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Obesity Decreases Perioperative Tissue Oxygenation

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Abstract

Background: Obesity is an important risk factor for surgical site infections. The incidence of surgical wound infections is directly related to tissue perfusion and oxygenation. Fat tissue mass expands without a concomitant increase in blood flow per cell, which might result in a relative hypoperfusion with decreased tissue oxygenation. Consequently, we tested the hypotheses that perioperative tissue oxygen tension is reduced in obese surgical patients. Furthermore, we compared the effect of supplemental oxygen administration on tissue oxygenation in obese and non-obese patients.

Methods: Forty-six patients undergoing major abdominal surgery were assigned to one of two groups according to their body mass index (BMI): BMI < 30 kg/m² (non-obese) and BMI ≥ 30 kg/m² (obese). Intraoperative oxygen administration was adjusted to arterial oxygen tensions of ≈150 mmHg and ≈300 mmHg in random order. Anesthesia technique and perioperative fluid management were standardized. Subcutaneous tissue oxygen tension was measured with a polarographic electrode positioned within a subcutaneous tonometer in the lateral upper arm during surgery, in the recovery room, and on the first postoperative day. Postoperative tissue oxygen was also measured adjacent to the wound. Data were compared with unpaired two tailed *t*-tests and Wilcoxon rank-sum tests; *P* < 0.05 was considered statistically significant.

Results: Intraoperative subcutaneous tissue oxygen tension was significantly less in the obese patients at baseline (36 vs. 57 mmHg, *P* = 0.002) and with supplemental oxygen administration (47

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vs. 76 mmHg, $P = 0.014$). Immediate postoperative tissue oxygen tension was also significantly less in subcutaneous tissue of the upper arm (43 vs. 54 mmHg, $P = 0.011$) as well as near the incision (42 vs. 62 mmHg, $P = 0.012$) in obese patients. In contrast, tissue oxygen tension was comparable in each group on the first postoperative morning.

Conclusion: Wound and tissue hypoxia were common in obese patients in the perioperative period and most pronounced during surgery. Even with supplemental oxygen tissue, oxygen tension in obese patients was reduced to levels that are associated with a substantial increase in infection risk.

Summary Statement: Wound and tissue hypoxia were both common in obese patients in the perioperative period and most pronounced during surgery. Supplemental oxygen only slightly increased tissue oxygenation in obese patients.

Introduction

Oxidative killing by neutrophils is the primary defense against surgical pathogens¹ and the risk of infection is thus inversely related to tissue oxygen partial pressure.^{2–4} Tissue oxygenation is especially important in the hours immediately following bacterial contamination when infections are established; this time is known as the decisive period.⁵ Factors that reduce tissue oxygenation thus augment infection risk.⁶ For example, hypothermia and smoking — each of which reduce subcutaneous oxygen tension^{7,8} — increase the incidence of infection.^{9–11} Obesity is also a major risk factor for surgical site infection and contributes to a high morbidity and mortality in the obese population.^{12,13}

Cardiac output, circulating blood volume, and resting oxygen consumption are all increased in obese persons;¹⁴ however, total blood flow is sub-normal in relation to body weight.¹⁵ Obesity augments the size of individual fat cells without increasing blood flow.¹⁶ Fat tissue is thus relatively hypoperfused¹⁷ and, therefore, likely to be poorly oxygenated. Even supplemental oxygen fails to increase tissue oxygenation in hypoperfused tissues.¹⁸ We thus tested the hypothesis that subcutaneous tissue oxygenation is inadequate in obese surgical patients and only minimally improved by administration of supplemental oxygen.

Methods

With approval from the Institutional Review Board at Washington University in St. Louis and written informed consent, we studied 46 patients aged 18–60 years undergoing elective major abdominal surgery involving a midline abdominal incision. Exclusion criteria included documented coronary or peripheral artery disease, insulin-dependent diabetes mellitus, recent history of smoking, and any symptoms of infection or sepsis. We also excluded patients with preoperative systolic arterial blood pressure > 170 mmHg or diastolic arterial blood pressure > 90 mmHg.

