QUANTATIVE EVALUATION OF MYOGLOBIN AND HEMOGLOBIN OXYGENATION DURING CONTRACTION USING NEAR-INFRARED SPECTROSCOPY

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Quantitative Evaluation of Myoglobin and Hemoglobin Oxygenation during Contraction using Near-Infrared Spectroscopy

Abstract

By

SABINA KUMAR

117 million people in the United States today suffer from a chronic disease associated with an insufficient oxygen delivery to muscle. Clinicians need a fast, easy, non-invasive way to measure oxygen saturation. Near-Infrared Spectroscopy (NIRS) is a non-invasive procedure whose signal reflects tissue oxygenation. We hypothesized however that near-infrared spectroscopy (NIRS) measures of hemoglobin and/or myoglobin O$_2$ saturation do not reflect the venous oxygen saturation because of the Hb and Mb contributions to the NIRS signal. A computational model previously developed to describe the dynamic and spatial changes of O$_2$ concentration in blood and tissue domains of contracting skeletal muscle was modified to quantify Hb and Mb contributions to the NIRS signal. In addition, the effect of blood flow kinetics in a normal and disease with NIRS signal was studied.
Introduction

The delivery of oxygen and potential role of insufficient oxygen delivery is very concerning when a clinician is dealing with diagnosing chronic diseases that are dependent on oxygen delivery to the skeletal muscle. Oxygen plays a critical role in energy metabolism in skeletal muscles. Skeletal muscles are voluntary muscles whose main role moves the bones and supports the skeleton through neural impulses that are sent from our brain to our muscle. Their main function occurs with contracting to facilitate movement of our skeleton. These contractions can vary to produce small precise actions to powerful fast movement. The decrease in oxygen delivery will lead to eventually inactivation of the skeletal muscles which could eventually lead to muscle atrophy and paralysis (1).

During dynamic muscle contraction there is an increase metabolic demand which is closely followed by an increase in blood flow kinetics. $\text{VO}_2$ kinetics relate to the oxygen uptake in transition from rest to exercise. The ATP demand in contracting muscles increases abruptly but the $\text{VO}_2$ response is relatively slow comparatively and therefore other energy producing metabolic pathways must be called on to meet the demand like an anaerobic or non-oxidative process(1).

An individual can alter their $\text{VO}_2$ kinetics by having a high-intensity exercise (bout 1) followed by subsequent high-intensity exercises (bout 2). The $\text{VO}_2$ kinetics will be faster in bout 2 compared with bout 1. Possible reasons for the increase $\text{VO}_2$ kinetics in bout 2 include: the accumulation of
lactic acid in bout 1 will increase blood flow which will thus make \( O_2 \) readily available to the muscle, high-intensity exercise could “prime’ the muscle mitochondria and thereby increase oxidative metabolism within the muscle cells themselves, or that high-intensity exercise favors the alteration of the pattern of muscle fibre recruitment during subsequent exercise(1).

\( O_2 \) supply to skeletal muscles are difficult to obtain during exercise, therefore a non-invasive method that continuously measures \( O_2 \) saturation in exercising muscles would be beneficial. Near-Infrared Spectroscopy (NIRS) is being used to estimate relative \( O_2 \) saturation. Previous research has shown close correlation between NIRS and venous \( O_2 \) saturation measurements as work rate has increased. Macdonald et.al compared femoral venous \( O_2 \) saturation (\( S_{f\text{vo}_2} \)) and NIRS measures of hemoglobin and myoglobin \( O_2 \) saturation (IR-\( \text{So}_2 \)) at the onset of leg-kicking exercises. The team measured NIRS measurements over the vastus lateralis muscle as well as drew blood simultaneously from the femoral artery and venous. The results indicated a rapid decrease in both \( S_{f\text{vo}_2} \) and IR-\( \text{So}_2 \) at the onset of exercise. However, IR-\( \text{So}_2 \) increased significantly after the first minute of exercise and \( S_{f\text{vo}_2} \) remained at low levels throughout exercise. Therefore, Macdonald and team concluded that NIRS does not provide a reliable estimate for IR-\( \text{So}_2 \) measurements (2). Costes et. al and team concluded during a steady-state cycling exercise experiment that \( S_{f\text{vo}_2} \) paralleled relative oxygenation changes measured via hypoxia. Normoxia did not follow this pattern. The team did not validate the NIRS measurements with effluent venous \( S_{f\text{vo}_2} \)(3). McCully et. al team did not understand why under some conditions did the NIRS-measured oxygen saturation increase at a time while the measured venous oxygen saturation remained constant(4). There are conflicting viewpoints on NIRS oxygenation and therefore more research must be done before this becomes a gold standard within a clinical setting.
Before the use of NIRS measurements is wide-spread in the clinic more research must be done to understand if there is or is not a difference between Venous $O_2$ measurements and NIRS measurements.

