The Isometric Torque at Which Knee-Extensor Muscle Reoxygenation Stops

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ABSTRACT

DE RUITER, C. J., J. F. A. GOUDSMIT, J. A. VAN TRICHT, and A. DE HAAN. The Isometric Torque at Which Knee-Extensor Muscle Reoxygenation Stops. Med. Sci. Sports Exerc., Vol. 39, No. 3, pp. 443–452, 2007. Purpose: We investigated the knee-extensor torque at which reoxygenation (inflow of arterial blood) during an isometric contraction stopped, whether this torque depended on maximal torque capacity (MTC), and whether there were differences among the synergists. Methods: Isometric knee-extension torque was measured using a dynamometer with 90° angles in the hip and knee. Maximal voluntary activation (established with superimposed nerve stimulation) was >90% in the 15 healthy male subjects (20–30 yr). Near-infrared spectroscopy (NIRS) was used to measure changes in muscle oxygenation of the vastus medialis (VM), vastus lateralis (VL), and rectus femoris (RF) muscle during submaximal isometric contractions at intensities of 20–45% MTC with 5% increments, applied in randomized order and divided over 2 d. At each torque, a contraction with an inflated pressure cuff (450 mm Hg), inducing full arterial occlusion, was followed (10 min of rest) by a second contraction without the cuff. Results: MTC ranged from 178 to 348 N. The torque at which maximal deoxygenation (all oxygen consumed) during contraction without the cuff became similar (P < 0.05) to the maximal deoxygenation reached with the cuff (indicative for complete occlusion of blood flow during the contraction without the cuff) was significantly higher for the RF (35% MTC) than for both vasti (25% MTC). There was no significant relation between MTC and relative (% MTC) torque at which muscle reoxygenation stopped. Conclusion: Knee-extensor reoxygenation stopped at lower torques than previously reported for blood flow in this muscle, and this occurred at the same % MTC in subjects of different strength but at different % MTC for the different synergists. Key Words: ACTIVATION, BLOOD FLOW, OCCLUSION, FATIGUE

In skeletal muscles, the intramuscular pressure caused by the contraction of muscle fibers rises with force (21,23,24). During isometric contractions at intensities of approximately 15–20% of maximal voluntary contraction (MVC), intramuscular pressure may become high enough to hamper blood flow (2,4,21,25). With a further increase of contraction intensity, blood flow may even completely stop (3,4,25). This will cause a rapid decrease of tissue oxygenation, and the active muscle fibers will largely depend on anaerobic energy supply, which will accelerate muscle fatigue. Clearly, reoxygenation of muscle (defined as inflow of arterial blood saturated with oxygen) during a submaximal contraction will postpone fatigue and preserve muscle function. Because muscle tissue oxygenation is the crucial factor in relation to muscle fatigue and not blood flow per se, in the present study, we did not measure muscle blood flow, which would be an indirect indication of oxygen supply to the muscle. Instead, we directly assessed the changes in muscle tissue oxygenation at different torque levels with the use of noninvasive near-infrared spectroscopy (NIRS) (12).

Although it seems generally accepted that blood flow is severely reduced when muscles contract at intensities greater than 30% of MVC, the reported forces (torques) at which intramuscular blood flow completely stops vary from 20 to 64% MVC (2,4,21), and even values higher than 75% MVC have been reported (3). This large variation may partly be attributable to differences in the methods used to measure blood flow, but morphological differences (23,25,28) and differences in anatomic surroundings (15,28) among muscles also may affect muscle blood flow and, consequently, tissue oxygenation. Notwithstanding these differences among the previous studies, the reported force range (20–75% MVC) remains large. Therefore, the goal of the present study was to establish more precisely the isometric torque at which quadriceps femoris muscle tissue oxygen supply was blocked.

To enable comparison of results between studies, subjects, and muscle groups, the torques at which blood flow...
stops are usually normalized to MVC torque (2–4,15,21). However, these previous studies did not control for potential differences in maximal voluntary activation among subjects. This may have increased the variation in torque at blood flow occlusion, both within and among studies, which will occur when torque is expressed relative to the MVC torque of subjects with different abilities to maximally activate their muscles. Therefore, to decrease the intersubject variation, in the present study, only subjects with confirmed high ability for maximal voluntary activation were included.

