Use of Near-Infrared Spectroscopy to Measure Tissue Oxygen Saturation During Total Knee Arthroplasty With Use of a Tourniquet

Riley R. Gaines, DNP, CRNA
Andi N. Rice, DNP, CRNA
Jeffrey C. Gadsden, MD, FRCPC, FANZCA
Brett T. Morgan, DNP, CRNA
Charles A. Vacchiano, PhD, CRNA, FAAN

The primary purpose of this proof-of-concept quality improvement effort was to evaluate the practicality of using near-infrared spectroscopy (NIRS) to measure tissue oxygen saturation (Sto2) during total knee arthroplasty (TKA) with use of a tourniquet. NIRS sensors were applied to the biceps femoris (BF) and gastrocnemius (GS) muscles of both lower extremities of patients undergoing TKA procedures. For a convenience sample of 15 patients, measurement of Sto2 was attempted at baseline, following subarachnoid block administration, and after tourniquet inflation and deflation. Mean baseline Sto2 (SD) was 71% (6%) in the BF muscle and 66% (7%) in the GS muscle. Significant changes in Sto2 values were observed following subarachnoid block, tourniquet inflation, and tourniquet deflation. The Sto2 returned to or above baseline in the BF muscle but did not return to baseline in the GS muscle following tourniquet deflation. Changes in tissue oxygen saturation resulting from use of a tourniquet can be continuously monitored with the use of an NIRS device. Further evaluation of the use of NIRS should be undertaken to determine if it could be used to guide safe duration and pressure limits for tourniquet inflation.

Keywords: Near-infrared spectroscopy, tissue oxygen saturation, total knee arthroplasty, tourniquet.
Near-infrared spectroscopy sensors (Equanox Advance Model 8004CA, Nonin Medical Inc) were applied to the biceps femoris (BF) and the gastrocnemius (GS) muscles on both the operative and nonoperative lower extremities of a convenience sample of 15 patients undergoing TKA (Figure 1). The BF sensor on the operative leg was positioned just distal to the tourniquet. Tissue oxygen saturation was monitored continuously from all 4 sensors with a precalibrated Model 7600 oximeter (Nonin Medical Inc) by the project participants who had extensive experience with the device, and the data were downloaded to a laptop computer at 1-second intervals.

All patients received a preoperative peripheral nerve block (PNB; adductor canal and posterior capsular), followed by either a subarachnoid block (SAB) administered in the preoperative holding area or general anesthesia with induction in the OR. Tissue oxygen saturation values were collected at the following times: (1) a 5-minute baseline period in the preoperative holding area before administration of the SAB, (2) following SAB, (3) throughout the intraoperative period following tourniquet inflation and deflation, and (4) postoperatively during transport to the postanesthesia care unit (PACU) and for the first 10 minutes in the PACU (Table 1).

Following baseline and post-SAB administration data collection, the oximeter sensor leads were disconnected and the patient was transported to the OR. On the patient's arrival in the OR, the sensor leads were reconnected and the data download resumed except for the GS muscle of the operative leg, for which the sensor was in the operative field. General anesthesia was then induced for those patients not receiving a SAB; sedation, primarily by means of a propofol infusion, was initiated for those receiving a SAB. A sterile securement dressing (Tegaderm, 3M Co) was placed over the GS muscle sensor on the

**Table 1.** Flow Chart Showing Data Collection Time Points

<table>
<thead>
<tr>
<th>Preoperative Holding</th>
<th>PNB</th>
<th>Transfer to OR</th>
<th>Tourniquet Inflation</th>
<th>Tourniquet Deflation</th>
<th>Transfer to PACU</th>
<th>End Data Collection</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline Sto₂ Data</td>
<td>Sto₂ Data</td>
<td>Sto₂ Data</td>
<td>Sto₂ Data</td>
<td>Sto₂ Data</td>
<td>Sto₂ Data</td>
<td>Sto₂ Data</td>
</tr>
</tbody>
</table>

Abbreviations: General Induction, general anesthesia begun for patients not receiving a subarachnoid block (4 of 15 patients); OR, operating room; PACU, postoperative anesthesia care unit; PNB, peripheral nerve block placed (all 15 patients); SAB, subarachnoid block placed (11 of 15 patients); SAB + Sedation = intravenous sedation begun for patients receiving a SAB (11 of 15 patients); Sto₂ Data, tissue oxygen saturation measured and recorded on bilateral lower extremities.

