Near-Infrared Spectroscopy versus Compartment Pressure for the Diagnosis of Lower Extremity Compartmental Syndrome Using Electromyography-Determined Measurements of Neuromuscular Function

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**Background:** Compartmental syndrome (CS) is difficult to diagnose in intensive care unit patients. Compartment perfusion pressure (CPP) is an invasive, indirect measure of ischemia. Near-infrared spectroscopy is noninvasive, and directly measures ischemia by transmitting light through tissues at wavelengths that react with hemoglobin to provide percent tissue oxygen saturation (StO₂). Animal studies demonstrate that StO₂ is superior to CPP for detecting CS. However, there are no studies in humans comparing StO₂ with CPP. We hypothesized that StO₂ can reliably detect CS, and is superior to CPP.

**Methods:** CS was induced in 15 human volunteers using a standard calf compression model. At 30-minute intervals, compression was increased to reduce StO₂ from baseline (86% ± 4%) to 60%, 40%, 20%, and < 10%, with simultaneous recording of CPP. Outcome variables included deep peroneal nerve conduction assessed by electromyography, cutaneous peroneal nerve sensitivity using Semmes-Weinstein monofilaments, and pain (visual analog scale).

**Results:** Both StO₂ and CPP significantly correlated with all ischemia outcome variables (p < 0.001). Receiver operating characteristic curves of deep peroneal nerve conduction demonstrated that StO₂ had higher sensitivity than CPP for detecting > 50% block. For example, when specificity was 83% for StO₂ and 84% for CPP, sensitivity was 85% versus 56%, respectively (p = 0.02). When specificity for both was 72%, sensitivity was 94% for StO₂ versus 76% for CPP (p = 0.04).

**Conclusion:** In intensive care unit patients who cannot alert physicians to symptoms, near-infrared spectroscopy may help clinicians to avoid delayed or unnecessary prophylactic fasciotomy, and provides the benefits of a continuous, noninvasive monitoring technique.

**Key Words:** Compartmental syndrome, Near-infrared spectroscopy, Fractures, Oximetry, Musculoskeletal physiology, Ischemia, Orthopedic equipment, Orthopedics.


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A cute compartmental syndrome is a serious complication in patients with extremity fractures, burns, vascular, crush, or ischemia-reperfusion injury. Although the diagnosis may be easily established in alert patients, it is frequently missed in the critically injured because physical examination has poor sensitivity and specificity. Objective measurement of compartment pressure has been of limited utility because of technical difficulties associated with providing frequent or continuous monitoring of compartment pressure. Also, clinicians have failed to reach a consensus regarding the pressure at which fasciotomy should be performed, with recommendations ranging from 30 to 45 mm Hg.

A defined compartment pressure at which fasciotomy should be performed may, in fact, be unobtainable because ischemia rather than pressure appears to be responsible for neuromuscular damage. This has been demonstrated in studies on human volunteers when an increase in pain and impairment of nerve conduction occurred when the compressed leg was elevated, reducing perfusion, and improved when the leg was leveled, while compartment pressure was held constant. Animal studies have confirmed that neuromuscular damage occurs at lower compartment pressures during systemic hypoperfusion. The limitations of current methods of detecting compartmental syndrome may lead to delayed fasciotomy in some patients, and unnecessary fasciotomy in others.

Some have suggested that perfusion pressure (mean arterial pressure minus compartment pressure) is a more valid means of determining the need for fasciotomy. However, if the ischemia concept is valid, perfusion pressure is an incomplete indicator of tissue oxygenation. It fails to take into account hemoglobin concentration and saturation, cardiac output, vascular spasm, venous outflow obstruction, capillary
compression, and skeletal muscle oxygen use, all of which are factors that can contribute to ischemia.6,7

Near-infrared spectroscopy (NIRS) is a noninvasive optical technique that transmits light at wavelengths that pass readily through skin and subcutaneous tissue, but are reflected or absorbed by hemoglobin. The amount of near-infrared light absorbed is dependent on the redox state of the iron molecule of hemoglobin (Hgb) in the muscle. Ischemia increases oxygen extraction, decreasing the oxyhemoglobin-deoxyhemoglobin ratio. NIRS therefore has the potential to quantitatively measure skeletal muscle or tissue oxygen saturation \([\text{StO}_2 = \frac{\text{Hgb O}_2}{\text{Hgb O}_2 + \text{Hgb}} \times 100]\), providing a continuous, noninvasive, method of monitoring for compartment ischemia.

