiment. It was suggested that the intraperitoneal perfusate might decrease the PaCO₂ if the diffusion gradient were high enough. The reason that the PaCO₂ was not decreased in this study is still unknown to us in spite of the broad disparity of diffusion gradients.

The lower viscosity of the Ringer's lactate as compared to that of the fluorocarbons might be more advantageous to oxygenation due to the free movement of the oxygenated perfusion across the peritoneum and between folds of the intestine in the abdominal cavity.

It is concluded that some degree of oxygenation can be obtained by peritoneal perfusion with oxygenated Ringer's lactate as with fluorocarbons but without the decrease in tension of the arterial carbon dioxide. Also, it could be argued that a more alkaline perusate be employed in a future study, as it might provide a significant movement of oxygen uptake to the portal venous blood through the peritoneum. The result of this preliminary trial have shown the possibility of employing peritoneal oxygenation in the treatment of some kinds of respiratory failure (Smith and Hanning 1986) in place of the others such as an extracorporeal membrane oxygenator.

REFERENCES

Transperitoneal Oxygenation with Lactated Ringer's Solution