Protocol

Based on their calculated body mass index (BMI, weight/height²), patients were assigned in advance to two groups: BMI < 30 kg/m² (non-obese) or ≥ 30 kg/m² (obese).^{19,20} Every single patient was evaluated at two different intraoperative arterial oxygen partial pressures: approximately 150 mmHg and approximately 300 mmHg. Treatment order was randomly assigned, based on computer-generated codes that were maintained in sequentially numbered opaque envelopes. Inspiratory oxygen fraction was adjusted in all patients to reach target partial pressures, which were maintained for one hour.

General anesthesia was induced with sodium thiopental (3–5 mg/kg) and fentanyl (1–3 µg/kg). Vecuronium (0.1 mg/kg) or succinylcholine (0.8–1 mg/kg) was given to facilitate endotracheal intubation. Ventilation was mechanically controlled to maintain arterial carbon dioxide tension

at approximately 40 mmHg. Positive end-expiratory pressure was maintained at 5 mmHg, and peak ventilatory pressure was kept less than 30 mmHg. Subsequently, anesthesia was maintained with sevoflurane (1–2%) in oxygen and air. Sevoflurane administration was adjusted to maintain mean arterial blood pressure within 20% of the preinduction value. A supplemental bolus dose of fentanyl (100 µg) was given when heart rate or arterial pressure exceeded 120% of the baseline value. No vasoactive drugs were given.

Following induction of anesthesia, a 20-g cannula was inserted into a radial artery. In all patients, normal body weight in kg was calculated according to an average normal BMI of 22.5 kg/m². A fluid bolus of 10 mL/kg normal body weight was given before induction of anesthesia. Subsequently, patients were given a maintenance dose of 10 mL/kg of estimated normal body weight each hour. Additionally, blood loss was replaced with crystalloid at a 4:1 ratio or colloid at a 2:1 ratio; supplemental fluid was given as necessary to maintain urine output greater than 1 mL/kg of estimated normal body weight per hour. Allogenic blood was administered as necessary to maintain a hematocrit greater than 26%. Lower-body forced air warming was used to keep patients normothermic.

At the end of surgery, wounds were dressed with standard surgical bandages that did not apply direct pressure to the wound. Anesthesia was discontinued, and the patients' tracheas were extubated. Oxygen was delivered *via* a facemask at a rate sufficient to maintain a PaO₂ of about 120 mmHg.

Postoperative pain was treated with intravenous morphine *via* a patient-controlled analgesia system. Supplemental morphine was given as necessary by nurses who were not involved in the study and were not informed of the study purpose. In the postoperative anesthesia care unit, crystalloid was administered at a rate of 3.5 mL/kg of estimated normal body weight per hour. Additional fluid was given as necessary to maintain a urine output of at least 1 mL/kg of estimated normal body weight per hour and a mean arterial blood pressure within 20% of baseline value. Fluid management, supplemental oxygen administration, and analgesia on the ward were managed by the surgical team.

Measurements

Demographic data, American Society of Anesthesiologists (ASA) Physical Status, preoperative laboratory values, and type and duration of surgery were recorded. All routine anesthetic, respiratory, and hemodynamic variables were also recorded. Detailed records of fluid management, including urine output, were kept. Inspired oxygen, end-tidal sevoflurane, and carbon dioxide concentrations were measured during anesthesia. Oxygen saturation was measured with pulse oximeters during anesthesia and postoperative measurement periods. Intraoperative core temperature was measured in the distal esophagus (Mon-a-therm, Tyco-Mallinckrodt Anesthesiology Products, St. Louis, MO). Postoperative core temperatures were measured with tympanic membrane probes (Mon-a-therm).

Arterial pressure was monitored continuously from the arterial catheter during surgery and in the postanesthesia care unit; it was measured non-invasively on the first postoperative morning. As the primary determinants of tissue oxygen availability are arterial oxygen tension, cardiac output, and local perfusion, cardiac output was measured non-invasively using the partial rebreathing Fick method (NICO, Novamatrix Medical System Inc, Wallingford, CT). Arterial blood gases were obtained intraoperatively and in the post anesthesia care unit as necessary to maintain target arterial oxygen pressure.