Differences in absolute muscle force among the subjects may be important in relation to tissue perfusion and oxygenation. Intramuscular pressure increases with the absolute force level (23). If tissue blood flow stops at a certain absolute force (intramuscular pressure) in all subjects, reoxygenation may become impossible at relatively (% MVC) lower torque levels in stronger subjects, as has been reported for handgrip muscles (3). Therefore, as our first hypothesis, we expected that in the knee-extensor muscles, there would be a negative relationship between the maximal torque and the torque (expressed as percentage of the maximal torque) at which complete occlusion of blood flow would occur and, consequently, at which muscle reoxygenation during a contraction would stop.

As indicated before, the anatomic differences among the four knee-extensor muscle heads may differentially affect local oxygen supply during isometric knee extension (22,23,28). Therefore, in the present study, tissue oxygenation of different muscle heads of the knee extensors was investigated. This is also important because we have strong indications that during submaximal contractions, the biarticular rectus femoris (RF) muscle is less strongly activated compared with the monoarticular vastus lateralis (VL) and medialis (VM) (8), although opposite findings also have been reported (20). Nevertheless, on the basis of our own data, we assumed that the RF muscle would be less active and would, consequently, produce relatively less force during submaximal isometric knee extensions than the VL and VM. This could lead to lower intramuscular pressures in RF muscle during submaximal knee extensions. Consequently, our second hypothesis was that complete occlusion of blood flow would occur at higher knee-extension torques in the RF muscle compared with the VL and VM muscles.

**METHODS**

**Subjects.** Fifteen healthy male subjects (20–30 yr, 76 ± 6 kg, and 1.83 ± 0.06 m) signed informed consent, and the local ethics committee approved the study. Only nonobese males were chosen, and females were excluded because the sensitivity of the NIRS measurements decreases with increasing subcutaneous fat layers (26). Fifteen subjects were used because we anticipated that at least 12 subjects would be needed for maximal torques to be distributed over a substantial (± 1.7-fold) range across subjects. The subjects were involved in various sports activities two to four times per week at a recreational level for (mean ± SD) 6.3 ± 3.6 h wk⁻¹. They came to our lab three times with at least 3 d in between and did not perform heavy exercise for at least 24 h before each experiment. They all had participated before in experiments involving electrical stimulation. Only subjects were included who previously had maximal voluntary activation levels > 90%, a level that was confirmed for all subjects in the present study (see below).

**Torque measurements.** Isometric knee-extension torque of the dominant leg was measured using a custom-made dynamometer with 90° angles in the hip and knee. They were firmly secured with straps fastening the hips and shoulders. The axis of rotation of the knee was aligned with the axis of rotation of the dynamometer. The lower leg was tightly strapped to a strain gauge transducer (KAP, E/200 Hz, Bienfait B.V. Haarlem, The Netherlands) placed 25 cm distally from the knee joint, to measure the force exerted at the shin. The real-time force applied to the force transducer was displayed online on a computer monitor and was digitally stored (1 kHz). Extension torque was calculated by multiplication of force with the 25-cm lever arm.

**Electrical stimulation and MVC.** During the first experimental day, maximal voluntary isometric knee-extension torque was determined. The highest torque of three attempts (4–5 s) with 3 min of rest in between was taken as MVC. In addition, maximal voluntary activation was obtained with superimposed electrical stimulation on MVC during three attempts with 3 min of rest in between.

Constant-current electrical stimulation (200-µs pulses) was applied using a computer-controlled stimulator (model DS7H, Digitimer Ltd., Welwyn Garden City, UK) and a pair of self-adhesive surface electrodes (Schwa-medico, Leusden, The Netherlands). After shaving of the skin, the cathode (5 × 5 cm) was placed in the femoral triangle above the femoral nerve, and the anode was placed transversely over the gluteal fold. Stimulation current was increased until force in response to a burst of three pulses applied at 300 Hz (triplet) leveled off. The latter always occurred between 300 and 500 mA, and it was assumed that at that point, all of the knee-extensor muscle fibers were activated.