Near-Infrared Spectroscopy (NIRS) is applied to study tissue oxygenation in brain and skeletal muscle under a variety of experimental conditions. NIRS signal reflects the pattern of tissue oxygenation that can indicate limitations associated with muscle $O_2$ delivery and utilization. Although NIRS is currently used to monitor cerebral oxygenation, it cannot distinctly characterize muscle perfusion and oxygenation\(^5\). The NIRS output signal provides a measure of oxygenation that depends on the relative contributions of myoglobin (Mb) and hemoglobin (Hb). These contributions, however, cannot be distinguished since their absorbance spectra are similar. Mancini et al. (1994) argued that on one hand it has been assumed that both the NIRS signals at rest and changes in the signal are derived from Hb(>90%) while MB is thought to contribute minimally (<10%) to the signal. However, Trans et al (1999), Richardson et al. (2001), and Mole et al. (1999) all suggested that Mb may be a predominate contributor to the NIRS signal. Additionally, oxy-Hb and oxy-Mb differ with respect to saturation and dynamic responses, which depend on muscle $O_2$ utilization and delivery rates\(^6\). Therefore, it is possible to differentiate their roles by simulation of the experimental NIRS signal using a mechanistic model that describes the underlying transport and metabolic processes occurring in contracting skeletal muscle.

The purpose of this paper is to find out if Venous $O_2$ and NIRS follow similar or different kinetics to see if in the future NIRS could take over Venous $O_2$ in a clinical setting. In addition, quantification of Mb and Hb from the NIRS signal will be sought out to explain the kinetics of the NIRS signal. We hypothesize that the kinetics of Venous $O_2$ will not follow NIRS because of the
contribution of Hb or Mb to the NIRS signal. To test our hypothesis a computational model (previously developed by Dr. Lai and team) will be used to simulate oxygen kinetics during muscular contraction. The model's input parameters will be modified to simulate blood flow kinetics and uptake kinetics of Hernandez's and teams experimental data. The experimental data of Hernandez and team was used because there was blood flow and uptake experimental data as well as corresponding NIRS oxygenation and deoxygenation data. In addition, Hern et. al experimental data came in two forms Bout 1 and Bout 2. Hern et. al and team studied the effects of a priming contractile bout on oxygen uptake kinetics in an oxidative skeletal muscle. The muscle was stimulated in 12 different dogs via their sciatic nerves at a rate of 2 contractions per 3 seconds for two 2-min bouts separated by 2 minutes.

Methods

A computational model previously developed to describe the dynamic and spatial changes of O\textsubscript{2} concentration in blood and tissue domains of contracting skeletal muscle will be used with additional equations describing the contribution of Hb and Mb contributions to the NIRS signal. This model was developed by Dr. Lai and team from Case Western Reserve University and is based upon dynamic mass balances for O\textsubscript{2}, ATP, and PCr and the equations in the model account for changes in cellular ATPase, oxidative phosphorylation, and creatine kinase fluxes in skeletal muscle during exercise and cellular respiration depends on [ATP] and [O\textsubscript{2}].

The input parameters to the computational model were modified using Hern et. al’s experimental data. The input parameters were modified so our computational model can be used to predict muscle NIRS measurements under different blood flow (e.g. high, low) and Vo2
conditions. The input parameters were found by fitting the blood flow and uptake experimental data with the following monoexponential function of this type:

\[ y(t) = Y_{\text{Bas}} + A[1 - e^{-(t-\text{TD})/\tau}] \]