After induction of anesthesia, a silastic tonometer was inserted into the lateral left upper arm for measurement of subcutaneous tissue oxygenation and temperature. At the end of surgery, a second tonometer was inserted 2–3 cm lateral and parallel to the surgical incision. Each

tonometer consisted of 15 cm of tubing filled with hypoxic saline; 10 cm of the tubing was tunneled subcutaneously. A Clark-type oxygen sensor and thermistor (Licor, Gesellschaft für Medizinische Sondensysteme, GmbH, Kiel, Germany) were inserted into the subcutaneous portion of both tonometers, as previously described.²¹

In vitro accuracy of the oxygen sensors is ± 3 mmHg for the range from 0 to 100 mmHg, and $\pm 5\%$ for 100 to 360 mmHg (in a water bath at 37°C). Temperature sensitivity is 0.25%/°C, and thermistors are incorporated into the probes and temperature compensation is included in the tissue oxygen tension (PsqO₂) calculations. Oxygen sensor calibration remains stable (within 8% of baseline value for room air) *in vivo* for at least 8 hours. The electrodes are individually factory-calibrated, but calibration was confirmed by exposing the electrode to room air (ambient PO₂ 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 investigation; none differed by more than 10% from baseline values.

PsqO₂ was recorded in 10-minute intervals during a 30-minute measurement period following a 30-minute equilibration period after reaching target PaO₂ values. Intraoperative PsqO₂ was recorded from the upper arm at an arterial oxygen partial pressure of 150 and 300 mmHg. Postoperative PsqO₂ was recorded from the upper arm and adjacent to the surgical incision. Again an equilibration period of 30 minutes was allowed before measurements were performed for 30 more minutes. On the first postoperative morning, baseline values were obtained from each site with the patient breathing room air or sufficient supplemental oxygen to maintain arterial saturation of at least 96%. Subsequently, measurements were repeated during oxygen challenge, which consisted of oxygen given at a rate of 10 L/min into a non-rebreathing facemask that provided an inspired oxygen fraction near 50%.

Data Analysis

Routine anesthetic measurements were recorded at ten-minute intervals. Values were first averaged within each patient over each treatment condition (*e.g.*, measurement period); these values were subsequently averaged among the patients in each group. Routine postoperative values were evaluated similarly. Tissue oxygenation and related values were recorded at 10-minute intervals during each measurement period. Measurements were first averaged within each patient over the relevant period; these values were subsequently averaged among the patients receiving each treatment.

Potential confounding factors and outcomes of the study were analyzed with unpaired, two-sided t-tests when the data were normally distributed. These results are presented as mean $\pm$ standard deviation. Data that were not normally distributed were compared using Wilcoxon rank-sum tests. These results are reported as median [25th percentile, 75th percentile]. For paired comparisons either a two-sided, paired t-test or a Wilcoxon signed-rank test was used. Once again, the test used depended upon the distribution of the data values. $P < 0.05$ was considered statistically significant.

Results

Age and height were similar in the two weight groups; there was a significant difference in weight (70 ± 14 kg vs. 144 ± 45 kg), BMI (24 ± 4 kg/m² vs. 51 ± 15 kg/m²) and ASA classification. There were also more women in the obese group than in the non-obese group. Duration of surgery was significantly longer in the obese patients (Table 1). Nearly all the obese patients had gastric bypass surgery; among the 23 others, 17 patients were undergoing colo-rectal surgery and 6 had gastrectomies.

Intraoperative Period

Anesthetic management, fluid replacement, urine output, blood loss, and core temperatures were similar at each oxygen concentration (Table 2). Hemodynamic values were also comparable.

To achieve an arterial oxygen tension of ≈ 150 mmHg, the obese patients required an FiO_2 of $51 \pm 18\%$ while the non-obese patients only needed $36 \pm 5\%$ ($P < 0.001$). The obese patients also needed a greater inspired oxygen concentration to reach an arterial oxygen tension of ≈ 300 mmHg: $95 \pm 5\%$ vs. $82 \pm 11\%$ ($P < 0.001$). Arterial oxygen (PaO_2) and carbon dioxide (PaCO_2) partial pressures were comparable in each weight group within each designated oxygen treatment.

Subcutaneous oxygen partial pressure (PsqO_2) was significantly less in the obese patients at an arterial oxygen tension near 150 mmHg: 36 vs. 57 mmHg, $P = 0.002$. PsqO_2 was also significantly less in the obese patients at an arterial oxygen tension near 300 mmHg: 47 vs. 76 mmHg, $P = 0.014$. Thus, supplemental oxygen increased PsqO_2 by 24 ± 34 mmHg in the non-obese patients, but by only 13 ± 18 mmHg in the obese patients $P = 0.18$, (Table 3, Figs. 1, 2 and 3).