Voluntary activation (VA) was established as follows. Short-electrical-burst stimulation (three pulses at 300 Hz: triplet stimulation) on the relaxed knee-extensor muscles was followed by triplet stimulation superimposed at the plateau of an MVC of 4- to 5-s duration. This was performed three times, with 3 min of rest in between. The attempt during which the highest voluntary force before the electrical burst was generated was used for further analysis. First, the force enhancement from the superimposed triplet was expressed as a percentage of the force obtained when the triplet was applied on the resting muscle. Next, this value was subtracted from 100%,
resulting in a measure of voluntary activation. VA = 1 – [triplet amplitude at MVC × (triplet amplitude at resting muscle)⁻¹] × 100% (7). For example, if with superimposed stimulation on MVC, VA is calculated to be 90%, this implies that during the MVC, the subject was able to make use of (recruit) 90% of his knee extensors’ maximal torque capacity (MTC). Next, MTC was calculated using the following formula: MTC = MVC × (VA⁻¹) × 100% (9). MTC is the torque the muscle would generate under conditions of real maximal activation. In subjects with high VA (> 90%), the calculated MTC is only slightly higher (maximally 100−90 = 10%) than the measured voluntarily exerted MVC. For all subjects, the submaximal torque levels used on the second and third experimental day were expressed as percentages of MTC. Given that our first hypothesis concerns the intersubject variation in maximal torque production, great care was taken to obtain a valid measurement of the maximal torque-generating capacity of the muscle. Expressing torque relative to MVC values, without checking the level of voluntary activation, will introduce additional unwanted intersubject variation with respect to the relative torque level (% MVC) at which blood flow becomes occluded. Because the method of calculating MTC involves extrapolation to torque values where superimposed stimulation on MVC would no longer lead to a torque increase, the higher the level of voluntary activation, the more reliable the subsequent calculation of MTC will be. Therefore, only subjects with VA > 90% were included in the present study.

NIRS. On the second and third visits, the changes in muscle oxygenation of the VM, RF, and VL during submaximal isometric contractions (ranging from 20 to 45% MTC) were determined by use of a continuous-wave near-infrared spectrophotometer (Oxymon, Artinis Medical Systems B.V., Zetten, Netherlands). The instrument has three transmitters and one receiver. Each transmitter drives two different semiconductor laser diodes with nominal wavelengths of 780 and 850 nm. The average output power per transmitter is less then 1 mW, and the peak output power is approximately 7 W per diode. To keep output power as well as wavelength stable through time, all transmitters are temperature stabilized using a Peltier cooler. The light is detected by a fast, large-area avalanche photo diode (APD). The APD is cooled to a temperature of 10°C to achieve a stable signal with low noise. Ambient light is filtered by means of a daylight cutoff filter (≈750 nm) (27). Data were collected with a sample frequency of 10 Hz. The three optode sets were each fixed in a mold with an interoptode distance of 45 mm. The molds were secured to the thigh using elastic velcro straps so that the optodes would not move during contraction. The optodes were positioned on the center of the muscle bellies. Before the optodes were placed, the skin was shaved and cleaned with ethanol. The positions of the optodes were marked to guarantee that optode positions were similar during the second and third visits.

NIRS is an optical, noninvasive method that can be used to determine the tissue-oxygenation level. NIRS actually measures the change in optical density of the tissue, which, by using a modification of the Lambert–Beer law (10), can be transformed into the change in concentrations of oxyhemoglobin ([O₂Hb]) and -myoglobin ([O₂Mb]) and deoxyhemoglobin and -myoglobin ([HHb] and [HMb]). Because of overlap of the spectrum, it is not possible to distinguish between hemoglobin and myoglobin; thus, the oxygenated and deoxygenated forms of both proteins will, respectively, be denoted by O₂Hb and HHb in the present study. If blood volume in the tissue under the optodes remains constant, total Hb (total Hb; the sum of O₂Hb and HHb) will stay constant, and the changes in [O₂Hb] and [HHb] usually will be mirror images (Fig. 1, lower panels, thick lines: RF and VL with cuff) (8). However, sometimes total Hb changes during contractions, even with full arterial occlusion (Fig. 1, VL). This probably is caused by some ongoing redistribution of blood within the leg during the contraction. The changes in [O₂Hb] and [HHb] were used to establish the torque at which blood flow became completely occluded and at which reoxygenation of muscle tissue would stop (see below).

