Paravertebral Analgesia with Levobupivacaine Increases Postoperative Flap Tissue Oxygen Tension after Immediate Latissimus Dorsi Breast Reconstruction Compared with Intravenous Opioid Analgesia


Background: Directly measured tissue oxygen tension (P\textsubscript{T}O\textsubscript{2}) reflects the adequacy of local tissue oxygenation and influences surgical wound healing. Epidural analgesia increases P\textsubscript{T}O\textsubscript{2} compared with intravenous morphine analgesia after abdominal surgery. The authors tested the hypothesis that paravertebral regional anesthesia and analgesia would increase P\textsubscript{T}O\textsubscript{2} compared with intravenous opioid-based anesthesia and analgesia.

Methods: Twenty patients scheduled to undergo mastectomy with immediate latissimus dorsi breast reconstruction were randomized to receive either general anesthesia with postoperative intravenous morphine analgesia or combined general-paravertebral anesthesia with continuous paravertebral postoperative analgesia using levobupivacaine in this prospective, cohort study. All patients had a local tissue oxygen sensor implanted in the flap muscle. Data were downloaded continuously for 20 h postoperatively.

Results: The mean P\textsubscript{T}O\textsubscript{2} over the 20-h period was significantly higher in patients receiving paravertebral anesthesia (75 ± 38 vs. 44 ± 23 mmHg [mean ± SD]; P = 0.03). Intraoperative blood loss was less in paravertebral patients (1.2 ± 0.4 vs. 1.7 ± 0.5 l; P = 0.04). Dynamic visual analog scale pain scores were significantly lower in paravertebral patients. Intraoperative and postoperative fluids administered, hemoglobin, core temperature, intraoperative end-tidal carbon dioxide, and mean arterial pressure were similar in both groups.

Conclusion: The postoperative latissimus dorsi flap P\textsubscript{T}O\textsubscript{2} was higher for 20 h after breast reconstruction with paravertebral analgesia compared with intravenous morphine analgesia.

DELAY or failure of healing of surgical wounds, usually a result of infection, is one of the most common causes of postoperative morbidity, long hospital stays, and increased costs.\(^4\) Successful surgical wound healing requires resistance to infection, which depends mainly on oxidative killing by neutrophils. Tissue oxygen tension (P\textsubscript{T}O\textsubscript{2}) is an especially important determinant of postoperative wound healing because the bactericidal ability of neutrophils is directly related to P\textsubscript{T}O\textsubscript{2}.\(^2\) The incidence of surgical wound infection is dependent on wound P\textsubscript{T}O\textsubscript{2}.\(^3\) Although evaluation of the factors contributing to surgical wound infection and healing has generally focused on surgical issues, there is emerging evidence that many features of anesthesia and perioperative care are influential in promoting postoperative wound healing.

Surgery and postoperative pain evoke profound neuroendocrine and cytokine activity, known as the stress response. Consequent activation of the sympathetic nervous system may evoke arteriolar vasoconstriction, reducing tissue perfusion and P\textsubscript{T}O\textsubscript{2}.\(^2\) It has also recently been shown that P\textsubscript{T}O\textsubscript{2}, measured at a site remote from the surgical incision, is higher in patients with superior postoperative pain relief, implying that poorly controlled surgical pain reduces tissue oxygen levels sufficiently to significantly increase the risk of surgical wound infection.\(^6\)

It is now accepted that immediate breast reconstruction is oncologically safe and psychologically superior to standard mastectomy in the management of women with breast cancer.\(^7,\)\(^8\) This involves using a myocutaneous flap (latissimus dorsi) to recreate a breast. Complications of elevating a latissimus dorsi flap are hypoxia and ischemia-induced necrosis.\(^9\) In subcutaneous tissue, oxygen tension levels above 40 mmHg are associated with normal wound healing if other major factors are unimpaired.\(^2,\)\(^3,\)\(^10\) The optimum P\textsubscript{T}O\textsubscript{2} to minimize the incidence of surgical wound infection and prevent ischemic necrosis of flap tissue is unknown, but it involves muscle, not subcutaneous tissue. A study of muscle P\textsubscript{T}O\textsubscript{2} during hypotensive anesthesia found baseline levels of P\textsubscript{T}O\textsubscript{2} in muscle tissue of 24–25 mmHg.\(^11\)

Epidural anesthesia and analgesia have recently been shown to increase P\textsubscript{T}O\textsubscript{2} in wound tissue after abdominal surgery.\(^12\) A comparison of thoracic paravertebral block with thoracic epidural anesthesia indicated that the former provided superior analgesia and attenuated the stress response to a greater extent.\(^13\) Therefore, we tested the hypothesis that patients undergoing mastectomy and immediate latissimus dorsi breast reconstruction with combined general anesthesia and paravertebral analgesia, followed by postoperative paravertebral infusion analgesia, have increased flap P\textsubscript{T}O\textsubscript{2} compared with general anesthesia alone followed by conventional intravenous morphine analgesia.

