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RESEARCH ARTICLE

Subcutaneous Oxygen Pressure in Spontaneously Breathing Lean and Obese Volunteers: a Pilot Study

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Abstract
Background Oxidative killing is the primary defense against surgical pathogens; risk of infection is inversely related to tissue oxygenation. Subcutaneous tissue oxygenation in obese patients is significantly less than in lean patients during general anesthesia. However, it remains unknown whether reduced intraoperative tissue oxygenation in obese patients results from obesity per se or from a combination of anesthesia and surgery. In a pilot study, we tested the hypothesis that tissue oxygenation is reduced in spontaneously breathing, unanesthetized obese volunteers.
Methods Seven lean volunteers with a body mass index (BMI) of 22±2 kg/m^2 were compared to seven volunteers with a BMI of 46±4 kg/m^2. Volunteers were subjected to the following oxygen challenges: (1) room air; (2) 2 l/min oxygen via nasal prongs, (3) 6 l/min oxygen through a rebreathing face mask; (4) oxygen as needed to achieve an arterial oxygen pressure (arterial pO_2) of 200 mmHg; and (5) oxygen as needed to achieve an arterial pO_2 of 300 mmHg. The oxygen challenges were randomized. Arterial pO_2 was measured with a continuous intraarterial blood gas analyzer (Paratrend 7); deltoid subcutaneous tissue oxygenation was measured with a polarographic microoxygen sensor (Licox).
Results Subcutaneous tissue oxygenation was similar in lean and obese volunteers: (1) room air, 52±10 vs 58±8 mmHg; (2) 2 l/min, 77±25 vs 79±24 mmHg; (3) 6 l/min, 125±43 vs 121±25 mmHg; (4) arterial pO_2=200 mmHg, 115±42 vs 144±23 mmHg; (5) arterial pO_2=300 mmHg, 145±41 vs 154±32 mmHg.
Conclusion In this pilot study, we could not identify significant differences in deltoid subcutaneous tissue oxygen pressure between lean and morbidly obese volunteers.

Keywords Subcutaneous oxygen pressure · Spontaneous breathing · Obesity · Tissue oxygenation · Volunteers

Introduction

All surgical wounds become contaminated to some degree. Host defense is the primary factor determining whether contamination progresses to clinical infection. Oxidative killing by neutrophils is the primary defense against surgical pathogens [1–4]. It is thus unsurprising that the risk of infection is inversely related to subcutaneous tissue oxygen partial pressure [2]. Furthermore, interventions—such as maintaining normothermia [5] and providing supplemental oxygen [6, 7]—that improve perioperative tissue oxygenation reduce the risk of surgical wound infection.

Obesity is a major risk factor for surgical site infection and contributes to a high morbidity and mortality in the obese population [8]. Subcutaneous tissue oxygenation is lower in obese than lean patients having surgery with general anesthesia and mechanical ventilation [9]. Furthermore, supplemental oxygen only slightly increases tissue oxygenation whereas it doubles tissue oxygenation in lean patients

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[7]. There are several reasons why tissue oxygenation might be reduced in the obese. For example, total blood flow is disproportionately low [10]. Furthermore, obesity augments the size of individual fat cells without increasing blood flow [11]. Fat tissue is thus relatively hypoperfused [12] and, therefore, likely to be poorly oxygenated.

However, it is also notable that the effects of mechanical ventilation, such as formation of atelectasis, decrease in functional residual capacity, are far more pronounced in obese than lean patients [13, 14]. It thus remains unknown whether reduced intraoperative tissue oxygenation in the obese results from obesity per se or from a combination of anesthesia and mechanical ventilation. We therefore tested the hypothesis that tissue oxygenation is reduced per se in spontaneously breathing, unanesthetized obese volunteers compared to lean volunteers. Furthermore, we evaluated the speed of increase of tissue oxygen pressure under various oxygen conditions. As no data on tissue oxygenation is available in the unanesthetized obese, we conducted a pilot study to assess the size of possible differences in tissue oxygenation.