It has been well established that the sensitivity of NIRS measurements is affected by subcutaneous fat under the optodes (8,26). Therefore, skinfold thickness was measured using a Harpenden skinfold caliper (John Bull; British Indicators Ltd., West Sussex, England). Skinfold was measured with subjects seated in the dynamometer with a 90° knee angle. The average value of six measurements—three on each day, taken at the position of the optodes—was used for each muscle. To get a clear indication of the nonmuscle (adipose and skin) tissue that the near-infrared light had to travel through, adipose tissue thickness was defined as half the skinfold thickness (26).

Establishing full occlusion. The crux of the present experiments was that at each contraction intensity, the changes in [O₂Hb] and [HHb] over time were compared during two subsequent contractions, one with an additional pressure cuff around the thigh to ensure complete arterial occlusion (see below) followed by a second contraction at the same torque but now without such a pressure cuff (10 min of rest in between). It was assumed that when the changes in the near-infrared signals during the contraction without the cuff were similar (see data analysis) to the changes observed with the cuff, the internal muscle pressure generated by the contracting muscle fibers was already high enough to cause full arterial occlusion without the need for an external pressure cuff. Thus, we assumed that with full internal contraction-induced arterial occlusion, application of an additional external pressure cuff would not lead to additional changes in muscle oxygenation over time. Different changes in [O₂Hb] and [HHb] over time between the contractions with and without the cuff would indicate that muscle oxygenation changes and therefore blood flow were not yet completely occluded.
by the contracting muscle at that particular torque level, and some reoxygenation of the muscle was still possible.

Three seconds before the submaximal isometric contractions were executed with a pressure cuff placed around the most proximal part of the thigh, blood flow was occluded by rapid (< 3 s) inflation (using a custom-made inflator) of the cuff (Hokanson SC 10D, Bellevue, WA) to a pressure of 450 mm Hg. To ensure that the cuff did not unwrap, an extra strap was secured around the cuff before inflation. Such a high pressure was necessary to guarantee arterial occlusion during forceful contractions of longer duration, when blood pressure is known to increase. Maximal torque production is not affected by the pressure cuff; more details and the rationale for the choice of pressure have recently been described (8). The cuff was deflated when maximal deoxygenation was reached in all three muscles. Maximal deoxygenation was defined as the absolute difference in [O$_2$Hb] and [HHb]) just after contraction onset and at the time when the O$_2$Hb and HHb signals had leveled off—that is, when virtually all O$_2$Hb had been converted into HHb (arrows in the right panels of Fig. 1). At contraction onset, there often were small changes in blood volume (total Hb) under the optodes, probably because of a redistribution of blood (Fig. 1). Leveling off was established by the eye during the measurements, but post hoc analysis showed that the mean change in [O$_2$Hb] and [HHb] at the plateau was 0.23 ± 0.41%·s$^{-1}$ (mean ± SD), which was not significantly different from zero. The next contraction, now without the pressure cuff, was executed at the same torque as the preceding contraction, and tissue oxygenation was compared at the same point in time during the contractions with and without the cuff. Contraction duration depended on the intensity and ranged from about 110 s at 20% MTC to 40 s at 45% MTC.

**Protocol.** The standard torque levels investigated were 20, 25, 30, 35, 40, and 45% MTC. These torques were selected on the basis of results from pilot experiments. The subjects matched their knee-extension torque, which was displayed in real time on a monitor in front of them, with a target line also displayed on the monitor. To our surprise, in three subjects during the experiments, full occlusion of blood flow in one of the three muscles investigated seemed to have occurred already at 20% MTC. Therefore, in these three subjects, an additional pair (with and without cuff) of contractions was performed at 15% MTC. Conversely, in another subject, an additional pair of contractions was necessary at 50% MTC. The torques presented are the torques that were actually exerted by the subjects during the contraction (averaged over the contraction duration); these torques were found not to be statistically different from the torques the subjects were asked to deliver.

On each day, the experiments started with a 1-min contraction at 20% MTC without the cuff; this contraction was not used in the analysis but served as warming up. Although the contractions did not last until torque failure, because they ended when no further changes in muscle oxygenation occurred, they were long lasting (40–110 s) and, thus, potentially fatiguing. Therefore, the six different intensities were randomly assigned throughout the second and third experimental days, and there were 10 min of rest in between contractions.