Materials and Methods

After approval of the Mater Misericordiae Hospital Ethics Committee (Dublin, Ireland) and written informed consent, twenty patients scheduled to undergo mastectomy and immediate latissimus dorsi breast reconstruction were randomized to receive either general anesthesia with postoperative intravenous morphine analgesia or combined general-paravertebral anesthesia with continuous paravertebral postoperative analgesia using levobupivacaine. All patients had a local tissue oxygen sensor implanted in the flap muscle. Data were downloaded continuously for 20 h postoperatively.
consent were obtained, 22 patients, aged 21–70 yr, presenting for mastectomy and immediate latissimus dorsi breast reconstruction were enrolled in this prospective, randomized cohort trial. Patients with a recent history of infection, pyrexia, malnutrition (serum albumin < 30 g/dl), immunosuppression (leukocyte count < 2.5 × 10^3 cells/ml), or coagulopathy; patients taking vasoactive drugs; and patients for whom regional anesthesia or morphine analgesia was contraindicated were excluded. Patients were randomly assigned to one of two groups using blocked randomization from a table of random numbers. The assignments were kept in sealed, sequentially numbered envelopes until use.

**Paravertebral Anesthesia**

Paravertebral anesthesia was induced with patients awake, in the lateral “knee-chest” position, and sedated with 1–3 mg intravenous midazolam. The upper thoracic spinous processes were identified with a marking pen. Opposite these, a series of points 3 cm lateral to the midline were marked on the ipsilateral side for surgery. A vertical line between these points, parallel to the midline, corresponds to the vertebral transverse processes. Under aseptic conditions, a Tuohy needle was advanced to contact the transverse process at a depth of 3–4 cm. The needle was then “walked” off the surface of the transverse process and advanced 1–2 cm further. Loss of resistance to saline indicated passage of the needle through the costotransverse ligament into the paravertebral space. An epidural catheter was then advanced into the space 3–4 cm, similarly to the approach taken with an epidural itself. A bolus of levobupivacaine, 0.25%, was given to all patients initially. Further boluses of 10–15 ml levobupivacaine, 0.25%, were given at hourly intervals thereafter, depending on the patient’s weight: patients exceeding 70 kg received a 15-ml bolus; patients below this weight received a 10-ml bolus. After the initial bolus, general anesthesia was induced without defining the dermatomal distribution of the block.

Patients allocated to the paravertebral group received an epidural catheter into the ipsilateral (side of surgery) paravertebral space at the T3 or T4 level under local anesthesia, with the precise level being at the discretion of the anesthetist caring for the patient. After an initial bolus of 10–15 ml plain levobupivacaine, 0.25%, general anesthesia was induced as for the morphine patient-controlled analgesia (PCA) group (see below). These patients received continuing paravertebral analgesia for at least 24 h postoperatively, with an infusion of plain levobupivacaine, 0.25%, at 8–10 ml/hr. They also had an intravenous line attached, delivering prescribed fluids on demand. Patients in the general anesthesia–morphine PCA group received induction of general anesthesia. Intraoperatively, these patients received a loading dose of 0.1 mg/kg morphine and a PCA device prescribed to deliver 1-mg boluses, with a 5-min lockout interval.

**Anesthetic Management**

General anesthesia was induced in all patients (irrespective of group allocation) with propofol (1–3 mg/kg), fentanyl (1–3 μg/kg), and atracurium (0.5 mg/kg) and was maintained with isoflurane in nitrous oxide and oxygen. Fresh gas flow rates were adjusted so that all patients received an intraoperative inspired oxygen fraction (FiO₂) of 0.75–0.8. End-tidal isoflurane values were adjusted to maintain mean arterial pressure (MAP) within 25% of the preinduction values. A crystalloid infusion of 10 ml · kg⁻¹ · h⁻¹ was administered during surgery. This rate of volume infusion was based on the need to replace the significant blood loss associated with the first and second stages of this surgery and the need to compensate for fluid deficit associated with a 12- to 18-h fast because this surgery is usually reserved until afternoon in our institution. Further fluid therapy was left to the discretion of the anesthetist, depending on the individual patient’s estimated blood loss.