Materials and Methods

With approval of the Human Studies Committee of Washington University in St. Louis and written informed consent, we studied seven morbidly obese volunteers with a body mass index (BMI) exceeding 35 kg/m^2 and eight lean volunteers with a BMI between 20 and 25 kg/m^2. One lean volunteer was subsequently excluded from the study because of technical problems with the tissue oxygen monitor. The volunteers were generally healthy nonsmokers who ranged in age from 27 to 44 years.

Protocol

All studies started between 8:00 and 9:00 A.M. to avoid the potential confounding effects of circadian rhythms. Volunteers were allowed to eat and to drink water. All volunteers were comfortably seated in a relax chair in a half-sitting position. The studies were conducted in a regular operating room at a room temperature of 22–24°C.

Volunteers were subjected to the following oxygen challenges: (1) room air; (2) 2 l/min oxygen via nasal prongs, (3) 6 l/min oxygen through a rebreathing face mask; (4) oxygen as needed through a rebreathing face mask to achieve an arterial oxygen pressure (arterial pO_2) of 200 mmHg; and (5) oxygen as needed through a rebreathing face mask to achieve an arterial pO_2 of 300 mmHg.

The oxygen challenges were randomized and each condition was maintained until tissue oxygenation (described below) reached a steady state. Tissue oxygen partial pressures were then measured for an additional 10 min before the oxygen challenge was discontinued. Between each oxygen challenge, the volunteers breathed room air until tissue oxygenation returned to stable values near baseline (approximately 35 min). We considered tissue oxygen pressures to be stable when values changed less than 4 mmHg over a 10-min period.

Measurements

After local anesthesia of the skin with 1% lidocaine, a silastic tonometer was inserted into the lateral left upper arm for measurement of subcutaneous tissue oxygen pressure and temperature. The silastic tonometer consisted of 15 cm of tubing filled with hypoxic saline; 10 cm of the tubing was tunneled subcutaneously. A polarographic oxygen sensor and thermistor (Licox, Integra Life Science, CA, USA) were inserted into the subcutaneous portion of the tonometer as previously described [15]. Subcutaneous tissue oxygen pressure ($PsqO_2$) and temperature were acquired on line with a multichannel interface (Biopac MP 150, Biopac, Goleta, CA, USA), which was connected to a portable computer.

Oxygen sensor calibration remains stable (within 8% of baseline value for room air) in vivo for at least 8 h. The electrodes are individually factory-calibrated, but calibration was confirmed by exposing the electrode to room air (ambient oxygen pressure of 154 mmHg); in all cases, measurements in air were within 10% of 154 mmHg. To exclude a significant drift of the oxygen sensor, probes were again exposed to room air after each study day; none differed by more than 10% from their baseline value.

Inspired and end-tidal gas concentrations were sampled from a thin soft plastic tube that was inserted through each volunteer's nostril into the oropharynx and then connected to a standard anesthesia gas analyzer (Capnomac Ultima, Datex-Ohmeda, Helsinki, Finland). Measurement accuracy as provided by the manufacturer is 2% for oxygen and the sampling volume was 200 ml/min.

After local anesthesia, a 20-g cannula was inserted into the left radial artery. Arterial oxygen pressure (arterial pO_2) was measured continuously with a Paratrend 7 monitor (Diametrics Medical, Buckinghamshire, UK). The design, mechanics, and operation of the Paratrend 7 system have been presented elsewhere [16]. Before insertion, the sensor requires computer-controlled calibration performed by diffusing precision gases of fixed concentration into a tonometer solution for 30 min. Trials of the device have demonstrated a high level of accuracy [17, 18]. All values are reported after being normalized to 37°C.

Initial placement of the Paratrend 7 sensor was performed by the principal investigator using sterile technique. The Paratrend 7 sensor was inserted for a minimum distance of 4 cm beyond the arterial catheter tip. This exceeded the

minimum distance necessary to avoid flush contamination of the blood gas sensing elements.