**Data analysis and statistics.** The near-infrared signals were analyzed using custom-written Matlab software. In the present study, the [O$_2$Hb] and [HHb] signals were analyzed as follows. First, both signals were corrected for their offset, which was taken as the average value of each signal over the first 2 s after leveling off of the torque at the target level. This offset at the start of a contraction was probably caused by a contraction-induced redistribution of blood. Next, the offset-corrected [O$_2$Hb] and [HHb] signals were rectified and subsequently averaged. This averaged signal (Fig. 1) was used to quantify changes in muscle oxygenation level. Note that whenever blood flow was not fully occluded during the contractions without the cuff (Figs. 1; VL and RF), the
changes in the [O2Hb] and [HHb] signals were already different early on during the contraction, resulting in lower levels of maximal deoxygenation during the later phase of the contraction. Therefore, after trying several different parameters (such as the area under the curves and the average slopes of the signals), the most straightforward, reliable parameter to establish the similarity in changes of muscle oxygenation between the contractions with and without the cuff was maximal deoxygenation reached during the contractions. At each torque, maximal deoxygenation was first calculated for the contraction with the cuff. The averaged and offset-corrected O2Hb and HHb signals (see above) were used, and the average value was taken for a 5-s period at the plateau of this signal (arrows in Fig. 1). Secondly, at the same time after the start of the contraction, maximal deoxygenation during the contraction without the cuff was obtained.

Results are presented here as means ± SD. Pearson’s correlation coefficient was calculated to establish significance (P < 0.05) of correlation. Test–retest reliability was analyzed using the intraclass correlation coefficient (ICC). In the first instance, repeated-measures ANOVA (SPSS 11.5) with three within-subject factors of torque (six different torque levels), muscle (VM, VL, RF), and cuff (with or without) was used to establish significant (P < 0.05) main effects in maximal muscle deoxygenation. This was followed by three separate repeated-measures ANOVA for each muscle, with two within-subject factors (torque and cuff), which were, for significance, followed by two separate (with and without cuff) repeated-measures ANOVA for each muscle (six torque levels). Finally, for significance of the latter, simple contrasts were used to test whether the maximal deoxygenation reached at each torque level was significantly different from the maximal deoxygenation obtained during the contraction at 45% MTC. These tests would establish the average torque at which, in each of the muscles, reoxygenation could no longer occur during contractions. However, this analysis of the whole-group data was not sufficient to relate the potential intersubject variation in torque-dependent muscle oxygenation to the individual maximal torques. Therefore, a second analysis was done to establish, for each individual muscle, the torque at which tissue reoxygenation stopped. As anticipated, at 45% MTC maximal deoxygenation was very similar (repeated measures ANOVA, F1,14 = 0.11, P = 0.74) during contractions with and without the cuff. This implied that at 45% MTC during the contraction without the cuff, reoxygenation did not occur, and there was full occlusion of blood flow in the VL, VM, and RF muscles of all subjects. For each of the three muscles (at 45% MTC), maximal deoxygenation found during the contraction without the cuff was subtracted from maximal deoxygenation found during the contraction with the cuff. The result was expressed as a percentage of maximal deoxygenation obtained during the contraction with the cuff. The resulting differences at 45% MTC for VM, VL, and RF, respectively, were 2.0 ± 7.5, −3.9 ± 6.6, and 2.0 ± 7.9%. These values were not significantly different from zero (paired t-tests: P = 0.57, 0.10, and 0.57, respectively), with an average SD of 7.3% across the muscles. For the contractions at torques other than 45% MTC, it was assumed that when maximal deoxygenation obtained with and without the cuff differed by less than 15% (which is the 95% confidence interval at twice the SD of 7.3%), they were similar. If this was the case, it was concluded that at that torque level, there was no significant reoxygenation of the muscle, and blood flow had completely stopped as a consequence of the internal muscle pressure generated by the contracting muscle fibers. Conversely, when maximal deoxygenation of a muscle during a contraction without the cuff was less than 85% of the value obtained with the cuff, it was concluded that some reoxygenation of the muscle was still possible and that blood flow occlusion was incomplete.

