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Supplemental Oxygen and Carbon Dioxide Each Increase Subcutaneous and Intestinal Intramural Oxygenation

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Abstract

Oxidative killing by neutrophils, a primary defense against surgical pathogens, is directly related to tissue oxygenation. We tested the hypothesis that supplemental inspired oxygen or mild hypercapnia (end-tidal PCO_2 of 50 mmHg) improves intestinal oxygenation. Pigs (25±2.5 kg) were used in two studies in random order: 1) Oxygen Study — 30% *vs*. 100% inspired oxygen concentration at an end-tidal PCO_2 of 40 mmHg, and 2) Carbon Dioxide Study — end-tidal PCO_2 of 30 mmHg *vs*. 50 mmHg with 30% oxygen. Within each study, treatment order was randomized. Treatments were maintained for 1.5 hours; measurements were averaged over the final hour. A tonometer inserted in the subcutaneous tissue of the left upper foreleg measured subcutaneous oxygen tension. Tonometers inserted into the intestinal wall measured intestinal intramural oxygen tension from the small and large intestines. 100% oxygen administration doubled subcutaneous oxygen partial pressure (PO_2) (57±10 to 107±48 mmHg, P=0.006) and large intestine intramural PO_2 (53±14 to 118±72 mmHg, P=0.014); intramural PO_2increased 40% in the small intestine (37±10 to 52±25 mmHg, P=0.004). An end-tidal PCO_2 of 50 mmHg increased large intestinal PO_2 approximately 16% (49±10 to 57±12 mmHg, P=0.039), while intramural PO_2 increased by 45% in the small intestine (31±12 to 44±16 mmHg, P=0.002). Supplemental oxygen and mild hypercapnia each increased subcutaneous and intramural tissue PO_2, with supplemental oxygen being most effective.

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Implications: Tissue oxygenation is the primary determinant of oxidative killing rate by neutrophils. Increasing inspired oxygen concentration from 30% to 100% or increasing end-tidal PCO_2 from 30 mmHg to 50 mmHg increased both subcutaneous or intestinal intramural tissue oxygenation, with supplemental oxygen being most effective. Either treatment is thus likely to reduce the risk of infection.

Keywords

Oxygen: Intramural, Subcutaneous Intestinal; Ventilation: Increased Oxygen, Carbon Dioxide

Introduction

The primary defense against surgical pathogens is oxidative killing by neutrophils (1). Oxidative killing of bacteria depends on tissue oxygenation throughout the physiologic range (2-4). It is thus not surprising that infection risk is inversely related to subcutaneous tissue partial pressure of oxygen (5). Factors that improve subcutaneous tissue oxygenation reduce infection risk whether oxygenation is improved directly by providing supplemental inspired oxygen (6) or indirectly by maintaining perioperative normothermia, which increases tissue perfusion (7). In contrast, factors such as smoking (8) that reduce subcutaneous tissue oxygenation increase risk (7,9).

Inadequate gastrointestinal oxygenation is also associated with gut dysfunction, particularly a loss of barrier function. Failure of the intestinal barrier leads to systemic endotoxin absorption and bacterial translocation, which is clinically manifested as shock and sepsis (10). Hypoxic bowel also releases mediators that can injure distant organs, contributing to multiple-organ failure (11). As might thus be expected, insufficient intestinal oxygenation increases mortality (10).

One of the easiest methods of improving subcutaneous oxygenation is simply providing supplemental inspired oxygen (6). Another is to permit mild hypercapnia which improves cutaneous perfusion and, thus, oxygenation (12,13) However, the effects of supplemental oxygen or mild hypercapnia on intestinal intramural oxygenation remain unknown. We therefore tested the hypothesis that supplemental inspired oxygen or mild hypercapnia improves intestinal oxygenation.