Postanesthesia Care Unit

Two obese patients could not be studied in the postoperative period because they required postoperative mechanical ventilation. We therefore evaluated 21 obese and 23 non-obese patients in the postanesthesia care unit. Mean arterial blood pressure, core temperature, fluid management, urinary output, and VAS pain scores were comparable in the groups (Table 4).

PsqO_2 in the arm (43 vs. 54 mmHg, $P = 0.01$) and adjacent to the wound (42 vs. 62 mmHg, $P = 0.01$) was significantly less in obese than in the non-obese patients. Within each weight group, however, tissue oxygen tensions measured at the arm and wound were virtually identical (Table 5).

First postoperative day

In two non-obese patients measurements could not be performed, because they withdrew from the study. We thus evaluated 21 obese and 21 non-obese patients on the first postoperative morning. Mean arterial pressure and pain were similar in the two groups; however, urine output was significantly less and core temperature was greater in obese patients (Table 6).

Subcutaneous oxygen partial pressures in the arm as well as adjacent to the wound were similar in the obese and non-obese patients at baseline and during oxygen challenge. Supplemental oxygen significantly increased tissue oxygenation, by 14.4 [2.5, 26.9] mmHg in obese patients and by 21.4 [14.4, 29.8] mmHg in the non-obese patients, $P = 0.21$. Within each weight group, PsqO_2 was virtually identical at the arm and wound (Table 7).

Discussion

Administration of 50% inspired oxygen to the obese patients, a typical concentration, produced a PaO_2 of 150 mmHg and an oxygen saturation of 99%. Intraoperative and immediate postoperative subcutaneous tissue oxygenation was nonetheless critically low (approximately 40 mmHg), a value associated with a high risk of infection. In contrast, tissue oxygenation in the non-obese patients was roughly 20 mmHg greater, a value which is considerably less likely to be associated with infection.² It is thus apparent that subcutaneous tissue is often hypoxic in obese patients undergoing routine anesthetic management. Although we did not evaluate the incidence of infection in this relatively small study, the link between subcutaneous

oxygenation and wound infection risk is well established. Inadequate subcutaneous oxygenation is likely to account for the observed increased number of postoperative infections in obese patients.¹²

An inspired oxygen concentration of 95% was required to increase PaO₂ to 300 mmHg in the obese patients. However, this 150-mmHg increase in arterial partial pressure improved subcutaneous oxygenation only 10 mmHg — to a value that remained marginal. Medical oxygen is possibly the least expensive drug, and supplemental intraoperative oxygen is easy to provide. Prolonged administration of oxygen at concentrations near 100% causes pulmonary toxicity.^{22,23} However, short-term exposure in the perioperative period is non-toxic and may even improve pulmonary resistance to infection.²⁴ Perioperative concentrations of oxygen restricted to 80% do not provoke atelectasis or other pulmonary dysfunction.²⁵ Available data thus suggest that it would be prudent to provide obese patients with an inspired oxygen concentration of at least 80% because the risk and cost are small, and doing so somewhat improves tissue oxygenation.

Supplemental oxygen administration is one of many factors influencing subcutaneous tissue oxygen partial pressure. For example, it is well established that hypothermia,⁷ surgical and postoperative pain,^{26,27} and smoking⁸ all reduce tissue oxygen tension. In contrast, administration of supplemental fluid,²⁸ hypercapnia,²⁹ and epidural anesthesia^{27,30} increase subcutaneous tissue oxygenation. Intraoperative tissue oxygenation was sub-optimal in our obese patients at a PaO₂ of 150 mmHg and remained marginal even with an inspired oxygen concentration of 95%. Thus, it is likely that combining supplemental oxygen with other treatments that have a potential of improving tissue oxygenation may prove beneficial in obese patients.