To date, there are no studies performed on humans that compare NIRS with perfusion pressure for detecting acute compartment ischemia. We hypothesized that NIRS has potential use as a diagnostic adjunct for the detection of compartmental syndrome. We also hypothesized that NIRS is more accurate than perfusion pressure because it is a direct measure of ischemia. These hypotheses were tested using a standard human volunteer model of compartmental syndrome.4,9 Ischemia was defined as significant block in deep peroneal motor nerve conduction as determined by electrophysiologic measurements. Secondary measures of ischemia included changes in peroneal sensory nerve function, and pain.

MATERIALS AND METHODS

The study was conducted at Harborview Medical Center, University of Washington School of Medicine, Seattle, Washington, in May 1999. Twenty-five paid volunteers were recruited from the community. Interim analysis was planned at the conclusion of data collection on the first 15 subjects. Eligibility requirements included age 18 to 40 years, and ability to lie supine for several hours. Subjects were excluded if they were pregnant; using oral contraceptives; had a history of diabetes mellitus, deep vein thrombosis, peripheral neuropathy, vascular disease, coagulopathy, hypertension, or previous lower extremity fracture; or currently used analgesics. Informed consent was obtained from all subjects. The study was approved by the University of Washington Institutional Review Board.

Near-Infrared Spectroscopy

An NIRS device (Biospectrometer-NB Oximeter, Hutchinson Technology, Inc., Hutchinson, MN) with a 25-mm probe was secured over the anterior compartment of the lower extremity at the midpoint of the anterior tibialis muscle. The probe (Fig. 1) contains one bundle of optical fibers that emits light, and a second bundle that receives reflected light. The approximate depth of penetration is equal to the separation distance between the bundles (25 mm). The device uses a 25-W tungsten halogen bulb as a light source and filters to remove the wavelengths outside the area of interest (<650 nm, >900 nm). The receiving fibers gather and transmit the reflected light to a custom fiberoptic spectrometer containing focusing mirrors and diffraction grating that illuminates the wavelength-separated light onto a charge-coupled device array. A computer processes the received signals and displays the calculated \(\text{StO}_2\) value. The device is calibrated in roughly 10 minutes using a uniform reflectance material and an internally stored algorithm, is portable, and weighs 29 lb.10–12

Compartmental Syndrome Model and Study Procedures

A 21-cm-wide sphygmomanometric blood pressure cuff with an air pump was placed around the leg extending from 2 cm below the tibial tuberosity to a point just above the ankle. Pressure in the cuff was controlled using a water seal chamber. This was performed by connecting the air pump to a three-way stopcock, connecting a length of rubber tubing to the third port, and suspending the tubing in a 1.5-m-tall plastic cylinder to which water was added or removed. The blood pressure cuff was inflated until bubbling of the water seal occurred, and pressure in the water column was converted to mm Hg (1.36 cm H2O = 1 mm Hg). Studies using simultaneous calf compression and wick catheter measurements have demonstrated a consistent relationship between cuff pressure and compartment pressure [compartment pressure = (1.02 \times \text{cuff pressure in mm Hg}) + 9.0].13–15 Therefore, to avoid the necessity of invasive measurements in volunteers, compartment pressure was calculated, not measured.

Mean systemic arterial pressure was obtained from the upper extremity using an automated blood pressure cuff, and compartment perfusion pressure was calculated. A standard pulse oximeter was placed on the second toe to confirm the continued presence of pulsatile pedal flow, and a hand-held Doppler device was used to confirm the continued presence of the dorsalis pedis pulse. Because of concerns about the
potential mechanical effects of high levels of pressure on the muscles of volunteers, the maximum cuff pressure used in this study was 70 mm Hg. If the desired StO₂ endpoint was not reached, perfusion pressure was further reduced by elevating the leg and measuring the distance of the midpoint of the compartment from the midaxillary line, and subtracting it (converted to millimeters of mercury) from the mean arterial pressure.

Baseline measurements were obtained after a 10-minute equilibration period. StO₂ was the independent variable, and markers of ischemia were the dependent variables. Cuff pressure was then increased by progressively adding to the water seal column until StO₂ decreased to 60%. At 30-minute intervals, StO₂ was further decreased to 40%, 20%, and < 10%. Ischemia measurements were taken after 15 and 30 minutes at each StO₂ level. After maintaining StO₂ at < 10% for 30 minutes, the cuff was released and measurements repeated after a 15-minute recovery period. The study was considered complete before achieving this endpoint if the subject no longer had a response to proximal nerve stimulation (complete conduction block), there was loss of pedal pulses, there was cyanosis of the foot, or if discontinuance was requested.