RESULTS

Maximal voluntary activation levels of the subjects were high and were similar (95 ± 3%). This strongly suggests that the intersubject differences in MTC, ranging from 178 to 348 N m on average 254 ± 47 N m, were indeed caused by differences in muscle strength and not attributable to different abilities for maximal voluntary activation.

Figure 1 shows a typical example of torque (top) and tissue oxygenation of the three muscles for contractions both with and without the pressure cuff. The first analysis (on the level of the whole subject group) showed that there were significant main effects for the factors of cuff (F1,14 = 63.0, P < 0.001), muscle (F2,28 = 14.8, P < 0.001), torque (F5,70 = 3.2, P = 0.01), and the interactions of torque and muscle (F10,140 = 2.1, P = 0.03), torque and cuff (F5,70 = 23.7, P < 0.001), and muscle and cuff (F2,28 = 8.4, P = 0.001). Subsequently, the results obtained for each muscle were analyzed separately. For all three muscles, there were significant effects of the cuff (F1,14 > 19.9, P < 0.001) and of the interaction between cuff and torque (F5,70 > 7.6, P < 0.001), meaning that the effect the pressure cuff had on maximal deoxygenation was torque dependent. During contractions with the cuff, there was no significant main effect of torque in VM (F5,70 = 0.9, P = 0.44), VL (F5,70 = 1.89, P = 0.11), and RF (F5,70 = 1.0, P = 0.41), meaning that maximal deoxygenation with the cuff was the same at all torques. During contractions without the cuff, there was a significant main effect of torque in VM (F5,70 = 3.4, P = 0.008), VL (F5,70 = 5.3, P < 0.001), and RF (F5,70 = 20.6, P < 0.001), meaning that maximal deoxygenation was significantly lower at the lowest torques compared with the higher torques (Fig. 2). Maximal deoxygenation during contractions without the cuff was similar compared with maximal deoxygenation reached during contractions with the cuff, from 25% MTC in the VM muscle (Fig. 2A), 25% MTC in VL muscle (Fig. 2B), and 35% MTC in the RF muscle (Fig. 2C).
Adipose tissue thickness was significantly \( F_{2,28} = 25.9, P < 0.001 \) higher for RF (6.9 ± 2.5 mm) than for VM (5.2 ± 1.5 mm) or VL muscle (4.3 ± 1.4 mm). This does not affect the foregoing results, because direct comparisons of maximal deoxygenation with and without the cuff were only made within a muscle. However, as a consequence of the difference in adipose tissue thickness among the muscles, maximal deoxygenation during arterial occlusion was significantly \( F_{2,28} = 6.7, P = 0.004 \) less in RF compared with the VM and VL (Fig. 2). This is further illustrated by the greater maximal deoxygenation reached in Figure 1 for VM and VL compared with RF.

Below, the results of the second analysis, which was done for each individual subject, are presented. This analysis was done to investigate whether the torque at which muscle reoxygenation stopped was strength dependent.

Typical examples of the changes in muscle tissue oxygenation in one of the subjects during isometric knee extensions at 20% MTC with and without a pressure cuff are shown in Figure 1. For the VM muscle, the changes in tissue oxygenation during the contraction without the cuff are very similar to the changes with the cuff. However, maximal deoxygenation during the contraction without the cuff in VL and RF muscle was clearly lower than the values obtained with the cuff. This shows that there was reoxygenation of the VL and RF muscle tissue during the contraction without the cuff. These findings indicate that when this subject exerted torque at 20% MTC, blood flow in his VM muscle was fully occluded, whereas some blood flow was still possible in VL and, particularly, in his RF muscle.

Figure 3 shows maximal deoxygenation of a subject’s VL muscle at different torques during contractions with and without a pressure cuff. It illustrates that reproducibility of maximal deoxygenation during arterial occlusion was high within and across days. For the whole group of subjects, the ICC of maximal deoxygenation during contractions with the cuff for VM, VL, and RF, respectively, were 0.88, 0.96, and 0.97. In addition, Figure 3 shows that when at a certain contraction intensity, maximal deoxygenation without the cuff became similar to that obtained with the cuff (in this example, at 30% MTC), maximal deoxygenation was also similar at all higher torque levels (in this example, 35, 40, and 45% MTC), and, conversely, maximal deoxygenation with and without the cuff was different at all the lower torques (20 and 25%...
MTC). This was a general finding for all three muscles in all our subjects, and it adds to our confidence in the method used to establish the torque at which muscle reoxygenation could no longer occur. It shows that although the measurements at different torque levels were made on two different days, and although torque order was randomized, the outcome was not affected.