In the above equation, \( Y_{\text{Bas}} \) indicates the baseline value obtained at rest before the contraction onset which typically indicated the average of the previous 5 seconds before the onset of contraction. \( A \) was the amplitude between the \( Y_{\text{Bas}} \) and the asymptote of the primary component. \( \tau \) is the time constant of the function and \( \text{TD} \) is the time delay. Fitting was accomplished via a nonlinear least squares procedure using OriginPro 2015 64 Bit (OriginLab, One Roundhouse Plaza, Northampton, MA). The directly measured baseline \( Q \) or \( \text{Vo}_2 \) was fixed while \( A \), \( \text{TD} \), and \( \tau \) were allowed to float. The best fit was obtained by running iterations until the fit converged and the chi-square tolerance level was reached. The minimizations of the sum of squares of errors were also sought out. The window that allowed for the above as well as a stable \( \tau \) value is where the parameters inputed into our computational model stemmed from. The input parameters found in the equation were directly used in our computational model. These input parameters include: Resting Blood Flow, Gain of Blood Flow, Time Constant of Blood Flow, Time Delay of Blood Flow, Uptake at Rest, and Gain of Uptake.

Simulations of our computational model, with our modified input parameters from fitting Hern et. al experimental data, were matched with the experimental data of Blood Flow and Uptake for Bout 1 and Bout 2 from Hern et. al. The model was validated when the kinetics of the experimental data matched with the output of our computational model.
The difference in \([O_2]\) between the arterial and venous blood \([C(a-v)]\) experimental data was calculated using Fick’s Principle \(V_{O_2} = Q \cdot C(a-v)O_2\). Where \(Q\) represents Hern et. al experimental data for blood flow on response for 120 seconds and \(V_{O_2}\) respresents the mean contraction-by-contraction uptake on response for 120 seconds from Hern et. al’s data. The experimental data calculated via Fick’s Principle as well as the ouput for the computational model for AV difference was plotted for Bout 1 and Bout 2.

Arterial \([O_2]\) was mainted constant for all the dogs in Hern et. al and was 0.1205 mM. The average arterial \([O_2]\) for all the dogs was used to calculate the experimental data Venous \([O_2]\). Venous \([O_2]\) was calculated by the following equations:

\[
Venous\ [O_2] = C(a-v) - \text{Arterial} \ [O_2]
\]

The value of 0.1205 mM was used as the Arterial \([O_2]\) in our computational model. Bout 1 and Bout 2 experimental data vs. simulation output were plotted.

Once the model was validated matching the experimental data, the model was used to understand the NIRS signal. NIRS signal was computed in our computational model. Please refer to Dr. Nicola Lai’s paper titled “Modeling oxygenation in venous blood and skeletal muscle in response to exercise using near-infrared spectroscopy” published in the Journal of Applied Physiology in 2009 for the methodology how NIRS was calculated in our computational model.

The hemoglobin and myoglobin contribution for bout 1 and bout 2 were extracted from the code and plotted along-side the total NIRS signal. The percent oxygenation and deoxygenation of Mb and Hb were derived from the following equations:

\[
Mb\% = \frac{TotMbOxy}{TotHbMbOxy}
\]
\[
Hb\% = \frac{TotHbOxy}{TotHbMbOxy}
\]
TotMbOxy represents the total contribution of myoglobin to oxygenation of the tissue while TotHbOxy represents the total contribution of hemoglobin to the oxygenation of the tissue. TotHbMbOxy represents the total NIRS-oxygenation signal. These were all simulated by our computational model.

\[ Mb\% = \frac{\text{TotMbDeoxy}}{\text{TotHbMbDeoxy}} \quad \text{Hb}\% = \frac{\text{TotHbDeoxy}}{\text{TotHbMbDeoxy}} \]

TotMbDeoxy represents the total contribution of myoglobin to deoxygenation of the tissue while TotHbOxy represents the total contribution of hemoglobin to the deoxygenation of the tissue. TotHbMbOxy represents the total NIRS-deoxygenation signal. These were all simulated by our computational model.

The last investigation we did to understand the NIRS signal was to see the effect of blood flow kinetics to the NIRS signal. This was done by using our computational model but using only our modified input parameters for bout 1. The time constant was changed in the input parameters file to 12, 19, 25, and 48 sections for us to interpret.

Methods: Analysis

Three different types of analysis were conducted on our results which include: tau constant calculation, paired t-test, and normalization.

Exponential models can be thought as a process that depends on a rate of growth or decay at a particular time which can be related to the quantity present. An exponential process is said to reach steady state when four time constants have elapsed. A fitting of bout 1 and bout 2 for measurements obtained in this experiment will be fitted with a monoexponential function with an exponential growth or decay and a time constant will be calculated for use to compare
bout 1 and bout 2 kinetics. Similar time constants will yield to conclusions of similar kinetics while different time constants will yield to conclusions of different kinetics. Individuals with advanced cardiovascular or pulmonary disease can have a time constant up to 90 seconds.