**Ischemia Measures**

The primary outcome measures were changes in neuro-muscular function measured by deep peroneal nerve conduc-
tion studies. Responses were recorded from two muscles: the extensor digitorum brevis (EDB) and the tibialis anterior (TA). Recording electrodes for the EDB (a distal muscle below the area of compression) were placed with the active electrode over the midpoint of the muscle and the reference electrode over the fifth metatarsal phalangeal joint. Recording electrodes for the TA (a muscle within the area of compression) were placed with the active electrode 8 cm distal to the tibial tuberosity and 2 cm lateral to the tibia, with the reference electrode placed 1 cm proximal to the bimalleolar line at the ankle. Stimulation was applied to the peroneal nerve at the fibular head, with recordings from the TA used to determine muscle function within the compartment, and from the EDB to measure nerve conduction through the compartment. Peroneal nerve stimulation was also applied at the ankle, distal to the area of compression, 8 cm proximal to the active recording electrode, over the extensor digitorum brevis. By comparing amplitude of the EDB response with stimulation from above and below the area of compression (i.e., knee vs. ankle stimulation), an estimate of the effect of compression on deep peroneal nerve function was derived (change in EDB amplitude-knee/ankle stimulation). Measurements of tibialis anterior muscle function were obtained because both nerve and muscle function may be affected by compartmental syndrome. The experimental preparation before the application of the pressure cuff is shown in Figure 2.

All stimuli were supramaximal (an increase in stimulus produced no further increase in action potential amplitude). The time latency between stimulus and the beginning of the compound muscle action potential of the EDB was measured from the proximal (above the compartment) and distal (extracompartamental) stimulation endpoints. The difference between these two times, divided into the distance between the two stimulation points, provided the velocity of the fastest conducting fibers, or the maximum motor nerve conduction velocity through the compartment. The amplitude (baseline to peak) for EDB recordings at each stimulation site was measured at each 15-minute measurement interval. For TA recordings, which had complex waveforms, area under the negative peak was used rather than amplitude. Baseline electrophysiologic measurements were obtained in triplicate, and the true baseline was taken as the average of these three measurements. The temperature of the foot and leg was monitored throughout the study. A 10% to 50% decrease from baseline in EDB amplitude with knee stimulation was considered mild ischemia; a decrease between 51% and 90% was considered moderate ischemia; and a reduction greater than 90% was considered severe ischemia.

Cutaneous sensibility testing in the peroneal nerve distribution (web space between first and second toes) was performed using Semmes-Weinstein monofilaments.16,17 These consist of a set of 20 nylon filaments of constant length and a progressively increasing diameter from 0.064 to 1.143 mm, which are pressed perpendicularly against the skin to the point of bending. Baseline sensation is established by using progressively larger filaments until the subject can sense the pressure of the filament before it bends. Testing was repeated at each 15-minute measurement interval, using progressively thicker monofilaments as needed. Monofilament testing provides a more controlled, objective, and reproducible test for detecting early peripheral sensory nerve dysfunction than standard two-point discrimination.18,19 Ischemia was defined as loss of three levels of monofilament sensitivity. Finally, at each measurement interval the subject was asked to subjectively quantify their level of pain using a serial 100-mm visual analog scale. An increase from baseline of more than 25% was considered as evidence of ischemia.
Regression methods were used to assess the associations between StO₂ and perfusion pressure with ischemia measures. To account for the correlated nature of the repeated measures taken on each subject, the generalized estimating equations methodology was used, with ischemia measures as the dependent variables and each subject as a cluster. Independent variables included StO₂, the individual’s calf circumference, and the “order,” where order indicated whether the measurements were made at the 15-minute or 30-minute interval at each target StO₂ level.

To compare the accuracy between StO₂ and perfusion pressure as diagnostic tools, receiver operating characteristic (ROC) curves were constructed to measure the power of each of these two tests to identify populations with and without ischemic characteristics (EDB amplitude decrease >10%, >50%, and >90%). The curves were constructed by plotting the true-positive fraction (sensitivity) against the false-positive fraction (1 − specificity), providing an infinite series of likelihood ratios. A cut-point for StO₂ was selected to define a positive test, then the corresponding sensitivity and specificity were calculated according to each predefined ischemia definition. ROC curves for perfusion pressure were performed similarly. Because of the skewed distribution of perfusion pressure, percentiles of data were chosen as the cut-points for defining positive tests, for example, perfusion pressure (4 [5th percentile], or 9 [10th percentile], or 12 [15th percentile]).