Tissue oxygen data were acquired online with a sampling rate of 10 Hz via a multichannel interface (MP 150; Biopac Systems, Goleta, CA, USA) with acquisition software (Acqknowledge 3.9. Biopac Systems, Goleta, CA, USA) to a portable computer. Blood gas parameters were continuously displayed and recorded. At the end of the experiment, data were exported for statistical analysis.

Core temperature was measured at the tympanic membrane using Mon-a-therm thermocouples (Tyco-Mallinckrodt Anesthesiology Product, St. Louis, MO, USA). The aural canal was occluded with cotton and the thermocouple taped in place.

The lean and obese volunteers were compared with an unpaired Student's *t* test using a commercially available software (Instat 3 for Mac, GraphPad, San Diego, CA, USA). Changes within a group were described using a repeated-measures, two-tailed ANOVA followed by a Tukey–Kramer post hoc test for multiple comparisons. Data are presented as the means±SDs; $P<0.05$ was considered statistically significant. A Pearson's correlation coefficient between the parameters arterial pO_2 and $PsqO_2$ for both groups was estimated and the hypothesis of their nullity tested. In addition, a linear regression model between arterial pO_2 and $PsqO_2$ was fitted and the nullity of the slope tested. Correlation coefficients among the two groups were compared to investigate possible differences between lean and obese patients. The same has been done with the slopes of the regression lines.

Table 1 Demographic data

	Lean	Morbidly obese	*P* value
BMI (kg/m^2)	22±2	46±4	<0.001
Age (years)	31±6	37±6	0.09
Gender (male/female)	2/5	0/7	
Heart rate (at room air; bpm)	65±5	77±11	0.03
Mean arterial pressure (mmHg)	85±11	91±6	0.29
Tympanic temperature (°C)	35.8±0.6	36.2±0.3	0.16
Hematocrit (%)	38±2	39±2	0.76
PaO_2 (FiO_2=0.21; mmHg at 37°C)	81.4±6	78.6±8	0.51
$EtCO_2$ (mmHg at 37°C)	39±2	41±3	0.18
$PaCO_2$ (mmHg)	39.5±2	40.8±3	0.85
Respiration rate (breaths/min)	13±1	17±2	0.02
Glucose (at room air; mmol/l)	5.6±1.8	6.0±1.2	0.71

Data presented as the mean and SD. Lean and obese volunteers demographic and baseline data. All measurements were performed at room air. *P* values were calculated with the Student's *t* test.
PaO_2: arterial oxygen pressure normalized to 37°C, *$EtCO_2$*: end-tidal carbon dioxide, *$PaCO_2$*: arterial carbon dioxide pressure normalized to 37°C

Results

Per protocol, BMI was significantly greater in the obese volunteers (22±2 vs 46±4 kg/m^2). In the lean group, two male and five female volunteers were studied, whereas in the obese groups, only female subjects were studied. All volunteers were of comparable age. Baseline arterial pressure, core temperature, hematocrit, plasma glucose, and arterial pO_2 and arterial carbon dioxide pressure (arterial pCO_2) were similar in lean and obese volunteers (Table 1) and did not change significantly throughout the study.

