Blood flow and muscle oxygenation during low, moderate and maximal sustained isometric contractions

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Running Head: Hemodynamics and sustained isometric contractions

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ABSTRACT

A reduction of blood flow to active muscle will precipitate fatigue and sustained isometric contractions produce intramuscular and compartmental pressures which can limit flow. The present study explored how blood flow and muscle oxygenation respond to isometric contractions at low, moderate and maximal intensities. Over two visits, 10 males (26±2 years; mean±SD) performed 1min dorsiflexion contractions at 30, 60 and 100% of maximal voluntary contraction (MVC) torque. Doppler ultrasound of the anterior tibial artery was used to record arterial diameter and mean blood velocity and calculate absolute blood flow. The tissue oxygenation index (TOI) of tibialis anterior was acquired with near-infrared spectroscopy (NIRS). There was a progressive increase in blood flow at 30% MVC (peak of 289±139% resting value); no change from rest until an increase in the final 10s of exercise at 60% MVC (peak of 197±102% rest); and an initial decrease (59±30% resting value) followed by a progressive increase at 100% MVC (peak of 355±133% rest). Blood flow was greater at 30 and 100 than 60% MVC during the last 30s of exercise. TOI was ~63% at rest and, within 30s of exercise, reached steady-state values of ~42%, ~22% and ~22% for 30, 60 and 100% MVC, respectively. Even maximal contraction of the dorsiflexors is unable to cause more than a transient decrease of flow in the anterior tibial artery. Unlike dynamic or intermittent isometric exercise, our results indicate blood flow is not linearly graded with intensity or directly coupled with oxygenation during sustained isometric contractions.

KEYWORDS
dorsiflexors, muscle blood flow, near-infrared spectroscopy
INTRODUCTION

Neuromuscular fatigue in response to voluntary effort can be defined as the reduced ability to exert force or power, regardless of whether or not the task can be performed successfully (4). This fatigue is task-dependent and results from central and peripheral mechanisms that act in concert or independently (1, 6). Because of the importance of blood flow to muscle metabolism, any impediment to muscle blood flow (MBF) will precipitate the development of fatigue in a variety of ways (e.g., 25). MBF is responsive to the intensity and type of muscle contraction (2, 23). Sustained isometric contractions pose particular challenges to muscle perfusion because the increased demand for MBF is opposed by increased intramuscular pressure (IMP) which will limit its delivery (5). IMP is positively related to contraction intensity (22, 24); thus, it is held that there is a critical force threshold beyond which mechanical pressure will occlude MBF (24, 25, 29, 30), and below which flow will be affected to varying degrees depending on the contraction intensity and anatomical location (compartmental pressure). Results obtained using a variety of techniques suggest that complete occlusion of MBF and perfusion occur at 50-60% of maximal voluntary contraction (MVC) force (13, 22, 25, 31). However, relatively few studies have recorded MBF directly during a sustained isometric effort and the degree to which perfusion is compromised by high-intensity contraction remains uncertain.

In the last two decades, hemodynamic responses to isometric exercise have been assessed using four principal techniques: venous occlusion plethysmography (VOP), functional magnetic resonance imaging (fMRI), positron emission tomography (PET) and Doppler ultrasound. The first two are indirect methods which cannot measure blood flow during exercise per se (VOP; e.g., 12), or can only assess a change in flow rather than provide absolute values (fMRI; e.g., 31). In contrast, the latter two methods can provide direct measures of exercise-induced changes in
muscle blood flow. PET measures perfusion directly from the muscle tissue (e.g., 14, 21), whereas Doppler ultrasound tracks changes in arterial size and mean blood velocity (MBV) to provide a direct measure of blood flow into a muscle compartment (e.g., 10, 19). The combination of Doppler ultrasound and near-infrared spectroscopy (NIRS) allows for the assessment of concurrent associations between flow and oxygenation of muscle (see 18 for review).

Despite its advantages, few studies have used Doppler ultrasound to measure MBF during a sustained isometric contraction (7, 8, 13, 27, 30) with only one report conjunctly employing NIRS (9). Most of these studies examined MBF at 2-4 contraction intensities between 10-70% MVC and reported an increase in MBF which was insensitive to the contraction intensity. The partial exception to this finding was a lesser increase in MBF at 50% compared to 20% MVC in males (but an equivalent increase in females; 27). As no previous study has measured MBF during maximal or even near-maximal efforts, our understanding of the relationships among contraction intensity (i.e., IMP), muscle perfusion and oxygenation during sustained isometric contractions remains limited.