All patients had a forced convective air warmer placed before commencement of surgery, with core temperature monitored by a nasopharyngeal thermometer. At the end of surgery, the sterile oxygen sensor implants were sutured into the muscle of the latissimus dorsi flap in all patients. All patients received standard antibiotic prophylaxis.

Target dermatomal distribution of paravertebral blocks was T1–T10. This was assessed using ethyl chloride spray between 30 and 60 min after emergence from general anesthesia. If the segmental range of block was less than the T1–T10 range or if a patient experienced pain in the surgical area, a further bolus of levobupivacaine, 0.25%, was given in the recovery room.

**Surgery**

Mastectomy was performed using a skin-sparing technique, and a periareolar incision was used in all cases. Skin flaps were elevated using an argon diathermy at variable depth depending on the patient’s body habitus, leaving only skin and subcutaneous tissue. This allowed the entire breast to be mobilized and harvested from the clavicle superiorly to the inframammary fold inferiorly. Axillary dissection was then performed, and the thoracodorsal pedicle was identified and protected.

The patient was then placed in the supine position and redraped. Reconstruction was completed by mobilizing the pectoralis major and making a complete muscle envelope for the expander prosthesis by suturing the pectoralis major and latissimus dorsi together and then suturing them to the inframammary fold inferiorly and the chest wall fascia laterally. Skin overlying the latissimus dorsi flap was used to replace the excised areola. At the completion of the procedure, a sterile oxygen sensor was placed through the native breast skin into the mobilized latissimus dorsi muscle flap and sutured in place.


$P_{O_2}$ Measurement

Tissue oxygen tension was measured using a tissue oxygen sensor, located within a saline-filled tonometer, on a 15-cm probe (CC1-SB; Licox Medical Systems, Integra Neurosciences, Hamps, United Kingdom). This was implanted at the end of surgery into the subcutaneous tissue of the wound, along its longitudinal axis, with only the electric connection protruding. The surgeon inserting the probe was unaware of the patient’s group assignment. The disposable microprobe, made of Revoxode (Harvard Apparatus, Holliston, MA) material, was connected to a digital bedside monitor (Licox CMP; Integra Neurosciences), which displayed $P_{O_2}$ values directly in mmHg, together with a graphical trend. Revoxode probes average the heterogeneous local $P_{O_2}$ values over their probe area of 14 mm$^2$, which reduces random positioning error of microprobe sensors. Oxygen diffuses from the tissue through the polyethylene wall of the catheter into its inner electrolyte chamber, where the Revoxode enables reversible electrolyte reactions to occur at the Clark electrode. This property of the Revoxode preserves sensitivity and offset within a narrow range (5%) over a 5-day period of operation.

The oxygen sensors were for single use and were individually calibrated by the manufacturer. A “smart card” within the packaging for an individual probe was inserted into the Licox bedside monitor and used only in conjunction with that specific probe. Sensors were calibrated at the factory to achieve a 3% maximum variance from room air partial pressure of oxygen. No probes exceeded this level of drift over the duration of the study. Polarographic oxygen measurements are temperature dependent. The Licox monitor was manually adjusted to the postoperative core temperature every 4 h during the observation period. The digital display monitor automatically corrects $P_{O_2}$ values to the set temperature. The oxygen sensors interface with a laptop computer, allowing data to be recorded on a continuous basis and stored electronically. We created a program from which the averaged hourly values were transferred into an excel spreadsheet for analysis.

Recording of $P_{O_2}$ commenced 30 min after insertion of the last surgical suture. These probes were removed from the patient 24 h later. All patients were kept in the recovery room for at least 1 h, after which they were discharged to the postoperative wards. Postoperatively, all patients received 10 l/min oxygen ($F_{IO_2} = 0.4$) via a conventional facemask, thus ensuring that $P_{O_2}$ remained largely a function of local tissue perfusion.

Oxygen therapy was continued after transfer of patients to the surgical postoperative wards. Arterial oxygen saturation ($SpO_2$) was monitored continuously after surgery using standard finger pulse oximetry.