Table 2 Oxygen data

	Lean	Obese	*P* value
Room air			
PaO_2 (37°C)	81±6	78±10	0.11
$PsqO_2$ (mmHg)	52±10	58±8	0.57
Local temperature (°C)	34.6±1.6	34.9±1.3	0.65
Heart rate (bpm)	66±6	78±12	0.03
MAP (mmHg)	85±11	91±8	0.29
2 l of oxygen (nasal prongs)			
FiO_2 (%)	32±3	31±2	0.39
PaO_2 (37°C)	128±17	114±18	0.16
$PsqO_2$ (mmHg)	77±25	79±24	0.97
Local temperature (°C)	34.8±1.5	35.3±1.1	0.49
Heart rate (bpm)	65±6	78±11	0.01
MAP (mmHg)	85±11	90±8	0.31
6 l of oxygen (mask)			
FiO_2 (%)	59±2	56±3	0.06
PaO_2 (37°C)	251±40	193±42	0.02
$PsqO_2$ (mmHg)	125±43	121±25	0.62
Local temperature (°C)	34.6±1.4	34.4±1.5	0.81
Heart rate (bpm)	61±5	74±10	0.01
MAP (mmHg)	84±11	90±8	0.28
Target PaO_2=200 mmHg			
FiO_2 (%)	58±4	61±7	0.32
PaO_2 (37°C)	217±14	216±11	0.97
$PsqO_2$ (mmHg)	115±42	144±23	0.19
Local temperature (°C)	34.7±1.6	34.9±1.3	0.77
Heart rate (bpm)	66±7	72±9	0.12
MAP (mmHg)	83±10	89±8	0.23
Target PaO_2=300 mmHg			
FiO_2 (%)	75±7	75±5	0.96
PaO_2 (37°C)	314±20	318±9	0.63
$PsqO_2$ (mmHg)	145±41	154±33	0.76
Local temperature (°C)	34.4±2.0	34.9±1.8	0.94
Heart rate (bpm)	63±5	71±12	0.12
MAP (mmHg)	84±11	90±9	0.28

Local temperature is the temperature measured in the tonometer tube at the tissue oxygen pressure measurement site. *P* values resulting from the comparison between the two groups using a two-tailed Student's *t* test.
FiO_2: inspired oxygen fraction, *PaO_2*: arterial oxygen pressure, *$PsqO_2$*: tissue oxygen pressure

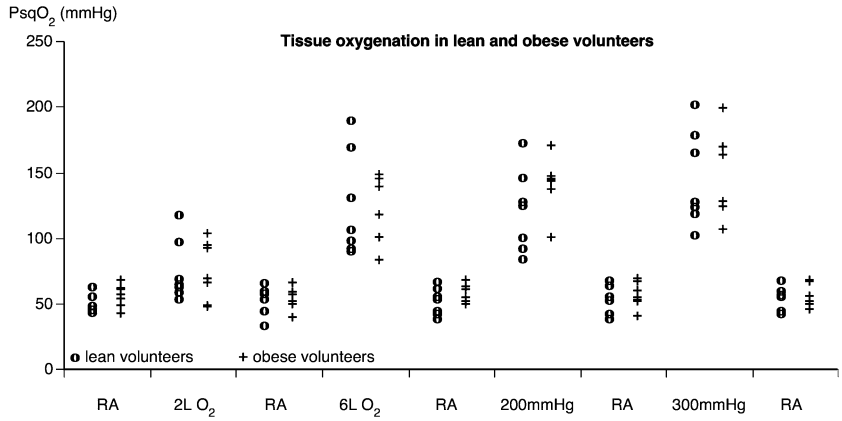

Fig. 1 Individual tissue oxygen pressure values during different oxygen conditions. Oxygen challenges were randomized. Between each oxygen challenge volunteers were breathing room air. Both lean (*open circles*) and obese (*cross*) had comparable subcutaneous tissue oxygen pressures. *RA* room air, *2L O_2* 2 l of oxygen given with nasal prongs, *6L O_2* 6 l of oxygen given with a rebreather face mask, *200mmHg* oxygen given to achieve an arterial oxygen pressure of 200 mmHg, *300mmHg* oxygen given to achieve an arterial oxygen pressure of 300 mmHg

Tissue and arterial oxygen partial pressures values during the initial and subsequent room air measurement periods were virtually identical; these values were thus averaged. Subcutaneous tissue oxygenation did not differ significantly in the lean and obese volunteers under any of the tested conditions: (1) room air, 52±10 vs 58±8 mmHg; (2) 2 l/min, 77±25 vs 79±24 mmHg; (3) 6 l/min, 125±43 vs 121±25 mmHg; (4) arterial pO_2=200 mmHg, 115±42 vs 144±23 mmHg; (5) arterial pO_2=300 mmHg, 145±41 vs 154±32 mmHg. Baseline tissue oxygen pressure between the different oxygen conditions were similar in both groups and all baselines were comparable (Table 2; Fig. 1)

Under most conditions, heart rate was significantly greater by about 10 bpm in the obese volunteers. Mean arterial pressure was typically 5 mmHg greater in the obese volunteers; however, this difference was not statistically significant (Table 2).