**Table 2.** Demographic Characteristics (N = 15)

<table>
<thead>
<tr>
<th>Demographic Value</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mean (SD), y</td>
<td>63 (9)</td>
</tr>
<tr>
<td>Height, mean (SD), cm</td>
<td>170 (9)</td>
</tr>
<tr>
<td>Weight, mean (SD), kg</td>
<td>95 (11)</td>
</tr>
<tr>
<td>Body mass index, mean (SD), kg/m²</td>
<td>32 (4)</td>
</tr>
<tr>
<td>Gender, No. %</td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>7 (46.7)</td>
</tr>
<tr>
<td>Male</td>
<td>8 (53.3)</td>
</tr>
<tr>
<td>Surgical extremity, No. %</td>
<td></td>
</tr>
<tr>
<td>Left</td>
<td>8 (53)</td>
</tr>
<tr>
<td>Right</td>
<td>7 (47)</td>
</tr>
<tr>
<td>Anesthesia type, No. %</td>
<td></td>
</tr>
<tr>
<td>Subarachnoid block and peripheral nerve block</td>
<td>11 (73)</td>
</tr>
<tr>
<td>General anesthesia and peripheral nerve block</td>
<td>4 (27)</td>
</tr>
</tbody>
</table>

Note that patients who were monitored as part of the project had sensors on both lower extremities.

*Figures and tables* have been integrated into the text as appropriate.
operative extremity, and a sterile ultrasonography probe cover (Safersonic–US Inc) was placed over the sensor lead and pod using sterile technique. The oximeter cable was then reconnected to the pod through a hole cut in the end of the probe cover, and data collection resumed. The covered sensor lead and pod were fixed in place at the patient’s ankle area with a sterile compression dressing (Coban, 3M Co), which was used to exsanguinate the extremity before tourniquet inflation.

A series of paired Student t tests were conducted using statistical analysis software (SPSS version, IBM Corp) to examine differences in Sto 2 between and within the BF and GS muscle groups in the operative and nonoperative extremities at baseline and following the SAB, and within the operative extremity at baseline, following tourniquet inflation, and following tourniquet deflation. The level of significance was set at a P value of < .05.

To determine the rate of change of Sto 2 from baseline following tourniquet inflation and deflation, we performed regression analysis with Sto2 plotted against time and then determined the slope of the line of best fit.

**Results**

A convenience sample of 15 consecutive patients admitted for TKA had Sto 2 measurements recorded. Patient demographics are noted in Table 2. The mean (SD) tourniquet inflation time for these 15 patients was 56.8 (14.9) minutes, with a range of 38.7 to 97.9 minutes. Eleven of the 15 patients had SAB, and the remaining 4 patients underwent general anesthesia. During surgical manipulation of the operative extremity, the monitor cable became disconnected from the oximeter sensor pod in 4 of 15 patients, preventing complete capture of Sto 2 measurements. In 3 of 15 patients the surgical extremity sensor pod itself malfunctioned and prevented complete capture of Sto 2 measurements. Therefore, complete Sto 2 data from baseline through recovery were available for 8 of the 15 patients evaluated.

A comparison of preoperative baseline mean Sto 2 in the BF and GS muscle groups between the nonoperative and operative extremity showed no significant difference (Figure 2). However, preoperative baseline mean Sto 2 in the GS muscle group was significantly lower than in the BF muscle group in both extremities.

We examined changes in Sto 2 following administration of the SAB because of the expected vasodilation that accompanies block administration. There was a measurable and significant increase in mean Sto 2 in both the BF and GS muscle groups in both extremities following SAB (Figure 3). The overall increase in Sto 2 from baseline in both extremities and for both the BF and GS muscle groups following SAB was 10.5%.

