# **Chapter 4 Dorsiflexor Muscle Oxygenation During Low, Moderate and Submaximal Sustained Isometric Contraction**

#### **Adkham Paiziev, Martin Wolf, and Fikrat Kerimov**

**Abstract** Sustained isometric contractions of skeletal muscles produce intramuscular pressures that lead to blood flow restriction. Thus, we have the paradox of rising  $O<sub>2</sub>$  demand due to muscle activity and at the same time reduced blood flow. The aim was to assess muscle oxygenation during sustained isometric low (30%), moderate (60%) and submaximal [90% of maximal voluntary contraction (MVC)] contraction of the dorsiflexor muscle. Experiments were conducted on the dominant (right) leg of 8 male students (age  $19 \pm 2$  years, weight  $75 \pm 6$  kg). Tissue oxygen saturation  $(StO<sub>2</sub>)$  was recorded from the tibialis anterior using near-infrared spectroscopy. StO<sub>2</sub> was higher at  $30\%$  compared to both 60% and 90% MVC at all time points after the start of the exercise and higher at 60% than 90%. This indicates that the supply of  $O<sub>2</sub>$  did not keep up with its consumption. During arterial occlusion the minimal  $StO<sub>2</sub>$  reached 52%, which is significantly higher than  $StO<sub>2</sub>$  during 60% and 90% MVC. After each contraction there was a large and immediate hyperemic response, whose resaturation rate continuously increased from 30% to 60% to 90% MVC. The  $StO<sub>2</sub>$  resaturation rate was positively correlated with the MVC, indicating a vasodilation depending on the intensity of the exercise.

**Keywords** Skeletal muscle • exercise • Near Infrared Spectroscopy • Blood occlusion

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H.J. Halpern et al. (eds.), *Oxygen Transport to Tissue XXXIX*, Advances in Experimental Medicine and Biology 977, DOI 10.1007/978-3-319-55231-6\_4

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## **1 Introduction**

Sustained isometric contraction (SIC) of skeletal muscles produces intramuscular pressure (IMP) that restricts muscle blood flow (MBF) and limits  $O_2$  delivery to tissue [[1\]](#page-5-0). MBF plays a key role in regulating the intensity and type of muscle contractions  $[2]$  $[2]$ . A limited MBF due to SIC leads to fatigue due lack of  $O_2$  and nutrients. Thus, we have the paradox of rising  $O_2$  demand due to muscle activity and at the same time reduced MBF. To clarify this situation much research has been performed mainly on MBF [[3–](#page-5-2)[7\]](#page-5-3). However, during exercise there are few measurements of MBF by Doppler ultrasound. Previous studies indicate that complete occlusion of MBF occurs at 50–60% of maximal voluntary contractions (MVC) during SIC [[3,](#page-5-2) [4,](#page-5-4) [6](#page-5-5)]. MBF was not occluded at the level of the conduit artery during any contraction intensity [\[3](#page-5-2)]. Thus, our understanding of the oxygenation of the skeletal muscle in response to different intensities of SIC (low, moderate, submaximal MVC) is still limited.

Despite advantages of fMRI, PET and Doppler ultrasound only one paper has been devoted to measure the hemodynamic response of muscles to SIC [[3\]](#page-5-2) by near infrared spectroscopy (NIRS). In the near-infrared (NIR) spectrum (700–900 nm) light penetrates deeply into the tissue and oxyhemoglobin  $(O<sub>2</sub>Hb)$  and deoxyhemoglobin (HHb) are the strongest absorbers, while myoglobin (Mb) absorbs less. NIRS is an established optical technique to monitor concentration changes of  $O_2Hb$ ,  $HHb$ , total haemoglobin (tHb) and tissue oxygen saturation  $(StO<sub>2</sub>)$  in a variety of tissues [\[8](#page-5-6), [9](#page-5-7)]. NIRS instruments are non-invasive, small, and applicable in exercise physiology.

The aim was to assess changes in muscle oxygenation during low, moderate and submaximal SIC of the dorsiflexor muscle.

#### **2 Methods**

Eight male students of USIPC were included (age  $19 \pm 2$  years, weight  $75 \pm 6$  kg) and measured on the dominant (right) leg. The foot was placed in an in-house built isometric torso-dynamometer (DC-200, Russia). Tissue  $O_2$  saturation (StO<sub>2</sub>) was recorded from the tibialis anterior by NIRS (SenSmart™ Model X-100, NONIN). The sensor was placed over the belly of the tibialis anterior muscle of each individuals with spacing of optodes of 4 cm.

The experiment consisted of pre-exercise (rest) and three successive periods (each ~1 min) of sustained isometric contractions at 30%, 60% and 90% of MVC and separated by rest periods of 3 min (Fig. [4.1\)](#page-2-0). To determine the minimal  $StO<sub>2</sub>$  in the dorsiflexors an arterial occlusion was performed at the end of the experiment. The dorsiflexor muscle was selected, because both venous outflow and arterial inflow can be occluded by a proximal cuff. Without blood supply, the muscle metabolism depends on the  $O_2$  in capillaries and muscle cells. Consequently, the  $O_2Hb$ 

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**Fig. 4.1** Protocol of experiment (*top*) and example of  $StO<sub>2</sub>$  trace (*bottom*). During moderate and submaximal SIC, subjects were not able to perform the exercise for one whole minute

and  $StO<sub>2</sub>$  decrease, while HHb increases and tHb remains constant. After the occlusion a hyperemic response occurs, i.e. a rapid increase in  $O_2Hb$ , tHb and  $StO_2$ , while HHb is washed-out. From this procedure, we calculated  $O_2$  consumption, reoxygenation rate and the half-recovery times of the signals [[10\]](#page-5-8).