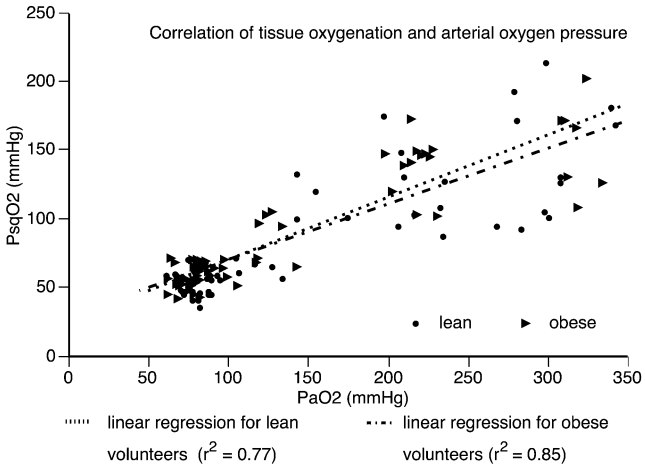

Fig. 2 Subcutaneous tissue oxygen pressure in correlation with arterial oxygen pressure. Both lean (*circles*) and obese (*triangles*) showed a linear correlation. *PaO_2* arterial oxygen pressure, *$PsqO_2$* subcutaneous tissue oxygenation above the deltoid muscle

The amount of oxygen required to achieve arterial oxygen targets were slightly greater in the obese than the lean volunteers. For example, the lean volunteers required 4.0±1.4 l/min to reach an arterial oxygen pressure of 200 mmHg whereas the obese volunteers needed 6.1±2 l/min. Similarly, the lean volunteers required 7.4±2.2 l/min to reach an arterial oxygen pressure of 300 mmHg whereas the obese volunteers needed 9.3±2.1 l/min. However, neither difference was statistically significant. There was a linear relationship between arterial and subcutaneous oxygen pressure in both lean (r^2=0.77) and obese (r^2=0.85) volunteers; the equation for each regression was: $PsqO_2 \approx 0.4$ arterial pO_2+33 mmHg. The comparison of the slopes in each group did not show a significant difference (P>0.5) (Fig. 2).

Subcutaneous tissue oxygen pressure increased at a comparable speed in the lean and obese patients in all conditions: 2 l/min, 1.0±0.55 vs 1.2±0.8 mmHg/min; 6 l/min, 1.4±0.5 vs 1.7±0.8 mmHg/min; 200 mmHg, 2.6±1.5 vs 2.3±0.8 mmHg/min; 300 mmHg, 2.9±2.1 vs 2.6±1.3 mmHg/min.

Discussion

We studied generally healthy lean and obese volunteers to determine the effects of obesity and supplemental oxygen on subcutaneous tissue oxygenation ($PsqO_2$) in the absence of anesthesia, mechanical ventilation, and surgery. Our results are notable for the similarities between the groups: baseline tissue oxygenation was similar in the lean and obese patients, and the similarity persisted at each tested inspired oxygen concentration and arterial oxygen pressure.

Similar tissue oxygenation in lean and obese volunteers is in distinct contrast to a previous study by Kabon et al. [9].

Subcutaneous oxygen partial pressure in that study was significantly less in obese patients compared to lean patients; and in the obese surgical patients, supplemental oxygen only slightly increased intraoperative tissue oxygenation. Furthermore, the obese patients needed a greater inspired oxygen concentration to reach an arterial oxygen pressure of ≈150 and 300 mmHg. Results were similar in a subsequent study by Fleischmann et al.: Subcutaneous tissue oxygen pressure at an arterial pO_2 of 150 mmHg was significantly less in the obese patients: 42±11 vs 57±15 mmHg [19]. Previous work thus suggests that more oxygen is required in obese surgical patients to produce a given arterial oxygen partial pressure, and that at any given arterial pO_2, subcutaneous tissue oxygenation is less in the obese.