### RESULTS

The study was terminated after 15 subjects. There were seven men and eight women, with a median age of 25 years (range, 19–34 years) and median calf circumference of 39 cm (range, 35–49 cm). Eleven subjects completed the entire 135-minute protocol, and four met early discontinuance criteria because of total block of nerve conduction. Table 1 displays the StO₂, calculated compartment pressure, and perfusion pressure at each 15-minute study interval.

### Statistical Analysis

Tables 2 and 3 illustrate percent changes from baseline in electrophysiologic measurements, shown at 15-minute intervals. The change from baseline of EDB amplitude demonstrated mild and moderate ischemia at StO₂ values of 38.6% ± 7.1%, and 19.1% ± 2.2%, respectively. During the 45-minute time period during which these ischemic changes occurred, there were minimal changes in compartment pressure (73.1 ± 4.2 mm Hg to 78.6 ± 4.0 mm Hg) and perfusion pressure (23.2 ± 8.2 mm Hg to 16.5 ± 7.3 mm Hg), suggesting that duration of ischemia is an important variable. Although the maximum change in EDB amplitude was only 73.2% (Table 2), this value should be interpreted with caution because data from subjects that achieved complete block (n = 4 [27%]) were only recorded until complete block occurred.

Changes in cutaneous sensitivity, defined by a need to increase monofilament fiber size by three levels, occurred at an StO₂ of 3.1% ± 2.4%. Visual analog pain scale increased by 25% when StO₂ was 19.1% ± 2.2%. As shown in Table 4, there was a highly significant association between both StO₂ and perfusion pressure for all ischemia measures. There was...
a nonsignificant trend relating larger calf circumference with less ischemia. The 15- versus 30-minute order was associated only with the visual analog scale score (p = 0.04). Because of the skewed EDB amplitude-knee/ankle ratio and visual analog scale scores, these two variables were also analyzed in logarithmic scales, and there were no qualitative differences in results.

ROC curves (Figs. 3 and 4) demonstrated that the StO₂ curves were above those of perfusion pressure, indicating a higher sensitivity for StO₂ when the specificity was the same. For example, the curve depicting >50% change in EDB amplitude demonstrates that when specificity was 83% for StO₂ and 84% for perfusion pressure, the sensitivity was 85% for StO₂ and 56% for perfusion pressure (p = 0.02). Similarly, when the specificity for both StO₂ and perfusion pressure was 72%, the sensitivity of StO₂ was 94%, compared with 76% for perfusion pressure (p = 0.04). Curves determined on the basis of >90% decrease were not informative, because there were only 12 such occurrences.

**DISCUSSION**

The primary finding of this study was that StO₂ was significantly related to the ischemic conditions, both as raw measurements and as predefined ischemic conditions. Similar relationships existed between perfusion pressure and the ischemic outcomes. However, the ROC analyses indicated that StO₂ has a higher sensitivity when compared with perfusion pressure at the same specificity.

Elevated tissue pressure compromises neuromuscular function, producing the first symptoms of compartmental syndrome: pain and paresthesias, and paralysis. We therefore believe that neuromuscular physiologic measurements are the most appropriate endpoints for studies comparing orthopedic equipment used for the detection of compartmental syndrome.

The results of this study are consistent with our previous animal studies using NIRS. In an animal model of compartmental syndrome, StO₂ was a better predictor of loss of nerve-stimulated plantar dorsiflexion than perfusion pressure. A subsequent study using severely hypotensive and hypoxemic animals demonstrated that detection of compartmental syndrome using NIRS was not confounded by severe shock, and compartmental syndrome occurred at a lower compartment pressure than in nonshocked animals, consistent with the need to base diagnostic tests on tissue oxygenation, rather than measurements of tissue pressure.

A compartmental syndrome is defined as compromise of the circulation and function of tissue by increased tissue pressure. Thus, by definition, it may be modeled by any means of locally increasing tissue pressure. We believe our model is a valid one for the study of compartmental syndrome. The foundational studies leading to our current understanding of the pathophysiology of compartment syndrome all used the external calf compression model.