Paired-T test on simulations and results were conducted by taking the difference between bout 1 and bout 2 of the experimental data or simulated data and running a 1-sample t test. The null hypothesis was that bout 1 and bout 2 were equal and the alternative hypothesis was that bout 1 and bout 2 were not equal. Obtaining a p-value of less than 0.05 made us conclude that bout 1 and bout 2 were different.

Normalization of the simulations and experimental data were done to get a clearer picture of the different kinetics between bout 1 and bout 2. Normalization was completed by taking the first value and the steady state value and having OriginPro 2015 run a normalization from [0 1].
Results

Parameter estimation and model validation. By fitting the experimental data obtained from Hern et. al. and team to a monexponential fit, the parameters inputted in the model for Bout 1 and Bout 2 were found. Table 1 below shows the values of model parameters that depend on experimental data that were found from the fitting.

Table 1: Values of Model Parameters that depend on Experimental Data Used in Computational Model

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Units</th>
<th>Bout 1</th>
<th>Standard Error</th>
<th>Bout 2</th>
<th>Standard Error</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vo2 at Rest</td>
<td>[mmol/L*min]</td>
<td>0.125</td>
<td>0</td>
<td>0.297</td>
<td>0</td>
</tr>
<tr>
<td>Gain Vo2 at Moderate Condition</td>
<td>[mmol/L*min]</td>
<td>6.17</td>
<td>0.07</td>
<td>6.2</td>
<td>0.04</td>
</tr>
<tr>
<td>Blood Flow at Rest</td>
<td>[L bl/Lmus*min]</td>
<td>0.156</td>
<td>0</td>
<td>0.219</td>
<td>0</td>
</tr>
<tr>
<td>Gain Blood Flow</td>
<td>[L bl/Lmus*min]</td>
<td>0.699</td>
<td>0.008</td>
<td>0.63548</td>
<td>0.005</td>
</tr>
<tr>
<td>Time Constant Blood Flow</td>
<td>[min]</td>
<td>0.324</td>
<td>0.02</td>
<td>0.15949</td>
<td>0.006</td>
</tr>
<tr>
<td>Time Constant Vo2</td>
<td>[min]</td>
<td>0.162</td>
<td>0.012</td>
<td>0.1831</td>
<td>0.006</td>
</tr>
<tr>
<td>Delayed Time</td>
<td>[min]</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Time Constant Vo2</td>
<td>[sec]</td>
<td>9.72</td>
<td>0.72</td>
<td>10.986</td>
<td>0.36</td>
</tr>
<tr>
<td>Time Constant Blood Flow</td>
<td>[sec]</td>
<td>19.44</td>
<td>1.2</td>
<td>9.5694</td>
<td>0.36</td>
</tr>
</tbody>
</table>

As stated in the methods section, Vo2 and Q at rest were fixed values and therefore there was no standard error. The delayed time was in the ten thousandths place and therefore was approximated to 0 in the parameters inputted into the code.

Arterial O2, Standard HCT, Muscle Volume, Arterial pH, and Total [Hb] were 20.9 ml/dl, 0.4837, 7.41, and 15.6 g/dl respectively.
Fig. 1. Comparison between model simulations and experimental data for Bout 1 and Bout 2 in Cannie Muscle (Average of 12 dogs) these comparisons validated the computational model used for this study. A. Comparison of Experimental and Simulated data for Blood Flow Kinetics ($Q$) under Bout 1 & Bout 2 Conditions B. Comparison of Experimental and Simulated data for Uptake Kinetics ($V_{O_2}$) under Bout 1 & Bout 2 Conditions C. Comparison of Experimental and Simulated data for AV Difference ($C(a-v)$) under Bout 1 & Bout 2 Conditions.
Fig. 2. Comparison between Venous Oxygenation and NIRS measurement of Oxygenation Saturation. A. Comparison between experimental data and model simulations for Venous Oxygenation under the conditions of Bout 1 and Bout 2. B. Comparison between normalized experimental data and model simulations for Near-Infrared Spectroscopy Muscle Oxygenation Data under the conditions of Bout 1 and Bout 2.