Outcome Measures

The primary outcome measure was postoperative $P_{O_2}$ values. Visual analog scale pain scores on moving were also documented. The visual analog scale is a rating scale for pain. Patients were shown a 10-cm line, with one end marked “no pain” and the other marked “intolerable pain.” Patients were asked to mark on this line the point that best reflected their pain on that scale at that time. These parameters were recorded at hourly intervals for 20 h postoperatively. Secondary outcome measures included demographic data, physical status of the patients, duration of surgery, total volume of intraoperative and postoperative fluid given, total amount of bupivacaine or morphine given, intraoperative MAP, $F_{IO_2}$, $SpO_2$, and core temperature, and postoperative MAP and arterial oxygen saturation. Preoperative laboratory findings (full blood count, urea and electrolytes, and coagulation profile) smoking history, coexisting systemic disease, American Society of Anesthesiologists risk grade, and drug therapy were also recorded.

Power Analysis and Statistics

A previous study has shown that additional analgesia can increase $P_{O_2}$ by 25 mmHg, which is clinically significant because this could significantly reduce surgical wound infection. The SD of $P_{O_2}$ values in postoperative patients using opioid PCA ranges from 10 to 20 mmHg, with an average of 15 mmHg. Therefore, accepting a type I error risk of 5% and a type II error risk of 10% (power 90%), nine patients would be required in each group to evaluate our hypothesis. We used intention-to-treat analysis (i.e., to consider patients according to the allocated group) and to analyze results using SPSS version 10 for Windows (SPSS Inc., Chicago, IL). Intraoperative and postoperative values were averaged for each patient, and the resulting values were averaged among the patients in each group. Values recorded at 10-min intervals were used for this purpose for MAP, $SpO_2$, heart rate, and other measures. As stated, $P_{O_2}$ values were recorded every 5 s and downloaded: an Excel program computed the mean values over each hour. These data were inspected for distribution and then compared between the groups using independent samples $t$ test, analysis of variance, or Mann–Whitney U test as appropriate.

Results

Twenty-two patients were enrolled in this study, with 12 randomized to the intravenous opioid group and 10 randomized to the paravertebral group. Equipment failure occurred in two patients in the intravenous opioid group, resulting in total loss of $P_{O_2}$ data. These patients were excluded from the analysis. One patient in the paravertebral group experienced total failure of the block, which was suspected because of hemodynamic instability in the intraoperative period and confirmed in the PACU, because of unacceptably high pain values.
Fig. 1. Study profile. IV = intravenous; PCA = patient-controlled analgesia.

This patient was given repeated boluses of morphine analgesia and then PCA, following the protocol for the intravenous opioid group (fig. 1). This patient was analyzed as being part of the paravertebral group, using intention-to-treat analysis.

Patient characteristics and intraoperative data for variables influencing \( P_tO_2 \) are shown in table 1. Values were first averaged across the duration of anesthesia for each patient. The resulting values were then averaged among patients in each treatment group. MAP, hemoglobin before and after surgery, intraoperative core temperature, and fluids administered over the 20-h observation period are shown in table 1. MAP values were similar in both groups across all observation times. There were no significant differences in any of these variables. However, mean intraoperative blood loss was less in paravertebral patients (1.2 ± 0.5 vs. 1.7 ± 0.4 l; \( P = 0.04 \); table 1).

The weight of breast tissue excised and the weight of the prosthesis inserted were not significantly different between the groups. Surgical outcome was satisfactory in all patients: All flaps were viable in the postoperative period and remained so in follow-up periods of at least 3 months. This occurred even among patients with \( P_tO_2 \) values that were 25 mmHg or lower. However, three patients with functioning paravertebral block reported brachial neuritis pain involving cervical dermatomes C4–C6, attributable to axillary surgery. These patients required boluses of intravenous morphine totaling 5 mg in the recovery room. No further supplementary analgesia was required, although two of these three patients also had mild brachial neuralgia the next day.

Dynamic visual analog scale pain scores are shown in figure 2. Paravertebral analgesia was associated with significantly less postoperative pain than intravenous opioid analgesia. Static visual analog scale pain scores also showed significantly less pain in the paravertebral patients.

Tissue oxygen tension values are shown in figure 3. After an initial rapid decrease of 30–40 mmHg in the first 1–2 h, values decreased more slowly, in a linear fashion, over the remaining 18–20 h of the observation period. Values were averaged over each 2-hourly time period and are displayed in figure 3. Apart from 0–2 h, \( P_tO_2 \) values at each subsequent epoch were significantly lower than baseline for both the paravertebral and intravenous groups. Paravertebral \( P_tO_2 \) values at each subsequent epoch were significantly lower than baseline for both the paravertebral and intravenous groups. However, the difference of 0.15 mmHg at 20 h at the 95% confidence interval was not statistically significant. A statistically significant difference of 0.19 mmHg was not observed for the intravenous group.