The compartmental syndrome encountered in patients, however, does present additional clinical considerations, including variations in ischemic times, the mechanical effects of crush injury to muscle resulting from the kinetic energy that caused the fracture, intramuscular hematoma, subcutaneous tissue, and muscular edema, and in some patients, isch-

### Table 3 Mean Changes in Tibialis Anterior Area and Nerve Conduction Velocity<sup>a</sup>

| Time (min) | No. of Patients | Δ TA Area (%) | Δ Nerve Conduction Velocity (%) |
|-----------|----------------|---------------|---------------------------------
| 15        | 14             | −7.0 (4.7)    | −1.0 (2.9)                      |
| 30        | 14             | −5.2 (5.3)    | −2.4 (3.3)                      |
| 45        | 14             | −11.9 (20.2)  | −2.8 (4.0)                      |
| 60        | 14             | −3.8 (14.9)   | −5.2 (3.8)                      |
| 75        | 14             | −8.0 (20.7)   | −7.4 (6.8)                      |
| 90        | 14             | −9.2 (24.2)   | −7.8 (7.1)                      |
| 105       | 14             | −16.6 (31.2)  | −12.9 (13.0)                    |
| 120       | 10             | −21.9 (26.9)  | −10.2 (9.4)                     |
| 135 (recovery) | 14 | 14.9 (13.6)   | −6.4 (7.8)                      |

<sup>a</sup> Standard deviations in parentheses. One subject did not have this measurement because of technical difficulties.

### Table 4 Generalized Estimated Equation: Estimated Change in EDB, TA, and Other Ischemia Variables Corresponding to Every Decrease of 20% for StO₂, and for Every 20 mm Hg Decrease in Perfusion Pressure<sup>a</sup>

<table>
<thead>
<tr>
<th>Ischemia Measure</th>
<th>Effect of StO₂ (SE)</th>
<th>p Value</th>
<th>CPP (SE)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>EDB amplitude</td>
<td>−1.0 (0.1)</td>
<td>&lt;0.001</td>
<td>−0.7 (0.08)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>EDB amplitude-knee/ankle</td>
<td>−12.9 (2.0)</td>
<td>&lt;0.001</td>
<td>−9.2 (1.4)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>TA area</td>
<td>−2.9 (0.9)</td>
<td>0.002</td>
<td>−2.7 (0.84)</td>
<td>0.001</td>
</tr>
<tr>
<td>Nerve conduction velocity</td>
<td>−1.0 (0.2)</td>
<td>&lt;0.001</td>
<td>−0.6 (0.2)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Semmes-Weinstein</td>
<td>0.8 (0.2)</td>
<td>&lt;0.001</td>
<td>0.7 (0.2)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Visual analog scale</td>
<td>9.8 (1.8)</td>
<td>&lt;0.001</td>
<td>6.9 (1.3)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

<sup>a</sup> EDB amplitude is in millivolts, nerve conduction velocity in milliseconds.
emia-reperfusion injury. These factors cannot be reproduced in a study on human volunteers. Prospective clinical studies performed on patients with the syndrome are needed to confirm the external validity of our findings.

Compartment pressure was calculated, rather than measured. Previous studies have directly measured compartment pressure while applying an air splint around the calf to determine how pressure applied outside of a limb is distributed within tissue. In general, the intramuscular pressure measured when external pressure is applied is equal to the external pressure plus normal intramuscular pressure (8–10 mm Hg) before external pressure is applied.13 More recent data indicate that the increment in measured pressure exceeds the applied pressure by a factor ranging from 1.02 when pressure is measured using the infusion technique, to 1.04 when using an intramuscular wick catheter.14,15 If external pressure did not strongly correlate with internal pressure within a limb, standard sphygmomanometric measurement of blood pressure would be uninterpretable.24 We therefore believe that our methodology of calculating compartment pressure was sound.

Duration of increased tissue pressure appeared to be a factor in producing compartmental syndrome, and time was not accounted for in this analysis. We could not analyze the effects of time because time was confounded by StO₂ since, by study design, StO₂ values were decreased at specific time intervals. However, for purposes of a study to determine the sensitivity and specificity of a test to detect the presence of ischemia, the manner with which ischemia developed is immaterial. A potential advantage of StO₂ is that it provides a measure of ischemia at a particular moment. A compartment pressure of 40 mm Hg may not produce ischemia for several hours, and a randomly obtained “spot check” measurement of tissue pressure may therefore be less useful in predicting the presence of compartmental syndrome because the duration of increased pressure would generally be unknown.