Figure 4 shows an example of a contraction during which, without the cuff, blood flow was on the border of being occluded. The changes in [O$_2$Hb] and [HHb] during the contraction with and without the cuff were very similar during the beginning of the contraction, although changes in total Hb seemed different, but later on during the contraction, some reoxygenation did occur (thin arrow in Fig. 4B). This occurred when torque was maintained at the same level with the objective criterion of 15% that we established (see Methods), it was concluded in this case that blood flow was occluded, although clearly some reoxygenation did occur, particularly during the second part of the contraction.

The average absolute torques at which reoxygenation could no longer occur for VM, VL, and RF muscle, respectively, were 62.2 ± 15.5, 61.0 ± 11.0, and 85.5 ± 20.3 N·m, with the torque for RF significantly higher than for the other muscles (Fig. 5A). As a percentage of MTC, these respective torques were 24.3 ± 4.7, 24.3 ± 4.4, and 33.9 ± 7.3% (Fig. 5B). The variation among subjects was substantial: the torque ranges at which reoxygenation stopped in VM, VL, and RF, respectively, were 20–35, 20–35, and 20–45%.

The correlation coefficients between MTC and the relative torque (% MTC) at which reoxygenation stopped for the VM, VL, and RF muscles, respectively, were 0.19 (P = 0.51), −0.49 (P = 0.07), and −0.31 (P = 0.27). The linear relations between MTC and the absolute torque at which reoxygenation stopped were stronger. The correlation coefficients for VM, VL, and RF, respectively,
FIGURE 6—During contractions without the cuff, there was a significant relation \((r = 0.72, P = 0.003)\) between the maximal torque capacity for the knee extensors (\(x\)-axis) and the torque (averaged across three muscles \(\pm SD\)) at which reoxygenation stopped and blood flow became occluded (\(y\)-axis).

were 0.74 \((P = 0.001)\), 0.49 \((P = 0.06)\), and 0.50 \((P = 0.06)\). In addition, when the absolute torques at which muscle reoxygenation stopped were averaged for the three muscles in each subject and these averaged torques were subsequently plotted as a function of MTC, a significant positive linear relation \((r = 0.72, P = 0.003)\) was found (Fig. 6). A similar operation for the relative torques at which muscle reoxygenation stopped did not result in a significant linear relation with MTC \((r = -0.32, P = 0.25)\).

DISCUSSION

The main new findings of the present study are that tissue reoxygenation in the superficial parts of the VL and VM muscles already stopped at isometric contraction intensities of 25% MTC but at significantly higher torques (35%) in RF. In contrast to our expectations, the relative torque (% MTC) at which tissue reoxygenation stopped was independent of the maximal knee-extension strength of our subjects, at least for the VM and RF muscles.

There are two major differences between the present study and previous studies that investigated the relationship between isometric torque production and blood flow. The first is that instead of measuring blood flow (3,4,21,25) as a more indirect measure of muscle oxygen supply, we directly studied the changes in blood flow–dependent tissue oxygenation using NIRS. With NIRS, changes in oxygenation at the capillary level can be measured directly. However, near-infrared light only travels about half the optode distance under the skin (6), and, consequently, in lean subjects, only several cubic centimeters of superficial muscle tissue are sampled. However, if tissue reoxygenation was stopped in the superficial muscle parts, it probably also would have stopped in the deeper muscle parts, because it has been shown that intramuscular pressure is higher in deep muscle parts than in the superficial ones (21,23).

It is also important to note that the optodes were fixed halfway along the longitudinal axis of the muscle belly. Consequently, we cannot exclude that there may have been regional differences in tissue oxygenation (11,18).