Fig. 3. Comparison between normalized experimental data and model simulations for Near-Infrared Spectroscopy Muscle Deoxygenation Data under the conditions of Bout 1 and Bout 2.
Fig. 4. Model Simulation of the Breakdown of Myoglobin and Hemoglobin Contributing to the NIRS signal for Bout 1 and Bout 2 during muscular contraction. A. Simulated Myoglobin and Hemoglobin Oxygenation Contribution to the Total NIRS Oxygenation Signal for Bout 1 and Bout 2 during muscular contraction. B. Simulated Myoglobin and Hemoglobin Deoxygentation Contribution to the Total NIRS Oxygenation Signal for Bout 1 and Bout 2 during muscular contraction.
Fig. 5. Percentages of Hemoglobin and Myoglobin contributing to the Near-Infrared Spectroscopy Muscle Oxygenation from our modified computational model. A. Percentage of Hemoglobin contribution to the total NIRS Muscle Oxygenation Data from our modified computational model under the conditions of Bout 1 and Bout 2. B. Percentage of Myoglobin contribution to the total NIRS Muscle Oxygenation Data from our modified computational model under the conditions of Bout 1 and Bout 2.

Fig. 6. Percentages of Hemoglobin and Myoglobin contributing to the Near-Infrared Spectroscopy Muscle Deoxygenation from our modified computational model. A. Percentage of Hemoglobin contribution to the total NIRS Muscle Deoxygenation Data from our modified computational model. B. Percentage of Myoglobin contribution to the total NIRS Muscle Deoxygenation Data from our modified computational model under the conditions of Bout 1 and Bout 2.
Fig. 7. Effect of Blood Flow Kinetics (Tau=12, 19, 25, 48 seconds) on the NIRS-oxygenation signal for Bout 1. The NIRS signal is broken down in Myoglobin Contribution and Hemoglobin Contribution.

Figure 8: Comparison of the Normalized Venous Oxygen Saturation with a Normalized NIRS-Oxygenation for Bout 1 with a Tau of 25 and 48 seconds. Tau of 48 seconds represents a disease state while a Tau of 25 represents a normal state.
Discussion

Figure 1(a-b) demonstrates the validity of our computational model. The simulation for Bout 1 and Bout 2 match the blood flow kinetics (Figure 1a) and oxygen uptake (Figure 1b) from Hern et. al. experimental data. The blood flow kinetics of bout 2 are significantly speeded compared with bout 1 (time constant=9.6 ± 0.4 vs. 19.4 ± 1.2 seconds). Compared with the first bout the primary $\text{Vo}_2$ time constant(9.7±0.7 vs. 11.0 ± 0.4) are reduced for the second bout. Figure 1c shows the difference in oxygenation between the arteries and veins for Bout 1 and Bout 2. The time constant for bout 1 compared with bout 2 is 8.0 vs. 8.2 seconds. These time constants are very similar and therefore we can conclude that bout 1 and bout 2 do not have significantly different kinetics for $\text{C(a-v)}$.

Figure 2b and Figure 3 provides further evidence of the validity of our computational model. Hern et. al’s experimental data monitoring the muscle’s deoxygenation and oxygenation using Near-Infrared Spectroscopy matched with our computational model’s analysis of the muscle during contraction for both Bout 1 and Bout 2. The second bout is significantly slower than the first bout during muscular deoxygenation (7.2 ±2.2 vs. 4.4 ± 1.2 seconds). Hern et. al and team believes this is likely to arise from the rapid adjustment of convective $\text{O}_2$ delivery to the contracting muscle stemming from the fact that deoxygenation is a balance between $\text{O}_2$ delivery and $\text{O}_2$ extraction. The rapid adjustment of the muscle perfusion may also likely be a reason why the second bout has a greater change in oxyhemoglobin concentration.