Hence, the purpose of this study was to assess real-time changes in the MBF and muscle oxygenation during a sustained MVC and compare these data to results obtained during contractions of moderate and low intensity (60 and 30% MVC, respectively). The dorsiflexor muscle group was selected for this experiment because the restrictive fascial sheath of the anterior compartment induces high levels of IMP during voluntary isometric contractions (26) and a proposed contraction intensity for complete occlusion has been identified in young males (60% MVC; 31).
MATERIALS AND METHODS

Subjects
Ten healthy, recreationally active male volunteers (25.8 ± 2.4 years, 174.8 ± 7.2 cm, 78.8 ± 7.4 kg) were recruited from the local university. Testing for the main experiment was performed over two visits separated by 2 to 4 days. Eight of the subjects participated in an additional experiment on a third day. The study was approved by the local university’s ethics review board, and informed written consent was obtained from each subject.

Experimental set-up
All tests were conducted using the subject’s dominant (right) leg. Subjects were seated in a custom-built isometric dynamometer with their right ankle positioned at 30º of plantar flexion and an angle of 90º at both the knee and hip joints (17). The distal aspect of the right thigh was clamped to minimize hip flexion during isometric dorsiflexion contractions. Straps across the toes and dorsum of the foot secured the limb to the dynamometer footplate.

Ultrasound imaging (8-10 MHz transducer, GE/Vingmed System Five) was used to measure anterior tibial artery diameter and Doppler ultrasound (4.7 MHz; 60º insonation angle, and full-width pulsed wave gate; GE System Five) was used to quantify mean blood velocity (MBV) through the vessel. The ultrasound probe was positioned on the anterior aspect of the right leg, distal and lateral to the tibial tuberosity, to record from the anterior tibial artery below the point at which it emerges through the interosseous membrane. Local tissue oxygenation levels were recorded from the tibialis anterior using NIRS (Hamamatsu NIRO 300, Hamamatsu Photonics KK, Tokyo, Japan). The emitting and detecting optodes of the NIRS unit were placed over the belly of the TA muscle, approximately 8 cm inferior and 2 cm lateral to the tibial tuberosity after the optimum position of the ultrasound probe had been determined and marked.
The optodes were held within a black rubber housing that maintained a constant optode spacing of 4 cm. The opaque housing was affixed to the skin with black tape to minimize any loss of near-infrared light as well as to prevent the intrusion of outside light.

Blood pressure (mean arterial pressure; MAP) was monitored with a pneumatic finger cuff (Ohmeda 2300 Finapres BP Monitor, Louisville, CO) on the middle finger of the left hand, which rested in the subject’s lap. Heart rate (HR) was monitored using a three-lead echocardiogram with Ag-AgCl soft cloth electrodes (1 × 1 cm; 3M, London, ON, Canada).

Electromyographic activity (EMG) of the dorsiflexors was recorded with Ag-AgCl electrodes (1.5 × 1 cm; Kendall-LTP, Chicopee, MA) arranged in a mono-polar configuration. The recording electrode was positioned on the belly of the TA superior to the NIRS housing, whereas the reference electrode was placed on the distal tendon of the tibialis anterior muscle. Finally, a ground electrode was placed on the patella.

**Experimental procedures**

Once a suitable image of the artery was found, two minutes of baseline data (Pre) were collected before exercise began. During pilot testing, it was discovered that sudden, intense dorsiflexion contractions caused the anterior tibial artery to shift position in relation to the probe in some subjects. To improve tracking of the artery, subjects used torque feedback displayed on an oscilloscope to gradually ramp up to the desired contraction intensity over a 5s period.

On Day 1, subjects performed a maximal voluntary contraction (MVC) and continued to contract maximally for 60s. Strong verbal encouragement and visual torque feedback were provided throughout the minute. Three minutes after the end of the sustained contraction, subjects performed another ramped MVC. This MVC was used to assess the recovery of torque, and was maintained for ~2s once torque reached a plateau.
The experimental set-up and procedures were similar for Day 2 except subjects were instructed to ramp their torque to a line on the oscilloscope that corresponded to 30% of their Day 1 MVC and to match that target line as closely as possible for 60s. Following the brief MVC in the recovery period, the subject rested for 15 minutes and then repeated the same test sequence, but at 60% of his Day 1 MVC. During the rest period between the 30% and 60% tasks, HR, MAP, MBV and TOI measures all returned to baseline resting values.

To determine the minimal oxygen saturation value in the dorsiflexors, a subset of participants (n = 8) participated in an additional experiment on Day 3. Participants were seated in the same position as previous experimental sessions; however, a blood pressure cuff was placed around their right thigh to occlude blood flow to the leg. The NIRS unit was affixed to the subject’s leg in the same location as in previous sessions. Blood flow was occluded to the leg by inflating the cuff beyond 240 mmHg, and this occlusion was maintained until oxygen saturation researched a stable minimum level.