Another limitation of the present setup is that, because of the torque steps made, there is an inherent (5% MTC) uncertainty with respect to the isometric torque at which reoxygenation of the muscle tissue was concluded to have stopped. Occasionally, delivered torque was on the boundary of the torque at which occlusion occurred, and some erroneous decisions may have been made (Fig. 4).

The second important difference between the present and the previous studies is that in the present study, maximal voluntary levels of activation were assessed. Maximal voluntary activation may differ considerably among healthy subjects. In our experience, even after practice sessions, it may range from 70 to 100% in the knee-extensor muscles (see also Babault et al. (1)). In other studies (3,21), blood flow occlusion is usually reported relative to MVC, possibly leading to relatively high torques at blood flow occlusion in subjects with poor voluntary activation.

In the present study, reperfusion of muscle tissue could be detected at torque levels that were on the boundary of complete occlusion (Fig. 4). It has been well established that heart rate and blood pressure increases with contraction duration (and torque level) during long-lasting isometric contractions (14,17). Therefore, at a certain torque, intramuscular pressure may be just high enough to occlude blood flow at the beginning of a contraction. However, because of the increase in blood pressure during the contraction, at some point, blood pressure may become higher than the intramuscular pressure, leading to reperfusion of the tissue. Alternatively, reoxygenation of an initially occluded muscle may occur if there is a change in the load sharing among the different heads of the knee extensors.

The presented knee-extension torques at which reoxygenation stopped (25–35% MTC) are much lower than the torques at which blood flow was completely occluded in the VL \((64 \pm 12\% \text{MVC})\) and RF muscles \((50 \pm 6.5\% \text{MVC})\), as Sadamoto et al. (21) reported in a study using 133-Xe clearance. Other than the great difference in methods and the experimental setup, we have no explanation for the large differences between our data and the data from this previous study of the same muscle group (21).

Our data are in line with those of Sejersted et al. (23), who predicted, on the basis of their study of intramuscular pressure in relation to the anatomy of the VM muscle, that VM muscle microcirculation may be compromised during contractions of less than 15% MVC in some subjects. Moreover, our data are in agreement with the recent finding that time to torque failure during sustained knee extension at 25% MVC in men was hardly affected by a
pressure cuff, suggesting that knee-extensor blood flow was almost abolished at 25% MVC in men (5).

Reoxygenation stopped at similar relative torques in our subjects, despite an almost twofold range in absolute MTC (178–348 Nm). These findings deviate from those of Barnes et al. (3), who found a moderate \( r = -0.58, P < 0.01 \) negative relationship between MVC and grip force at blood flow occlusion in the forearm. They measured total forearm blood flow with a plethysmograph, which gives a rather global measure of total limb blood flow, whereas in the present study, we specifically investigated tissue oxygenation of the torque-generating muscles. Moreover, in the study of Barnes et al. (3), there was a 2.7-fold range in MVC, and the force range at occlusion (15–85% MVC) was much greater than in the present study. This may also have contributed to the different results. However, note that in the present study, the relative torque at which reoxygenation stopped tended \( P = 0.07 \) to be negatively \( r = -0.49 \) related to MTC for the VL muscle. This would be in line with the findings of Barnes et al. (3) and with our first hypothesis. It also would be in line with the longer times to task failure found during long-lasting submaximal contractions in women compared with (stronger) men for the elbow flexors (14) and knee extensors (5). However, in the latter study, muscle endurance was not negatively related to maximal strength, and in the study of Hunter et al. (14), the relation became weak \( r = -0.46 \) when only men were analyzed.

In relation to these previous data (3, 5, 14) and the present results, it is important to note that strength differences among subjects depend on many different neural and anatomic factors, the most important being the capacity for maximal voluntary activation, muscle cross-sectional area, and the internal joint moment arm. Patellar tendon moment arm varies considerably among subjects and is smaller in females than males (16). This clearly may affect the relation between torque and blood flow and the intersubject variation in vivo. In addition, blood flow during sustained isometric contractions may not only depend on the contractile force–dependent intramuscular pressure around the capillaries relative to perfusion pressure. The kinking of arterioles where they pass the fascia can also hamper blood flow (13). Moreover, blood pressure responses may have varied among our subjects, also possibly contributing to the observed intersubject variation of the torque at which muscle reoxygenation stopped.

**REFERENCES**