Divergent results might be explained because our current study was conducted in generally healthy volunteers whereas the others were conducted in surgical patients. There are at least five potential reasons why tissue oxygenation may be disproportionately reduced in obese surgical patients: (1) concomitant illnesses; (2) surgical stress; (3) general anesthesia; (4) mechanical ventilation; and (5) fluid management. Concomitant illnesses were reasonably well controlled in our previous studies and do not appear to be the explanation. The effects of surgical stress and general anesthesia are probably different in lean and obese patients. Furthermore, mechanical ventilation and fluid management most likely contributed to the different results in the previous study.

In both previous studies that compared tissue oxygenation in lean and obese patients [9, 19], we maintained a positive end-expiratory pressure (PEEP) of 5 cm H_2O. Contemporaneous work suggests that PEEP of 10 cm H_2O is superior to 5 cm H_2O in morbidly obese patients [13, 14, 20]. Arterial oxygenation may thus have been improved in our obese patients had we provided more PEEP. However, PEEP alone would not explain the observation in both studies that tissue oxygenation was reduced in obese patients at a given arterial oxygen pressure. Another notable difference of the present study compared to the work by Kabon et al. is that subcutaneous tissue oxygen temperature was similar in the lean and obese. It is known that both core and local temperature significantly affect perfusion and thus tissue oxygen pressure [21]. The lack of a difference in peripheral tissue temperature could at least in part explain why no difference in tissue oxygenation was found. It further points out that differences in volume status and/or environmental temperature management might have occurred in the previous studies.

Thus, the most likely explanation for the differences between the present and previous studies appears to be fluid management. It is well established that tissue oxygenation is exquisitely sensitive to intravascular volume and vasomotor tone. For example, subcutaneous tissue oxygenation can be critically reduced in patients who maintain normal heart rate, blood pressure, and urine output [15, 22]. (In contrast, isovolemic hemodilution does not reduce $PsqO_2$ [15]). It remains unknown how best to hydrate obese surgical patients. Many anesthesiologists fear that fluid replacement strategies based on milliliters per kilogram body weight are likely to result in fluid overloaded obese patients. Therefore, most anesthesiologists give obese patients considerably less fluid, often basing fluid administration on patients' lean body mass instead of actual mass. This is the approach we used in our previous studies; the lean and obese patients were thus given essentially equal volumes. It is thus probable that the obese patients were relatively hypovolemic. The extent to which possible hypovolemia in the obese surgical patients contributed to reduced tissue oxygenation remains unknown, but could be substantial. However, as fluid management in the obese was not the purpose of this study, this remains a speculation. Perioperative fluid management in the obese during surgery needs to be addressed in a further study.

An additional difference is that our current volunteers, whereas morbidly obese at a BMI averaging ≈46 kg/m^2, were not as obese as the patients in our previous studies who averaged 52 kg/m^2. Physiologically, there is a substantial increment in morbidity when BMI increases from ≈46 to ≈52 kg/m^2. Furthermore, obese patients undergoing surgery are likely to have comorbidities, which affect tissue oxygenation such as diabetes and cardiovascular impairments [23–25].

Supplemental oxygen substantially improved tissue oxygenation: in both lean and obese volunteers, the relationship was linear, had an intercept of ≈33 mmHg, and a slope of ≈0.4. Each 5 mmHg increase in PaO_2, thus increased tissue oxygenation 2 mmHg over the entire tested range.