The recovery baseline (RB) value is the  $StO<sub>2</sub>$  value after stabilisation during the rest period following a test period. The performance baseline is the minimum StO2 value reached during SIC. For each individual the NIRS signal was normalized to its maximal value reached during reperfusion after cessation of occlusion. Differences in the  $StO<sub>2</sub>$ , desaturation rate (De) and resaturation rate (Re) between muscle contraction intensities were analysed by Student's t-test in the statistical software package Statistica for Windows (version 13).

## **3 Results**

Figure [4.1](#page-2-0) displays a typical measurement in one subject. The drop in  $StO<sub>2</sub>$  increased from low to moderate to submaximal SIC (group values: Table [4.1](#page-3-0)). Figure [4.2](#page-3-1) compares the StO<sub>2</sub> traces for the three different intensities of SIC in one subject. StO<sub>2</sub> was higher at 30% compared with both 60% and 90% MVC and higher at moderate than submaximal MVC ( $p < 0.05$ ). Desaturation rate (De) increased from slow (30%) to moderate (60%) to submaximal contractions ( $p < 0.05$ ) (Table [4.1](#page-3-0)). Trends of De as a function of MVC are shown in Fig. [4.3.](#page-4-0) In Fig. [4.3](#page-4-0) the deoxygenation rate has an S-like shape and its linear approximation is shown as black line. Similarly the linear approximation of the reoxygenation rate in Fig. [4.3](#page-4-0) shown as black line. After each contraction there was a large and immediate hyperemic response (Fig. [4.2](#page-3-1)). The resaturation rate (Re) of StO<sub>2</sub> after SIC depends on the intensity of the SIC and reflects the integrity and functionality of vascular system. It corresponds to a blood vessel vasodilation in response to the SIC. Re increased from slow to moderate ( $p < 0.05$ ), but remained similar for moderate to submaximal SIC (Table [4.1](#page-3-0) and Fig. [4.2](#page-3-1)).

<span id="page-3-0"></span>**Table 4.1** Muscle StO<sub>2</sub> parameters during sustained isometric contractions and arterial occlusion (AO)

<b>MVC</b>	30%	60%	90%	AO
F(N)	$5.40 \pm 1.03$	$10.90 \pm 1.03$	$16.40 \pm 1.03$	$\overline{\phantom{a}}$
$RB(\%)$	$73.16 \pm 0.29$	$73.08 \pm 0.87$	$78.66 \pm 3.17$	$77.75 \pm 3.46$
De $(\%$ /s)	$-1.06 \pm 0.09$	$-4.19 \pm 0.16$	$-4.80 \pm 0.16$	$-0.23 \pm 0.03$
$Re(\% /s)$	$0.84 \pm 0.19$	$1.54 \pm 0.25$	$2.65 \pm 1.44$	$2.5 \pm 0.08$
$\Delta \text{StO}_2$ (%)	$16.83 \pm 4.62$	$54.5 \pm 9.24$	$59.51 \pm 5.14$	$26.45 \pm 1.77$

*F* force, *RB* baseline during rest, *De* desaturation rate during contraction and AO, *Re* resaturation rate after contraction and AO, ∆*StO2* difference between rest and minimum during contraction and AO. Values in mean ± SD

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**Fig. 4.3** Deoxygenation and reoxygenation rate depend on the intensity of the sustained isometric contraction. MVC = percent of maximal voluntary contraction

During the arterial occlusion the minimal StO2 was 52% (Table [4.1](#page-3-0) and Fig. [4.1\)](#page-2-0), which is significantly higher than  $StO<sub>2</sub>$  after moderate or submaximal SIC.

#### **4 Discussion**

According to the literature [[11\]](#page-5-9) an arterial occlusion is applied by inflating the cuff to a pressure of at least 60 mmHg above the systolic pressure. This way, both venous outflow and arterial inflow are blocked. In our case the mean pressure was  $88 \pm 6$ mmHg what correspond to a systolic arterial pressure of about 115–120 mmHg. Blood flow was occluded to the leg by inflating the cuff beyond 180 mmHg. But either this external pressure was not high enough to block blood inflow, or the lower extremity circumference of greater than 75 cm would have required a more efficient tourniquet. Due to these reasons, in dorsiflexor muscle we did have not a full blocking of blood inflow and accordingly we observed during the arterial occlusion a minimal  $StO<sub>2</sub>$  which was significantly higher than the  $StO<sub>2</sub>$  after moderate or submaximal SIC.

The muscle deoxygenation rate was more enhanced at 90% than 60% MVC because  $O_2$  consumption in exercising muscle is higher at 90% than 60% which we can see as statistically significant increasing of  $StO<sub>2</sub>$  from 60% to 90% MVC (Fig. [4.3](#page-4-0)).

## **5 Conclusion**

The sharp decreases in  $StO<sub>2</sub>$  after the start of moderate and submaximal SIC, indicate that the blood vessels are occluded due to intramuscular pressure. This indeed shows that  $O<sub>2</sub>$  delivery is impeded and cannot cope with the increased  $O<sub>2</sub>$ consumption.

**Acknowledgments** The authors gratefully acknowledge funding by the Swiss National Science Foundation (Gr. no. 137423).

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