The consequences of missed compartmental syndrome are devastating. Tissue pressure measurements have failed to completely resolve this problem, partly because interpretation of results is difficult because of the variety of factors that contribute to ischemia, making it difficult to establish a critical pressure at which blood flow becomes critically compromised.23,25 There also appears to be considerable patient-to-patient variation in susceptibility to increased tissue pressure, which further confounds the diagnosis of ischemia.

In a study of 42 patients at risk for compartment syndrome who were continuously monitored using the infusion technique, no patient with a compartment pressure of 45 mm Hg or less developed the syndrome, and all of those with pressure greater than 60 mm Hg did.13 Of the seven patients with pressures between 45 and 60 mm Hg, five developed compartmental syndrome, and two did not. This suggests that there is no critical pressure that can consistently predict the need for fasciotomy, and direct measurement of tissue oxygenation may inherently be more accurate than measurements of pressure.

Another factor affecting pressure measurements is concurrent circulatory shock in trauma patients. Skeletal muscle Po₂ may decrease to as low as 1 to 5 mm Hg during hypotension.26 One study measured technetium-99m uptake in dogs, and noted that external pressure of 25 mm Hg in a hypotensive animal produced the same effects as a pressure of 40 to 50 mm Hg in a normotensive one. Measurement of tissue oxygenation is not confounded by shock, but instead, takes it into account, as demonstrated in our previous work.12

The anterior compartment of the leg is the least compliant and most susceptible to compartmental syndrome, and is easily monitored by NIRS. One of the potential weaknesses of current NIRS devices is that the depth of penetration precludes measurement of the deep posterior compartment. Future improvement in illumination and detector sensitivity

![Fig. 3. ROC curves comparing tissue oxygen saturation (StO₂) with perfusion pressure (PP) for detecting ischemia defined as extensor digitorum brevis peak amplitude decrease greater than 10% from baseline.](image1)

![Fig. 4. ROC curves comparing tissue oxygen saturation (StO₂) with perfusion pressure (PP) for detecting ischemia defined as extensor digitorum brevis peak amplitude decrease greater than 50% from baseline.](image2)
may enable interrogation of deeper tissues. Isolated involvement of the deep compartment is unusual, and other means of continuously monitoring this compartment are unavailable. All four compartments are usually exposed to the same insult, and involvement of one compartment is usually associated with impending involvement of the remaining ones.

NIRS may prove to be useful in conditions such as acute arterial occlusion, which may cause muscle ischemia, although local tissue pressure remains normal. However, studies to demonstrate this have yet to be performed. Other potential advantages of NIRS are that it is noninvasive and may be used continuously, enabling the clinician to detect an evolving compartmental syndrome, and to institute measures to increase perfusion to the lower extremity by manipulating systemic arterial pressure, removing compressive dressings, and avoiding elevation of the leg. Finally, invasive compartment pressure monitoring must be performed by a physician, but a nurse can use continuous NIRS at the bedside.

The literature clearly depicts the limitations of relying on tissue pressure measurements to detect compartmental syndrome, which frequently leads to delayed fasciotomy, with serious sequelae. In one study, the complication rates for early and late fasciotomies were 4.5% and 54%, respectively.27 Eleven of 24 extremities with delayed fasciotomy developed infections, half of which led to amputation. Another study showed infection rates of 7.3% and 28% respectively.27 Eleven of 24 extremities with delayed fasciotomy developed infections, half of which led to amputation. Another study showed infection rates of 7.3% and 28% for early compared with delayed fasciotomy.1 Because of the devastating consequences of delayed diagnosis, physicians often perform prophylactic, or expectant fasciotomy. However, a recent study performed long-term follow-up on patients who underwent this procedure.28 Complete vascular examinations demonstrated that fasciotomy places patients at a significant risk for development of chronic venous insufficiency. Performance of fasciotomy in patients who would not have gone on to develop the syndrome can no longer be considered a benign procedure.

In summary, the improvement in sensitivity and specificity demonstrated by NIRS may help clinicians to avoid delayed or unnecessary fasciotomy. We conclude that prospective clinical trials of NIRS as a means of detecting compartmental syndrome in at-risk patients should be performed.