Despite the linear relationship, we noted that the increase in tissue oxygenation with increasing arterial oxygenation becomes slightly less at higher arterial pO_2 levels. In both groups, the administration of 6 l of oxygen resulted in subcutaneous tissue oxygen levels around 120 mmHg. Such tissue oxygenations have been shown to halve the rate of wound infections in patients undergoing colon surgery. This is of considerable clinical importance, specifically for postoperative oxygen administration. It is likely that not only intraoperative supplemental oxygen administration decreases the incidence of postoperative wound infections, but that oxygen administration in the first 24 to 48 h improves outcome especially in morbidly obese patients. Our data suggests that the administration of 6–8 l of oxygen, which is fairly well tolerated by most patients, might be sufficient in spontaneously breathing subjects.

Supplemental oxygen administration is only one of many perioperative factors influencing subcutaneous tissue oxygen partial pressure. For example, it is well established

that hypothermia [21], surgical and postoperative pain [26, 27], and smoking [28] all reduce tissue oxygen tension. In contrast, administration of supplemental fluid [29], hypercapnia [30], and epidural anesthesia [27, 31] increase subcutaneous tissue oxygenation. From a clinical perspective, obesity is thus but one of many factors influencing tissue oxygenation and thus, presumably, wound infection risk. By studying spontaneously breathing volunteers, we specifically addressed the factor obesity and thus excluded the abovementioned anesthesia and surgery-related confounders. Subcutaneous tissue oxygenation increased at a comparable speed in the lean and obese patients in all conditions. This is interesting and shows that tissue perfusion in the obese patients is probably not much different to lean patients. This is in contrast to the common opinion that obesity augments the size of individual fat cells without increasing blood flow [11]. Fat tissue is thus believed to be relatively hypoperfused [12] and, therefore, likely to be poorly oxygenated. If perfusion is insufficient, even supplemental oxygen fails to increase tissue oxygenation in hypoperfused tissues. However, in our volunteers, perfusion in lean and obese subjects was similar.

Subcutaneous oxygen, measured from experimental wounds on the shoulder, is the standard method to evaluate tissue oxygenation. We have shown that it correlates well with tissue oxygenation adjacent to abdominal surgical incisions, within surgical incisions, and even in the wall of small and large intestines in lean patients. Measurements of subcutaneous oxygenation in the deltoid region have been used in various studies in the obese [9, 19, 32], and at least in the postoperative period have shown a good correlation to tissue oxygenation at the site of interest, the surgical wound. It is conceivable that the positioning of the volunteers and patients, respectively, influences tissue oxygenation. By tightly controlling arterial pO_2, we aimed to minimize possible effects on tissue oxygenation caused by the positioning.

The main limitation of our study is the relatively small number of volunteers studied. In a power analysis performed before the study, a clinically significant difference in tissue oxygen pressure of 15 mmHg between lean and obese volunteers with a standard deviation of at least 15 mmHg was assumed. To detect this difference in a *t* test with 80% power, 17 volunteers would have been necessary in each group. Because of the fact that the intravital blood gas sensor became suddenly unavailable, the pilot study had to be stopped after seven volunteers in each group. A post hoc power analysis with the standard deviation from our volunteers at baseline showed a 73% power to detect a clinically significant difference of 15 mmHg in tissue oxygen pressure at room air. Higher concentration of inspired oxygen resulted not only in increased tissue oxygenation but also considerably increased SD. The study was designed for maximal control of arterial oxygen pressure yet SD of tissue oxygenation in both study groups was high—indicating that factors other than arterial oxygen pressure have considerable influence on tissue oxygenation. A sample size calculation based on our data reveals that at an arterial oxygen pressure of 300 mmHg, 113 volunteers in each group would be needed to provide 80% power to detect a significant difference in tissue oxygen pressure of 15 mmHg between lean and obese volunteers.

In summary, deltoid subcutaneous tissue oxygenation did not differ significantly in lean and obese volunteers. This is in contrast to previous observations in lean and obese surgical patients who were given general anesthesia and mechanically ventilated. Consequently, factors other than obesity alone apparently contribute to the previously described decrease in tissue oxygen pressure during general anesthesia, mechanical ventilation, and surgery.

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