Table 1. Patient Characteristics and Potentially Confounding Variables

<table>
<thead>
<tr>
<th></th>
<th>Paravertebral (n = 10)</th>
<th>Intravenous (n = 10)</th>
<th>( P ) Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>44.3 ± 8.4</td>
<td>38.4 ± 8.7</td>
<td>0.1</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>65.4 ± 12.9</td>
<td>64.0 ± 9.9</td>
<td>0.8</td>
</tr>
<tr>
<td>Preoperative hemoglobin, g/dl</td>
<td>12.9 ± 0.9</td>
<td>12.2 ± 0.5</td>
<td>0.47</td>
</tr>
<tr>
<td>Postoperative hemoglobin, g/dl</td>
<td>7.5 ± 0.6</td>
<td>7.7 ± 0.5</td>
<td>0.8</td>
</tr>
<tr>
<td>Mean arterial pressure, mmHg</td>
<td>67.4 ± 6.0*</td>
<td>76.2 ± 13</td>
<td>0.03</td>
</tr>
<tr>
<td>Core temperature, °C</td>
<td>36.3 ± 0.5</td>
<td>36.4 ± 0.7</td>
<td>0.5</td>
</tr>
<tr>
<td>End-tidal carbon dioxide, mmHg</td>
<td>35.3 ± 4.5</td>
<td>37.5 ± 0.25</td>
<td>0.5</td>
</tr>
<tr>
<td>Duration, h</td>
<td>5.0 ± 0.8</td>
<td>5.2 ± 0.6</td>
<td>0.9</td>
</tr>
<tr>
<td>Colloid, l</td>
<td>0.67 ± 0.5</td>
<td>0.83 ± 0.25</td>
<td>0.2</td>
</tr>
<tr>
<td>Crystallloid, l</td>
<td>4.1 ± 1.5</td>
<td>3.7 ± 1.0</td>
<td>0.3</td>
</tr>
<tr>
<td>Weight breast tissue excised, kg</td>
<td>655 ± 190</td>
<td>695 ± 225</td>
<td>0.6</td>
</tr>
<tr>
<td>Weight prosthesis, kg (median [IQR])</td>
<td>450 (300–550)</td>
<td>450 (300–600)</td>
<td>0.9</td>
</tr>
<tr>
<td>Total 0.25% levobupivacaine, ml</td>
<td>255 ± 25</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Postoperative ( SpO_2 ), %</td>
<td>98 (97–100)</td>
<td>98 (97–100)</td>
<td>0.9</td>
</tr>
<tr>
<td>Blood loss, l</td>
<td>1.2 ± 0.5*</td>
<td>1.7 ± 0.4</td>
<td>0.02</td>
</tr>
</tbody>
</table>

Values are expressed as mean ± SD, except where indicated otherwise.
* Statistically significant.
IQR = interquartile range; \( SpO_2 \) = oxygen tension measured by pulse oximetry.
higher in patients in the paravertebral group compared with the opioid analgesia group. Rescue morphine values given in the PACU were 3.0 \pm 2.5 versus 13.4 \pm 6.6 mg in patients of the paravertebral and intravenous opioid groups, respectively (P = 0.04).

Discussion

We have demonstrated that combined general–paravertebral anesthesia and analgesia increases postoperative \( P_{O_2} \) in the latissimus dorsi flap after mastectomy with immediate breast reconstruction for breast cancer. The scale of the difference in \( P_{O_2} \) values in latissimus dorsi flaps is larger than that which we observed between epidural and opioid analgesia. In that prospective, randomized, double-blind study, patients with epidural analgesia had mean \( P_{O_2} \) values 14 mmHg (SD = 14 mmHg) higher than patients receiving intravenous opioid analgesia.\(^{12}\) This may reflect differences in local perfusion attributable to flap physiology.\(^{16}\)

The SD of \( P_{O_2} \) values in the current study is also much greater than that of the epidural study, suggesting greater biologic variability in factors affecting latissimus dorsi \( P_{O_2} \) than in abdominal wound tissue. Muscle \( P_{O_2} \) is predictably more variable than that of subcutaneous tissue \( P_{O_2} \) because of variable oxygen consumption and more local control of blood flow by muscle tissue than subcutaneous tissue. Major factors influencing \( P_{O_2} \) (MAP, hemoglobin, fluid volume, and core temperature) were similar in the groups. Moreover, there was no difference in values of end-tidal carbon dioxide, which has also been shown to influence \( P_{O_2} \) in volunteers.\(^{17}\)