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REFERENCES


**DISCUSSION**

**Dr. Marc A. Levison** (Phoenix, Arizona): Dr. Maier, Dr. Shackford, members, and guests, this article is now at or almost the above the fourth in a continuing series from Dr. Gentilello and his colleagues on the application of near-infrared spectroscopy to the challenge of early diagnosis of posttraumatic ischemia.

This is a technology with which we have all gained familiarity in the past decade. Its most common application, pulse oximetry, measures the light absorption of hemoglobin at two wavelengths, 660 nm and 940 nm, in arterial blood using a detection system that focuses on the pulsatile fraction of hemoglobin within the probe’s detection field.

At these wavelengths, biologic tissue such as skin, subcutaneous tissue, and bone are relatively transparent to the infrared light energy. The light is readily absorbed, however, by chromophores such as oxygenated and deoxygenated hemoglobin.

This technology has also been adapted and is widely used for pulmonary artery mixed venous and jugular venous oximetry. That technology differs somewhat in that these catheters measure reflected, rather than transmitted, light and because they have no reason to limit their region of interest to the pulsatile portion of hemoglobin.

You have had the opportunity at this meeting, in the booth, to see the apparatus that Dr. Gentilello was referring to, demonstrated. In a presentation at this meeting 2 years ago, Dr. Gentilello began to apply this technology to the study of compartment syndromes.

As he has stated in a pig hind-leg model, they demonstrated that oxyhemoglobin saturation fell as compartment pressures rose during colloid infusion. These changes correlated with loss of muscle twitch.

In addition, oxyhemoglobin concentration and muscle twitch returned after fasciotomy. Using that same model, they reported that whereas hypovolemic shock and hypoxemia resulted in a measurable fall in oxyhemoglobin concentration, subsequent increased compartment pressure was easily distinguished using this technology. Oxyhemoglobin saturation fell to 65% after hemorrhagic shock and hypoxemia. With the addition of elevated compartment pressure, the concentration fell to 15%.

This contrasts, interestingly, to the clinical study of Dr. Cocanour and Moore of this organization, who showed in a clinical trial measuring deltoid oxyhemoglobin concentration that their levels were only 15% in unresuscitated trauma patients.

Dr. Gentilello now presents data on the application of this technology to human volunteers. Fifteen volunteers had oximetry of their lower extremity anterior compartment, measurement of deep peroneal nerve conduction, cutaneous peroneal nerve sensitivity, and pain assessment using an 8-inch blood pressure cuff inflated to approximately 70 mm Hg for 2 hours. Cuff pressure was adjusted every 30 minutes to achieve incremental decreases of oxyhemoglobin from a baseline of 86% to less than 10%. Some patients also required calf elevation to achieve their endpoints. Moderate reductions of peroneal nerve conduction occurred at an oxyhemoglobin saturation of 40% and further reductions occurred at 20%. During the 45 minutes these levels were maintained, compartment pressure was estimated to be 73 to 78 mm Hg. Pain did not develop, however, until saturations dropped to 20%. On the basis of this experiment, the Harborview group postulates that an oxyhemoglobin saturation of less than 40% may represent a threshold for ischemia. I have several questions for Dr. Gentilello.

First, it is generally agreed that muscle ischemia begins at a compartment pressure of approximately 40 mm Hg because rising tissue pressure impedes flow of oxygenated fluids across capillary beds. In this experiment, ischemia was not observed until compartment pressure exceeded 70 mm Hg. You have noted that generally compartment syndromes will develop when pressures exceed 45 mm Hg. Can you explain this difference? Second, wide ranges of oxyhemoglobin saturations from 60% to less than 10% were observed over a very narrow range of compartment pressures in this experiment. Do you believe that this observation accurately reflects clinical compartment syndromes? Do compartment syndromes develop over these narrow ranges of compartment pressures? Third, because pain is usually considered an early warning of impending compartment syndrome in alert patients, how do you interpret the observation that oxyhemoglobin saturation had to fall to less than 20% before the objective complaint of pain developed? Fourth, how does the presence of tissue hemorrhage and hematomas in a fractured extremity monitored with oximetry affect the measurement of hemoglobin saturation?

Fifth, in these injured extremities, since the depth of penetration of the probe is only about 20 to 25 mm, will subcutaneous edema affect the ability to sample adequate tissue beds for oxy- and deoxyhemoglobin?

Finally, have you taken this technology to the bedside in the intensive care unit? Have the observations of Dr. Cocanour and Moore affected your thinking regarding the use of this technology to monitor for compartment syndrome?
Again, I enjoyed the opportunity to review this manuscript.