Methods

Following approval from the Washington University Animal Studies Committee, we studied 11 healthy female domestic pigs. Pigs were chosen because they are omnivores and the intestinal physiology of the pig closely approximates that of humans. They were 3 ± 1 months old and weighed 25 ± 2.5 kg. All animals were fasted for 12-16 hours and given 8.5 mg magnesium citrate solution orally the night before the study; a standard mechanical bowel preparation using an electrolyte solution was administered the morning of the study.

A peripheral intravenous catheter was inserted for administration of fluids and medications. Lactated Ringer's solution was given IV as a 10-ml/kg bolus, followed by an infusion at a rate of 7 $ml \cdot kg^{-1} \cdot h^{-1}$; fluids were warmed to 37°C. The pigs were sedated with intramuscular Telazol (2 mg/kg), ketamine (1 mg/kg) and xylazine (1 mg/kg). Anesthesia was induced by inhalation of isoflurane and maintained with isoflurane (1.5-2.0%) in 30% oxygen and 70% nitrogen. All pigs had endotracheal intubation, and their lungs were mechanically ventilated at 12 breaths per min with a tidal volume of ≈8 ml/kg. The animals were actively warmed with surface warming to maintain normothermia. A femoral arterial catheter was inserted for direct blood pressure monitoring; a pulmonary artery catheter was inserted *via* the femoral vein. A urinary catheter was placed as well.

After induction of anesthesia, silastic tonometers were inserted into the lateral left upper arm for measurement of subcutaneous tissue oxygenation and temperature. Each tonometer consisted of a 15 cm tube filled with hypoxic saline; 10 cm of the tubing was tunneled subcutaneously. A Clark-type oxygen sensor and thermistor (Licox, Gesellschaft für

Medizinische Sondensysteme, GmBH, Kiel, Germany) were inserted into the subcutaneous part of the tonometer as previously described (14).

In vitro accuracy of the optodes (in a water bath at 37°C) is ± 3 mmHg for the range from 0-100 mmHg, and ± 5% for the range 100-360 mmHg. Temperature sensitivity is 0.25%/°C, but thermistors are incorporated into the probes and temperature-compensation is included in the $PsqO_2$ calculations. Optode calibration remains stable (within 8% of baseline value for room air) *in vivo* for at least 8 hours. Optodes (oxygen sensors) were calibrated in room air (ambient pO_2 154 mmHg). For calibration purposes a calibration card was inserted into the Licox device. The calibration data of the connected optode and other data are electronically stored on this card (factory calibration setting). All $PsqO_2$ values measured prior to insertion were within 10% of 154 mmHg.

In order to exclude a significant drift of the optode (>10%), probes were again exposed to room air after each investigation. No significant drift was observed throughout the entire study. At least 30 minutes were allowed for electrode equilibration. Values were subsequently recorded at 15 minutes intervals.

To measure intramural intestinal oxygen partial pressure ($PimO_2$), the abdomen was opened and similar intramural probes were inserted through 20-g cannulae into the small intestine and colon. The probes were inserted into the tissue plane between the serosa and mucosa. Great care was taken to minimize handling of the intestine and to return the bowel to a neutral position. Intestinal retractors were not used.

Each of the pigs was used in a study evaluating inspired oxygen concentration (Oxygen Study) and a separate study evaluating end-tidal carbon dioxide partial pressure (Carbon Dioxide Study). The order in which the protocols were performed was randomly assigned, and each protocol contained its own internal randomization. Each randomization was based on computer-generated codes maintained in sequentially numbered opaque envelopes until just before use.

In the oxygen study, the two study treatments were inspired oxygen concentrations of 30% or 100%. In each case, the concentration designated by the randomization was maintained for 30 minutes to establish steady-state conditions. Subsequently, $PsqO_2$ and $PimO_2$ were recorded for one hour (treatment period). Measurements were then repeated during the alternative oxygen concentration, and again 30 minutes was allowed to elapse to establish steady-state conditions before measurements were taken. End-tidal PCO_2 was kept at 40 mmHg throughout the oxygen study.