Interestingly, arm and wound tissue oxygen tensions were comparable in obese and non-obese patients on the first postoperative day. Furthermore, supplemental oxygen administration was considerably more effective postoperatively in the obese patients than intraoperatively (≈ 20 mmHg vs. ≈ 10 mmHg) — although delivery of oxygen (*via* a face mask) was surely less effective. A potential explanation for this different response is that the obese patients had significantly higher core temperatures. Hyperthermia causes peripheral vasodilation, hyperemia, and increased tissue perfusion.³¹ Furthermore, it is likely that obese patients are fairly hypercapnic in the postoperative period; mild hypercapnia is known to improve tissue oxygenation.²⁹

Intraoperatively we recorded subcutaneous oxygen partial pressure from a needle-induced surrogate wound in the arm. This is the classical method of evaluating perioperative tissue oxygenation and has been used in numerous previous studies.^{16,26,29} The primary benefit of this location is convenient access and the fact that measurements can be conducted during surgery. However, the tissue of interest is actually the surgical incision. We therefore simultaneously recorded tissue oxygenation adjacent to the surgical incision postoperatively. The results were encouraging in that average values at each site were virtually identical. Similar arm and wound partial pressures are consistent with the single previous direct clinical comparison by Chang, *et al.*,³² who reported that measurements in the chest wound are only about 10 mmHg less than in the arm. In that study, though, PsqO₂ measurements were performed directly in a mastectomy wound whereas our values were recorded from surrogate wounds adjacent to the surgical incision. While Chang did not measure subcutaneous tissue temperature (Ts_q), our PsqO₂ values are temperature corrected. This is of importance because Ts_q was greater in the wound than in the arm under all conditions, presumably as a result of inflammation and erythema. Taken together, these results suggest that values recorded from the arm are reliable substitutes for less convenient measurements from surgical wounds.

The observed tissue oxygen values in our study were slightly less than in previous studies, even in the non-obese patients.⁶ This may be explained by the fact that our patients were randomly assigned to two oxygen treatments. Consequently, in 50% of all patients the high oxygen condition was initiated at a point in time during anesthesia and surgery when peripheral tissue perfusion might have already been compromised because of sympatho-adrenergic stimulation and relative hypovolemia.

Naturally, it was impossible to randomly assign patients to the two weight groups. A consequence is that there were more women in the obese than non-obese group. At present, there are no convincing data available that gender *per se* might be a confounding factor of tissue oxygenation and perfusion. Nevertheless, some endocrinologic influence cannot be excluded. For example, 17 β -estradiol is a vasodilator and increases cardiac output.

The two primary determinants of subcutaneous oxygenation are arterial oxygen partial pressure and tissue perfusion, both of which depend critically on anesthetic and fluid management. Fluid management was thus strictly controlled by protocol, using estimated normal body weight as the basis for management. However, there is no consensus on what constitutes optimal or even comparable fluid management in the obese.³³ It is thus possible that this conservative regimen made our obese patients relatively hypovolemic. Nonetheless, hemodynamic parameters, such as mean arterial blood pressure and cardiac index were similar in both groups, as was urine output. These data suggest that our perioperative fluid administration was adequate even in the obese patients.

Surgery lasted significantly longer in the obese than non-obese patients. However, intraoperative tissue oxygen was recorded at similar times, usually during the first two hours of surgery in each weight group. It thus seems unlikely that observed intraoperative differences between the obese and non-obese patients was an artifact of anesthetic or fluid management. There is evidence that duration of surgery is a confounding factor in regards to postoperative tissue oxygen tension,² we cannot exclude that duration of surgery effected our PsqO₂ values in the postoperative period. However, all patients underwent major abdominal procedures with similar surgical stress responses and pathophysiological alterations.³²

It seems likely that preoperative tissue oxygenation differed substantially in the obese and non-obese patients. However, we cannot confirm this assertion because our recording began after induction of general anesthesia. The primary reason we delayed data collection was that unanesthetized patients find probe insertion uncomfortable. Furthermore, measurements during baseline conditions in uninjured tissues before initiation of surgical trauma and bacterial contamination are of less clinical impact. Thus, we considered only intra- and post-operative measurements to be of major importance.

In summary, our data indicate that obesity, defined as a BMI ≥ 30 kg/m², is a major determinant of perioperative tissue oxygenation. In obese surgical patients, subcutaneous tissue hypoxia was common. Even with supplemental oxygen administration, tissue oxygen tension was reduced to a level that is associated with a substantial increase in infection risk. Tissue oxygenation recorded from a surrogate wound in the arm was similar to that recorded from a probe inserted adjacent to the surgical incision, suggesting that the technically simpler arm measurements can be substituted for more challenging wound recordings.

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Fig. 1. Intraoperative tissue oxygen tensions during both oxygen conditions in individual obese patients. Group means and 95% confidence intervals are shown to the side.