However, in rotational flaps, such as the latissimus dorsi flap, the risk of necrosis is of greater concern than the risk of infection. Whether maintaining the disparity in \( P_{O_2} \) values seen in this study for 20 h postoperatively confers any additional benefit is unknown. The vessels of the latissimus dorsi flap were rotated 180° to reconstruct the breast and may have been partially obstructed by the weight of the flap. Unfortunately, we did not directly measure blood flow through the flap vessels; therefore, we cannot confirm whether the observed differences in \( P_{O_2} \) values are attributable to regional anesthesia-induced vasodilatation or differences in the quality of analgesia. However, the fact that the weight of the breast tissue excised and the weight of the prosthesis implanted were remarkably similar in all our patients, with no significant differences between the groups, suggests that factors relating to latissimus dorsi flap size did not appreciably affect our results.

The mechanism of our observation of increased \( P_{O_2} \) in latissimus dorsi flaps with levobupivacaine paravertebral analgesia is not adequately explained by this study. The \( P_{O_2} \) values in the latissimus dorsi flap were corrected for core temperature rather than local flap temperature. Manual adjustment of core temperature correction was undertaken at 4-hourly intervals; therefore, temperature changes occurring between these times would have affected \( P_{O_2} \) without being adequately temperature compensated. Moreover, we did not measure latissimus dorsi flap flow, which might also have contributed to the observed difference in latissimus dorsi flap \( P_{O_2} \).

Because paravertebral analgesia has only recently been introduced in our unit, we believed it would have been unsafe and unethical to conduct this study in a double-blind manner. It is possible that the open design could have contributed to the large difference in \( P_{O_2} \) values we observed. However, the primary outcome measure \((P(\text{O}_2))\) is an objective, numerical measurement obtained from a bedside monitor, and it is unlikely to have been influenced by observer bias.

Postoperative pain values were significantly less in patients with paravertebral analgesia, consistent with other studies.\(^{13}\) Pain per se is known to reduce \( P_{O_2} \) values,\(^{5}\) and it is possible that this could have accounted for part of the difference in \( P_{O_2} \) values we observed. Functional regional anesthesia techniques, including paravertebral analgesia, consistently achieve superior analgesia compared with parenteral opioids,\(^{18–20}\) and it would be impossible to attain similar levels of analgesia between these two techniques. Paravertebral analgesia also effectively inhibits the surgical stress response,\(^{18–20}\) thereby reducing sympathetic nervous system adrenergic activity, which may have contributed to improved local perfusion and hence \( P_{O_2} \).

Interestingly, we observed a reduction in blood loss of approximately 500 ml in patients receiving paravertebral anesthesia. Spinal and epidural anesthesia have previously been shown to reduce perioperative blood loss,\(^{21}\) possibly by reducing MAP. We also observed lower intraoperative MAP in patients receiving paravertebral analgesia, to which the observed reduction in blood loss may be attributable. This difference was not maintained into the postoperative period. However, the greater blood loss in the intravenous group suggests these patients may have inadvertently become more hypovolemic than the paravertebral patients because both groups...
were administered similar volumes of intravenous fluid. Volume status is an important determinant of $P_{EO_2}$\textsuperscript{22} and it is possible that this may have reduced peripheral perfusion sufficiently to explain our finding of decreased $P_{EO_2}$ in the intravenous group. However, our data were from latissimus dorsi muscle, which is not as sensitive an indicator of peripheral perfusion as subcutaneous tissue.\textsuperscript{15}

In conclusion, this prospective, randomized cohort study comparing paravertebral anesthesia and analgesia with intravenous opioid analgesia in patients undergoing immediate latissimus dorsi breast reconstruction has shown that the paravertebral technique results in higher latissimus dorsi flap $P_{EO_2}$. Future studies are warranted to distinguish whether the mechanism of our observation is attributable to latissimus dorsi flap tissue blood flow or temperature differences, superior pain relief, sympathetic activity, or covert hypovolemia.

The authors thank the Recovery Room and Ward Nursing staff (Mater Misericordiae Hospital, Dublin, Ireland) for their cooperation with data collection.

References