Dr. Carl J. Hauser (Newark, New Jersey): No one could be more convinced than myself that the proper measurement of tissue oxygenation is tissue oxygenation.

I am concerned, though, with this technique with respect to spatial resolution. How does this technique respond in the presence of a localized area of ischemia or compartmental hypertension, which in the lower extremity can be important?

For instance, if you have an isolated deep posterior compartment or peroneal compartment syndrome, does this technique miss the trees for the forest? Can you detect localized compartment or peroneal compartment syndrome, does this hypertension, which in the lower extremity can be important?

Presence of a localized area of ischemia or compartmental hypertension, which in the lower extremity can be important?

Dr. Larry M. Gentilello (closing): Thank you, Dr. Levine. Your first comment was that ischemia generally occurs at compartment pressures of 40 mm Hg in the clinical arena, but in this experiment compartment pressure reached 70 mm Hg.

Since we were using human volunteers and discomfort was involved, the experiment had to be completed within a reasonable amount of time. At a compartment pressure of 40 mm Hg, there are ischemic changes, but without a doubt, tissue damage occurs as a function of both compartment pressure and the duration of increased pressure.

At a compartment pressure of 40 mm Hg, we would have had to leave the cuff inflated for several hours before significant muscle ischemia occurred. That was not possible in a study on human volunteers. Since our goal was to determine whether NIRS can detect ischemic changes as measured by electromyography, we increased compartment pressure to reach targeted levels of tissue ischemia, or StO₂. It is interesting to note that StO₂ decreased over time without the need to manipulate the compartment pressure over a range of pressures. This confirms that time is a critical determinant of the development of ischemia. One of the potential disadvantages of pressure measurements is that the clinician making the initial measurement does not know how long the elevated compartment pressure has been present. A potential advantage of StO₂ is that it provides a measure of the degree of ischemia at that particular moment.

Time is the factor that allowed StO₂ to range from 60% to 10% over a narrow range of compartment pressures. Tissues became more ischemic over time, whereas compartment pressure was relatively constant.

You also mentioned that pain was not present until the StO₂ level was less than 20%. Pain is an early sign of compartmental syndrome. That does not mean that pain begins immediately on reaching a given compartment pressure. The development of ischemia is required. We used a visual analog scale and required that pain sensation go up by more than three levels on a 1- to 10-point scale. It is interesting to note that there was no pain despite elevated compartment pressures as high as 70 mm Hg, until tissue ischemia was also present. Elevated compartment pressure alone was not sufficient to cause pain, probably because elevated compartment pressure does not mean that the tissues immediately become ischemic.

You asked about the effects of hematomas. We would not place the detector directly over a hematoma. The lower leg compartments are relatively long and, although there may be regional pressure differences, the pressure within them should be relatively uniform. Tissue damage from compartmental syndrome is not isolated to a region where a hematoma is present.

As for the effect of edema on the readings, as you mentioned, skin, bone, and water are transparent to near-infrared wavelengths. So although edema will increase the distance that the light must travel to reach the muscle, it would also increase the depth of penetration by the same amount because there would be no absorption of the light as it traversed the edematous tissue.

You asked about whether or not it has been brought to the bedside yet. There is currently an ongoing clinical trial, not at our institution, and I have no data on that.

Finally, you asked about the study performed at the University of Texas in Houston by Drs. Cocanour and Moore using deltoid muscle. They found that patients in shock had StO₂ levels of about 40% in the deltoid muscle. We have found the deltoid muscle to be an unreliable place for obtaining readings. Where the skin is thinner, such as the thenar eminence or over the anterior compartment, more reliable readings are obtained.

Dr. Hauser, you can have locally ischemic muscle because of a direct blow or impact to the muscle, but a compartmental syndrome usually means that there is increased pressure throughout that compartment, with resulting ischemia. If the measurement is made too far away from the area of interest, an area of discrete, focal ischemia could be missed. The same limitation applies to measurement of compartment pressure. If the area you choose to measure is not under increased pressure, the area of ischemia will be missed. The NIRS probe is noninvasive, and can more easily be used to take measurements from multiple areas.

As far as interrogating the deep posterior compartment, no, the probe cannot reach that, but generally, an increase in pressure in one compartment means impending increase in pressure in the remaining ones. An isolated deep posterior compartment syndrome is very rare, and one cannot reach that compartment anyway with tissue pressure monitoring.