Fig. 2.
Intraoperative tissue oxygen tensions during both oxygen conditions in individual non-obese patients. Group means and 95% confidence intervals are shown to the side.

Fig. 3. Intraoperative tissue oxygen tensions during both oxygen conditions in individual obese and non-obese patients. Both horizontal and vertical error bars are interquartile ranges.

Table 1

Demographics and Morphometric Characteristics.

	BMI ≥ 30 kg/m ²	BMI < 30 kg/m ²	<i>P</i>
Number	23	23	
Age (y)	44 $\pm$ 9	44 $\pm$ 13	0.619
Weight (kg)	144 $\pm$ 45	70 $\pm$ 14	<0.001
Height (cm)	166 $\pm$ 10	170 $\pm$ 10	0.253
BMI (kg/m²)	51 $\pm$ 15	24 $\pm$ 4	<0.001
Gender (M/F)	3/20	12/11	0.005
Preoperative Hemoglobin (mg/dl)	13.2 $\pm$ 1.4	12.7 $\pm$ 1.4	0.207
ASA Status (I/II/III)	0/8/15	2/20/1	<0.001
Duration of Anesthesia (min)	245 [215, 300]	180 [155, 210]	0.017
Duration of Surgery (min)	195 [172, 260]	130 [115, 177]	0.004

Data are presented as means $\pm$ SDs, medians [25th percentile, 75th percentile], or as counts for the categorical outcomes. Means were compared with an unpaired two-sided t-tests; medians with Wilcoxon rank-sum tests, and counts with the chi-square tests or Fisher's exact tests.

Table 2

Intraoperative Management

	BMI ≥ 30 kg/m ²	BMI < 30 kg/m ²	<i>P</i>
Core Temperature (°C)	36.1 $\pm$ 0.5	35.9 $\pm$ 0.5	0.125
End-tidal Sevoflurane (%)	1.7 $\pm$ 0.5	1.6 $\pm$ 0.5	0.497
Fentanyl (mg)	0.25 [0.25, 0.50]	0.25 [0.25, 0.50]	0.879
Blood Loss (mL)	300 [200, 500]	300 [150, 300]	0.531
Crystalloid (mL/h)	919 $\pm$ 235	874 $\pm$ 327	0.589
Colloid (mL/h)	0 [0, 86]	0 [0, 143]	0.661
Urine Output (mL/h)	115 [91, 192]	115 [71, 234]	0.974
Hemoglobin (mg/dL)	11.6 [10.5, 12.1]	11.2 [10.5, 11.6]	0.523

Data are presented as means $\pm$ SD or median [25th percentile, 75th percentile]. Means were compared with unpaired, two-sided, t-tests and medians with Wilcoxon rank-sum tests.

Table 3
Intraoperative Hemodynamics, Respiratory Parameters, and Tissue Oxygenation

	BMI ≥ 30 kg/m ²		BMI < 30 kg/m ²	
	PaO ₂ = 150 mmHg	PaO ₂ = 300 mmHg	PaO ₂ = 150 mmHg	PaO ₂ = 300 mmHg
MAP (mmHg)	86 $\pm$ 9	89 $\pm$ 11	81 $\pm$ 11	86 $\pm$ 9*
Cardiac Index (L/m ²)	4.0 $\pm$ 0.5	3.8 $\pm$ 1.0	4.3 $\pm$ 1.4	3.8 $\pm$ 1.4
PaO ₂ (mmHg)	159 [137, 174]	293 [269, 324]*	178 [145, 196]	294 [276, 334]*
PaCO ₂ (mmHg)	41 [36, 43]	39 [37, 42]	38 [35, 40]	39 [38, 42]
PsqO ₂ (mmHg)	36 [30, 55] [#]	47 [39, 75]* [#]	57 [53, 72]	76 [56, 92]*
Tsq (°C)	32.8 [32.1, 33.3] [#]	32.9 [32.0, 33.4] [#]	33.9 [33.3, 34.8]	34.1 [33.4, 34.8]

Data are presented as means $\pm$ SDs or medians [25th percentile, 75th percentile]. Asterisks (*) indicate $P < 0.05$ compared to PaO₂ = 150 mmHg (paired t-test or a Wilcoxon signed-rank test). Pound signs (#) indicate $P < 0.05$ compared to non-obese group value (unpaired t-test or Wilcoxon rank-sum test). PsqO₂ = the subcutaneous oxygen tension; Tsq = the subcutaneous temperature.