In the carbon dioxide study, the two study treatments were an end-tidal PCO_2 of 30 and 50 mmHg. To achieve an end-tidal PCO_2 of 50 mmHg, we removed the soda lime and slightly decreased the respiratory rate. In each case, the end-tidal concentration designated by the randomization was maintained for 30 minutes to establish steady-state conditions. Subsequently, $PsqO_2$ and $PimO_2$ were recorded for one hour (treatment period). Measurements were then repeated at the alternative end-tidal PCO_2. The inspired oxygen concentration was kept at 30% throughout the carbon dioxide study.

Additional measurements in both studies included hemodynamic and respiratory values. Arterial blood for gas analysis was obtained at the beginning of each one-hour-long treatment period. Cardiac output was determined by thermodilution at the beginning and end of each treatment period. All other measurements, including tissue oxygenation, were recorded at 5-minute intervals throughout each treatment period.

All values obtained during each treatment period were averaged in each animal. Our primary comparisons were between the two treatments in each study (*i.e.*, 30% *vs.* 100% oxygen and 30 mmHg *vs.* 50 mmHg end-tidal PCO_2). Data were compared with two-tailed, paired *t* tests. We similarly compared the effects of supplemental oxygen and changes in end-tidal PCO_2 on subcutaneous and intestinal oxygenation. Data are presented as means ± SDs; $P < 0.05$ was considered statistically significant.

Results

Bowel at the tonometer insertion sites appeared entirely normal at the end of the studies and was not edematous, nor were any hematomas observed. Arterial and end-tidal PCO_2 values were similar. Initial hemoglobin concentration was 10.1 ± 1.4 g/dl, and the concentration did not change significantly during the study.

Heart rate, blood pressure, pulmonary arterial pressure, cardiac output, and systemic and pulmonary vascular resistances did not differ significantly during 30% and 100% inspired oxygen. The pH, PCO_2, base excess, and HCO_3 were similar during the 30 and 100% oxygen treatments. As might be expected, arterial PO_2 was 108 ± 21 mmHg with 30% inspired oxygen and 454 ± 53 mmHg with 100% oxygen.

Subcutaneous tissue oxygenation increased by 50 ± 41 mmHg during 100% oxygen ($P = 0.006$). Supplemental oxygen increased small intestine oxygen partial pressure by 15 ± 11 mmHg ($P = 0.004$). Supplemental oxygen increased large intestine intramural PO_2 considerably more, by 65 ± 62 mmHg ($P = 0.014$) (Table 1).

Systolic and diastolic pulmonary artery pressures, cardiac index, and stroke volume all increased significantly when end-tidal $PCO_2$was at 50 mmHg, whereas systemic vascular resistance decreased (Table 2).

Arterial blood pH, PO_2, and PCO_2 differed significantly during 30 and 50 mmHg end-tidal PCO_2. Increasing end-tidal PCO_2 from 30 to 50 mmHg increased subcutaneous PO_2 by 11 ± 8 mmHg (23%) ($P = 0.003$); small intestinal PO_2 by 13 ± 9 mmHg (45%) ($P = 0.002$); and large intestinal PO_2 by 8 ± 10 mmHg (16%) ($P = 0.039$) (Table 3).

Discussion

Supplemental inspired oxygen increases subcutaneous oxygen partial pressure in surgical patients (15). Our study extends this observation by showing that supplemental oxygen also improves intestinal intramural oxygenation. This was especially the case in the large intestine where supplemental inspired oxygen doubled intestinal partial pressure. Our study similarly extended previous studies by showing that increasing end-tidal PCO_2 from 30 to 50 mmHg not only improves cutaneous oxygenation, but also improves intestinal oxygenation. Although the increase in subcutaneous oxygenation was slightly less in our swine than previously observed in volunteers (12) subjected to an end-tidal PCO_2 of 50 mmHg, the increase was still of a magnitude that is considered clinically important (5).

When the pigs were subjected to an end-tidal PCO_2 of 50 mmHg, hemodynamic changes occurred including significant increases in pulmonary arterial pressure, cardiac output, and stroke volume. However, systemic vascular resistance decreased, presumably because of peripheral vascular dilation. Heart rate and blood pressure thus remained essentially unchanged.