Figures 4A and B show changes in mean Sto 2 from baseline as a result of tourniquet inflation and deflation in the operative leg in the BF and GS muscles for the SAB and the general anesthesia groups. There was a significant decrease from baseline in mean Sto 2 on tourniquet inflation in both muscle groups with both anesthetic types. The average decrease in mean Sto 2 across both anesthetic types was 24% and 27% in the BF and GS muscle groups, respectively. On tourniquet deflation there was a significant increase of 8.6% above baseline in mean Sto 2 in the BF muscle in the SAB group, whereas the mean Sto 2 simply returned to baseline in the general anesthesia.
The mean $\text{StO}_2$ value in the GS muscle after achieving stability on tourniquet deflation was 17% below baseline. Table 3 shows the mean time to return to or above baseline in the BF muscle (2 minutes, 28 seconds) or to achieve a stable $\text{StO}_2$ value in the GS muscle (16 minutes, 51 seconds) as well as the rate of change in mean $\text{StO}_2$ expressed as the slope of the line of best fit following tourniquet deflation in the operative extremity. The mean time to achieve a stable $\text{StO}_2$ in the GS muscle following tourniquet deflation was, on average, 14 minutes and 23 seconds longer than the time to return to baseline or above in the BF muscle. In addition, the variability associated with the mean time to return to baseline or achieve a stable $\text{StO}_2$ following tourniquet deflation was greater in the GS muscle (SD = 10 minutes, 12 seconds) compared with the BF muscle (SD = 37 seconds). Finally, the mean slope of the line of best fit was 0.301 for the BF muscle and 0.024 for the GS muscle. Figure 6 shows a representative example of the changes from baseline in the BF and GS muscles in a patient with a SAB following tourniquet inflation and deflation. The example demonstrates the consistently higher $\text{StO}_2$ in the BF muscle vs the GS muscle starting at baseline, the increase in $\text{StO}_2$ after placement of the SAB, the decreased $\text{StO}_2$ following tourniquet inflation, and the increase in $\text{StO}_2$ above baseline in the BF muscle and the failure to return to baseline in the GS muscle following tourniquet deflation.

**Discussion**

The primary purpose of this pilot project was to determine the practicality of using a NIRS device to measure lower extremity $\text{StO}_2$ during TKA procedures using a
tourniquet. We were indeed able to acquire Sto2 values using a Nonin Model 7600 oximeter and Model 8004CA sensors at baseline, and following tourniquet inflation and deflation. Our secondary aim was to determine if changes in lower extremity Sto2 during tourniquet inflation and deflation could be captured using NIRS technology. We were able to obtain baseline Sto2 measurements in 15 patients in the BF and GS muscles of both lower extremities and therefore have added to the knowledge base with respect to “normal” Sto2 values in the leg. Our findings of a baseline mean (SD) Sto2 of 65% (7%) in the GS muscle support the findings of Comerota et al., who reported a mean (SD) Sto2 in the GS muscle of 65% (19%) in a group of “normal” subjects. In our study, we were able to capture an increase in Sto2 values in both the BF and GS muscles following SAB, presumably associated with the resultant vasodilation. We were also able to capture the predicted decline in Sto2 on tourniquet inflation and the consequent reduction in blood flow.

In most of our sample for which we obtained complete data, there was an increase in Sto2 above baseline in the BF muscle following tourniquet deflation, which is consistent with the reactive hyperemia after a period of vascular occlusion reported in the literature. Comerota et al., reported a mean time for Sto2 to return to baseline in the GS muscle of 1.95 minutes in normal subjects after a walking exercise, which was comparable to our finding of a mean time to return to or above baseline in the BF muscle following tourniquet deflation of 2 minutes and 28 seconds. An unexpected finding was the failure of the Sto2 to return to baseline in the immediate postoperative period. This finding suggests there is incomplete resolution of the regional hypoxia in the muscles of the lower leg following tourniquet deflation in the immediate postoperative period.

The Model 7600 oximeter calculates Sto2 based on a 70% contribution from venous blood and 30% from arterial blood and therefore is purported to be a measure of both oxygen supply and demand. Applying this premise to our findings and assuming that oxygen demand would increase following a period of reduced blood flow, ongoing cellular metabolism, and the resultant oxygen debt incurred during tourniquet inflation suggest that in the BF muscle on tourniquet deflation there is an increase in oxygen delivery; however, there appears to be a decrease in oxygen delivery in the GS muscle. Contributors to a decreased oxygen delivery to the GS muscle could include any of the following: (1) surgical trauma, edema, pressure created by the wound dressing, and elevation of the extremity; (2) an alteration in local mediator–driven

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Mean (SD)</th>
<th>Time, min:s Median</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Biceps femoris</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time to return or above baseline</td>
<td>2:28 (0:37)</td>
<td>2:00</td>
<td>1:52-3:20</td>
</tr>
<tr>
<td>Slope</td>
<td>0.301 (0.146)</td>
<td>0.299</td>
<td>0.145-0.596</td>
</tr>
<tr>
<td>Gastrocnemius</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time to stability</td>
<td>16:51 (10:12)</td>
<td>16:28</td>
<td>4:02-23:48</td>
</tr>
<tr>
<td>Slope</td>
<td>0.024 (0.028)</td>
<td>0.022</td>
<td>0.002-0.079</td>
</tr>
</tbody>
</table>