Table 4

Postanesthesia Care Unit Management

	BMI $\geq$ 30 kg/m ²	BMI < 30 kg/m ²	<i>P</i>
MAP (mmHg)	103 $\pm$ 11	104 $\pm$ 15	0.952
Core Temperature (°C)	36.2 $\pm$ 0.7	36.0 $\pm$ 0.7	0.323
Crystalloid (mL/h)	605 $\pm$ 263	489 $\pm$ 246	0.149
Urine Output (mL/h)	205 [100, 500]	190 [110, 335]	0.979
Pain (VAS)	77 [45, 84]	80 [65, 98]	0.061
PaO ₂ (mmHg)	108 [97, 164]	144 [130, 176]	0.255
PaCO ₂ (mmHg)	48 [45, 51]	44 [41, 45]	0.012

Data are presented as means $\pm$ SDs or medians [25th percentile, 75th percentile]. Means were compared with unpaired, two-sided, t-tests and medians with Wilcoxon rank-sum tests. VAS is a 100-mm visual analog scale with 0 mm as no pain and 100 mm as the worst imaginable pain.

Table 5

Postanesthetic Care Unit Tissue Oxygenation

	BMI ≥ 30 kg/m ²		BMI < 30 kg/m ²	
	Arm	Wound	Arm	Wound
PsqO₂ (mmHg)	43 [37, 54] [#]	42 [36, 60] [#]	54 [47, 64]	62 [49, 68]
Tsq (°C)	32.9 [32.2, 33.5] [#]	34.9 [33.9, 35.9] [*]	33.8 [33.0, 34.3]	34.8 [34.0, 35.7] [*]

Data presented as medians [25th percentile, 75th percentile]. Asterisks (*) indicate $P < 0.05$ compared to the arm (Wilcoxon signed-rank test); pound signs (#) indicate $P < 0.05$ compared to the non-obese patients (Wilcoxon rank-sum test). PsqO₂ = the subcutaneous oxygen tension; Tsq = the subcutaneous temperature.

Table 6
Postoperative Management on the First Postoperative Morning

	BMI ≥ 30 kg/m ²	BMI < 30 kg/m ²	<i>P</i>
MAP (mmHg)	84 $\pm$ 11	87 $\pm$ 17	0.418
Core Temperature (°C)	37.5 $\pm$ 0.8	36.8 $\pm$ 0.8	0.017
Fluid Intake/24hours (ml)	1700 [1480, 1918]	2100 [1560, 2744]	0.091
Urine Output/24hours (ml)	900 [550, 1500]	1405 [1238, 2215]	0.028
Pain (VAS)	47 [30, 75]	45 [10, 50]	0.524

Data are presented as means $\pm$ SDs or medians [25th percentile, 75th percentile]. Means were compared with unpaired, two-sided, t-tests, and medians were compared with Wilcoxon rank-sum tests.

Table 7
Tissue Oxygenation on the First Postoperative Morning

		BMI ≥ 30 kg/m ²		BMI < 30 kg/m ²	
		Arm	Wound	Arm	Wound
Baseline					
SaO ₂ (%)			96 [96, 97]		97 [96, 98]
PsqO ₂ (mmHg)		48 [39, 58]	51 [42, 57]	52 [45, 58]	52 [45, 62]
Tsq (°C)		34.7 [33.5, 36.0]	36.4 [36.0, 37.1]*	34.6 [33.6, 35.5]	36.3 [35.5, 36.8]*
Oxygen Challenge					
SaO ₂ (%)			100 [99, 100] [#]		100 [99, 100] [#]
PsqO ₂ (mmHg)		69 [43, 84] [#]	72 [49, 81] [#]	75 [55, 85] [#]	81 [62, 105] [#]
Tsq (°C)		35.0 [34.4, 36.3]	36.4 [36.1, 37.1]*	34.4 [33.7, 35.2]	36.3 [35.6, 36.8]*

Data presented as medians [25th percentile, 75th percentile]. Asterisks (*) indicate $P < 0.05$ compared to arm; pound signs (#) indicate $P < 0.05$ compared to the baseline values (Wilcoxon rank-sum test). Tissue oxygenation did not differ significantly in the obese and non-obese patients.