Comparison of Figure 2a and Figure 2b shows that there is a difference between Venous $\text{O}_2$ and Near-Infrared Spectroscopy. Mathematically speaking, since there was no significant difference between the $\text{C(a-v)}$ for bout 1 and bout 2 one can expect to see the same trend in
the Venous O₂. The time constant for bout 1 and bout 2 are 10.8 vs. 9.2 seconds which demonstrates that venous oxygenation for bout 1 and bout 2 are similar. Figure 2b shows the non-invasive method for determining oxygen saturation via NIRS. Clinicians want to believe that this non-invasive method for determining oxygen saturation matches the invasive method of venous oxygenation however these graphs are proving their theory wrong. The time constants for bout 1 and bout 2 for oxygen saturation determined by NIRS are 11.4 to 16.2 seconds respectively. There is a significant difference between bout 1 and bout 2 oxygen saturation according to the NIRS measurements. Therefore, I am hesitant to recommend non-invasive oxygenation measurements in critical care settings. NIRS measurements are affected by different factors including “heterogeneity of blood flow and oxygen consumption distributions leading to non-uniform oxygenation in muscle capillary recruitment, the signal from a specific muscle region depends on fractional volume distribution of skin, adipose tissue, capillaries, small arterioles and venoules, and further more NIRS depends on the relative contributions to Mb and Hb to the output signal which NIRS methodology alone cannot distinguish. Lai et. al and team predicted that during exercise the relative contribution of hemeproteins may differ during exercise.

Figure 4 breaks down the Mb and Hb contributions to the total oxygenation and deoxygenation of the NIRS signal. Figure 4a demonstrates that Mb contribution is substantial in the signal compared with Hb in both bout 1 and bout 2. There is more oxygen extraction in bout 1 compared to bout 2 which is evident in the breakdown of Hb and Mb. Figure 4b the kinetics of Hb contribution is faster than the kinetics of the NIRS signal and the kinetics of Mb contribution is slower than the kinetics of the NIRS signal. The effect of slow and fast kinetics on
the contribution Hb and Mb can be demonstrated by reviewing Figure 6a and Figure 6b. Figure 6 breaks down the contribution of Hb and Mb as percentages of the total NIRS signal. Figure 6b shows an increase of percentage of Hb contribution at the onset of muscular contraction to about 1 min (47% to 65%) which is attributed to the faster kinetics of Hb contribution relative to the total NIRS signal. Figure 5c shows a decrease of percentage of Mb contribution at the onset of muscular contraction to about 1 min (53% to 35%) which is attributed to slower kinetics of Mb contribution relative to the total NIRS signal. To investigate the difference of blood flow kinetics on NIRS measurements the bout 1 case was isolated and simulations were conducted on different time constants. A time constant of 48 seconds was used to represent a person in a particular disease state with a very slow blood flow kinetics. From Figure 7 the tau of 48 seconds has a slower Hb oxygenation, Mb oxygenation, and total NIRS signal compared with the 12, 19, and 25 second time constants respectively. This figure also demonstrates that the higher the blood flow the faster the oxygen extraction will be. Figure 8 shows an important comparison of venous oxygenation and NIRS total oxygenation in disease vs. normal state. The NIRS signal is highly amplified for the disease state while the venous oxygenation does not show this amplification. Both the graphs show lower oxygen extraction for the disease state however a clinician might come up with a different diagnosis if he or she only looked at the amplified NIRS signal.

**Conclusion**

Physiologists and clinicians will be able to use this computational model in the future to investigate muscle oxygenation and metabolism in health and disease. Our hypothesis that venous oxygenation and NIRS measurements do not show the same kinetics was proven to be
true. This was supported by using a computational model and modifying the input parameters to represent experimental data of study that stimulated a skeletal muscle twice with 2 contractions per 3 seconds in two bouts separated by two minutes of rest. The model was validated with matching the blood flow and uptake kinetics of bout 1 and bout 2 with the experimental vs. simulated data. Further investigation was conducted using our computational model to understand why the kinetics of Venous $O_2$ and NIRS measurements do not match by looking at the specific Hb and Mb contributions. Mb was found to be a main contributor to the NIRS-oxygenation signal. The kinetics of the hemoglobin contribution after muscular contraction to the NIRS-deoxygenating signal moved faster than the signal itself which lead to an increase of 47 to 65 percent of hemoglobin contribution between 0.5 to 1 minute. Lastly, bout 1 was isolated to show the effect of blood flow kinetics using a normal and disease state. The NIRS signal was highly amplified compared with the venous oxygenation however both correctly showed that the disease state would reduce the oxygen extraction. Clinicians should always try to get a venous oxygenation reading if possible since kinetics of NIRS and Venous $O_2$ are different. More research into the NIRS signal specifically the myoglobin and hemoglobin contribution linked to a particular disease could lead to interesting uses of NIRS in a hospital setting.
References