Table 3. Time for Tissue Oxygen Saturation to Return to or Above Baseline in Biceps Femoris Muscle or to Achieve Stable Value in Gastrocnemius Muscle

| a Eight observations. |
| b Rate-of-change slope after tourniquet deflation. |
vasodilation as a result of tissue injury and/or nerve block; or (3) a “steal” phenomenon whereby increased oxygen consumption in the large upper leg muscles following the period of tourniquet-induced tissue hypoxia results in a reduction in available oxygen to the lower leg muscles. Such a reduction in oxygen delivery could contribute to the muscle weakness, electromyographic changes, and increased pain reported to be caused by mechanical compression from a tourniquet.19-21 In this regard, Ejaz et al8 showed that TKA procedures without use of a tourniquet result in better functional outcomes and improved knee range of motion in the early period of rehabilitation.

The primary limitation of this proof-of-concept pilot study is the small number of attempted observations complicated by the even more limited number of complete datasets from baseline through tourniquet deflation. A larger sample size is not only necessary to verify the apparent failure of Sto2 to return to baseline in the GS muscle in the immediate postoperative period, and perhaps beyond, but also to support evaluation of the effect of longer tourniquet inflation times and patient comorbidities that could potentially affect lower extremity oxygen supply. We did not collect blood pressure data, and it is possible that episodes of “hypertension” and “hypotension” during tourniquet inflation or on deflation could have contributed to changes in perfusion and Sto2. There were several logistical and technical problems associated with the data capture. Maintaining sterility of the operating field with a nonsterile sensor applied to the area of the GS muscle required the development of a technique to enclose the sensor, sensor lead, and its associated pod within a sterile envelope. The sensor lead with pod was subsequently fixed to the ankle area and exposed to frequent motion and impact during manipulation of the extremity required by the TKA procedure. Despite the careful connection of the pod to the oximeter cable and fixing of the pod to the patient’s ankle area, there were instances in which the cable became disconnected from the pod and in which the sensor pod failed to function, resulting in loss of data transfer.

To determine the value of tissue oximetry as an objective method to make decisions about the safe duration of tourniquet application and inflation pressure would require that a threshold Sto2 value clearly associated with tissue injury and negative patient outcomes be defined. A starting point for establishing that threshold value would be correlation of Sto2 measurements in muscles groups of the lower extremity during varying tourniquet inflation periods and pressures to biomarkers of tissue injury such as lactic acid, pH, base deficit, and serum creatine phosphokinase.

Conclusion
We have found that changes in Sto2 resulting from the use of a tourniquet can be continuously monitored with the use of an NIRS device. Determining the potential applicability of NIRS technology to surgical tourniquet use during TKA procedures is the first step needed toward establishing objective Sto2 guidelines. Further study of the measurement of Sto2 by application of this technology and correlation with markers of tissue injury could lead to an objective guide to determine safe duration and pressure limits for tourniquet inflation.

REFERENCES
17. Martin DS, Levet DZ, Bezemer R, Montgomery HE, Grocott MP; Caudwell Xtreme Everest Research Group. The use of skeletal muscle near infrared spectroscopy and a vascular occlusion test at

www.aana.com/aanajournalonline


AUTHORS

Riley R. Gaines, DNP, CRNA, practices at Lynchburg General Hospital in Lynchburg, Virginia. At the time this work was being performed she was a student in Duke University School of Nursing, Nurse Anesthesia Program, Durham, North Carolina.

Andi N. Rice, DNP, CRNA, has practiced in academic institutions, community hospitals, and office-based practices and is a consulting associate in Duke University School of Nursing, Nurse Anesthesia Program.

Jeffrey C. Gadsden, MD, FRCPC, FANZCA, is an associate professor and chief, Division of Orthopedics, Plastics, and Regional Anesthesiology, and director, Regional Anesthesiology and Acute Pain Medicine Fellowship at Duke University Hospital, Durham, North Carolina.

Brett T. Morgan, DNP, CRNA, is an assistant professor and program director at Duke University School of Nursing, Nurse Anesthesia Program.

Charles A. Vacchiano, PhD, CRNA, FAAN, is a professor at Duke University School of Nursing, Nurse Anesthesia Program.

DISCLOSURES
The authors have declared no financial relationships with any commercial entity related to the content of this article. The authors did not discuss off-label use within the article. Disclosure statements are available for viewing upon request.