Interestingly, increasing end-tidal PCO_2 to 50 mmHg similarly increased intramural oxygenation in both the small and large intestines, whereas supplemental oxygen was

considerably more effective in the large intestine. Overall, supplemental oxygen was far more effective than increasing end-tidal PCO_2 from 30 to 50 mmHg. It also had the advantage of being easier to implement. While it is likely that the combining supplemental oxygen and mild hypercapnia would further improve tissue oxygen partial pressure, we did not specifically test this theory.

In contrast to previous studies, oxygen tension in the large intestine was significantly greater than in the small intestine during all conditions (16,17). Although oxygenation of both the small- and large-intestines are of interest, anastomotic leaks are more common in the large intestine (18) and large-intestinal surgery entails a far greater infection risk. It was thus encouraging to observe that tissue oxygenation in the large intestine and skin were remarkably similar under each study circumstance. In contrast, tissue oxygenation in the small intestine was consistently less than subcutaneous oxygen tension, usually by about 40%. Nevertheless, it needs to be noted that our experiments were performed on healthy, normovolemic animals. Oxygenation in the large intestines and the subcutaneous tissue might be different under certain pathologic conditions such as for example bowel surgery, hypovolemia or shock.

Microelectrodes were inserted directly into the tissue plane separating the mucosal and serosal surfaces of the intestines to avoid tissue damage and interference with the microcirculation. In this respect, our technique differed from previous studies that evaluated intestinal oxygenation with Clark-type multiwire electrodes placed onto the serosal or mucosal surfaces (19-21) or with serosal and mucosal surface tonometers, which were introduced through needle enterostomies (22). Our concern about these methods is that measurements can be confounded by air or fecal contamination, or by poor contact between the electrode and the tissue surface. Our baseline values for intestinal oxygen partial pressure were nonetheless similar to those reported previously (16). Although we did not observe edema or hematomas at the insertion sites, some tissue damage is inevitable. In this respect, insertion of the electrode served as a surrogate wound, much as subcutaneous measurements in the arm deliberately mimic surgical incisions.

An obvious limitation of our study is that we evaluated swine rather than humans. The gut in pigs, however, approximates the human intestinal system. Consistent with this theory, subcutaneous oxygenation with 30% oxygen was similar to that observed in humans, as was the effect of supplemental oxygen (15). Our study was restricted to minimally disturbed intestine; the effects of supplemental oxygen and end-tidal PCO_2 on intramural oxygenation may differ after intestinal manipulation and, especially, after intestinal anastomosis.

Another limitation was that during the CO_2 study, the PaO_2 was significantly less during an end-tidal of PCO_2 of 50 than during 30 mmHg. This could explain why the increase in tissue oxygenation with a PCO_2 of 50 mmHg was slightly less than observed previously in humans (12).

In summary, tissue oxygenation in the large intestine and skin were similar, whereas tissue oxygenation in the small intestine was consistently less than subcutaneous oxygen tension, usually by about 40%. Administration of 100% oxygen nearly doubled subcutaneous and large intestinal intramural oxygenation, while intramural oxygenation increased 40% in the small intestine. Changing the end-tidal PCO_2 from 30 to 50 mmHg increased tissue oxygenation approximately 20% in the subcutaneous tissue and in the large intestine, while intramural oxygenation increased by 45% in the small intestine. Supplemental oxygen and mild hypercapnia thus each increased subcutaneous and gut tissue PO_2, with supplemental oxygen being most effective.

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Table 1

Blood Gas Results and Tissue Oxygenation during the Oxygen Study.

	30% Oxygen	100% oxygen	P
PH	7.45 ± 0.06	7.45 ± 0.03	0.05
PO_2 (mmHg)	108 ± 21	454 ± 53	<0.001
PCO_2 (mmHg)	42 ± 3	43 ± 6	0.64
HCO_3^- (mEq)	29.7 ± 3.4	25.2 ± 9.8	0.17
Base Excess (mEq)	6.4 ± 2.5	6.0 ± 2.0	0.27
Core Temperature (°C)	37.1 ± 0.4	37.2 ± 0.6	0.67
Leg			
$PsqO_2$ (mmHg)	57 ± 10	107 ± 48	0.006
Temperature (°C)	36.4 ± 1.4	36.5 ± 1.7	0.64
Small Intestine			
$PimO_2$ (mmHg)	37 ± 10	52 ± 25	0.004
Temperature (°C)	37.9 ± 1.1	37.9 ± 1.4	0.92
Large Intestine			
$PimO_2$ (mmHg)	53 ± 14	118 ± 72	0.014
Temperature (°C)	37.8 ± 1.3	38.1 ± 1.4	0.24

$PsqO_2$ = partial pressure of subcutaneous oxygen tension. $PimO_2$ = partial pressure of oxygen in intestinal tissue. End-tidal PCO_2 was 40 mmHg. Results presented as means ± SDs.

Table 2

Hemodynamic Responses during the Carbon Dioxide Study.

	30 mmHg $EtPCO_2$	50 mmHg $EtPCO_2$	P
Heart Rate (beats per min)	110 ± 18	118 ± 19	0.27
Arterial Pressure (mmHg)			
Systolic	82 ± 10	82 ± 8	0.71
Diastolic	51 ± 15	47 ± 12	0.27
Mean	63 ± 13	61 ± 10	0.439
Pulmonary Artery Pressure (mmHg)			
Systolic	21 ± 13	25 ± 5	0.005
Diastolic	13 ± 3	16 ± 4	0.002
Mean	17 ± 3	10 ± 4	0.002
Wedge Pressure (mmHg)	8 ± 2	8 ± 3	0.75
Central Venous Pressure (mmHg)	7 ± 2	6 ± 3	0.29
Cardiac Output (L/min)	2.7 ± 0.6	3.7 ± 0.6	0.001
Cardiac Index ($l/min/m^2$)	2.9 ± 0.6	3.9 ± 0.6	<0.001
Stroke Volume (ml)	25.8 ± 5.5	31.5 ± 6.8	0.004
Systemic Vascular Resistance (Dynes)	1612 ± 329	1167 ± 195	<0.001
Pulmonary Vascular Resistance (Dynes)	276 ± 82	292 ± 109	0.63

The fraction of administered oxygen was 30%. Results presented as means ± SDs.

Table 3

Blood Gas Results and Tissue Oxygenation during the Carbon Dioxide Study.

	30 mmHg $EtPCO_2$	50 mmHg $EtPCO_2$	P
PH	7.56 ± 0.05	7.39 ± 0.04	0.001
PO_2 (mmHg)	121 ± 23	101 ± 13	0.03
PCO_2 (mmHg)	30 ± 3	53 ± 5	<0.001
HCO_3^- (mEq)	28.1 ± 1.9	32.0 ± 2.9	0.002
Base Excess (mEq)	5.0 ± 3.0	7.3 ± 2.9	0.06
Core Body Temperature (°C)	37.2 ± 0.5	36.9 ± 0.6	0.54
Leg			
$PsqO_2$ (mmHg)	47 ± 12	58 ± 13	0.003
Temperature (°C)	36.0± 1.1	36.2 ± 2.0	0.74
Small Intestine			
$PimO_2$ (mmHg)	31 ± 12	44 ± 16	0.002
Temperature (°C)	37.4 ± 1.1	3.7 ± 1.5	0.54
Large Intestine			
$PimO_2$ (mmHg)	49 ± 10	57 ± 12	0.039
Temperature (°C)	37.5 ± 1.0	37.4 ± 1.0	0.94

$PsqO_2$ = partial pressure of subcutaneous oxygen tension. $PimO_2$ = partial pressure of oxygen in intestinal tissue. The fraction of administered oxygen was 30%. Results presented as means ± SDs.