Data analysis and statistics

HR, MAP and MBV data were stored on personal computer and analyzed off-line (second-by-second) using Chart software (version 5; ADInstruments Inc., Colorado Springs, CO). In addition to storage of these data on computer, ultrasound data from each trial were recorded on S-VHS tape for the off-line measurement of the anterior tibial artery diameter. Using electronic calipers within the ultrasound software, a single investigator measured vessel diameter during the baseline period (pre-exercise) and at 5, 15, 25, 35, 45 and 55 s of exercise. At each time point, three separate diameter measures were taken and the mean value was used in further analyses. To calculate mean blood flow during exercise, diameter data were combined with MBV (10s of velocity data centered about the diameter measurement) using the following equation:
MBF (ml·min⁻¹) = MBV (cm·s⁻¹) × πr² (cm²) × 60, where \( r \) is the radius of the anterior tibial artery. Subsequently, vascular conductance was calculated as MBF ÷ MAP.

NIRS oxygen saturation (tissue oxygen index; TOI) data were also recorded and transferred to a personal computer for off-line analysis. TOI represents the ratio of oxygenated to total hemoglobin (expressed as a percentage). The half-time of oxygen desaturation (from Pre to the minimum value reached during exercise) was calculated to assess the rate of decrease in oxygen saturation at the onset of exercise. Torque and EMG data were analyzed using Spike 2 software (version 4.13; Cambridge Electronic Design Ltd., Cambridge, UK). Maximal torque was classified as the peak value attained at the onset of the sustained 100% MVC contraction. To match cardiovascular measures, torque was averaged over 10s intervals during exercise. These mean torque values were then normalized to the peak value attained at the onset of the sustained 100% MVC contraction on Day 1. Similar to torque data, root mean square (RMS) EMG amplitude was averaged over 10s intervals during exercise. EMG data during the 100% MVC contraction were normalized to the mean value over the first 10s. Because the submaximal contractions (30 and 60% MVC) were performed on a separate day (and hence electrode placement was not identical), EMG data during the 30 and 60% MVC contractions were normalized to the greatest value over 1s from the recovery MVC performed three minutes after the 30% MVC contraction.

Two-way repeated measures ANOVAs, with contraction intensity and time as within-subject variables, were used to compare HR, MAP, arterial diameter, MBV, MBF, vascular conductance and TOI among contraction intensities (SPSS version 21). All measures except MAP had a significant intensity × time interaction. Thus, at each intensity, results of paired-samples \( t \)-tests were compared to a two-tailed Dunnett’s table to determine which time points in
exercise were different from Pre. For MAP data during exercise, a Bonferroni correction factor was applied to the multiple time comparisons in post-hoc testing. One-way repeated measures ANOVAs were used to compare the half-time of oxygen desaturation among contraction intensities. Separate one-way repeated measures ANOVA were used to test for a main effect of time on torque and EMG during the 100% MVC contraction as well as EMG during the 30 and 60% MVC contractions. Paired-samples $t$-tests were used to compare the torque of each recovery MVC to the peak value of the 100% MVC contraction on Day 1. All data are reported in the text as group mean ± standard deviation. The significance level was $p < 0.05$.

RESULTS

Torque and EMG

Maximal torque was 39.4 ± 4.4 Nm at the onset of the 100% MVC contraction. Torque declined progressively during the sustained maximal contraction ($p < 0.001$) and was 60% of the initial value by the final 10s of exercise (Fig. 1). As intended, submaximal torques of 30 and 60% MVC were held constant throughout exercise (Fig. 1). Peak torque of the recovery MVC was equivalent to the maximal value at the onset of the 100% MVC contraction for all levels of contraction ($p > 0.05$; 103.1 ± 4.8, 100.1 ± 7.1, 96.1 ± 6.2% for 30, 60 and 100% MVC, respectively). EMG activity did not change during the 30 or 100% MVC contractions but increased significantly during the 60% MVC contraction ($p < 0.001$; Fig. 1).

HR and MAP

Heart rate responses to the exercise were graded with contraction intensity. The two-way repeated measures ANOVA revealed main effects of intensity and time as well as an interaction between the two variables (all $p < 0.001$). Specifically, HR was unchanged at 30% MVC but
elevated by 26.2 ± 14.7% at 60% MVC and even higher (59.8 ± 14.4%) at 100% MVC (Fig. 2A). A significant main effect of time indicated an increase of MAP during exercise ($p < 0.001$) but no differences were observed among the three contraction intensities (Fig. 2B).

**Diameter, MBV, MBF and vascular conductance**

Anterior tibial artery diameter was affected by both contraction intensity ($p = 0.002$) and time ($p < 0.001$). There was no change in diameter at 30% MVC but the diameter was significantly compressed during the early stages of the contractions at 60 and 100% MVC which led to differences among the three levels of contraction intensity (Fig. 3A). The patterns of change were nearly identical for MBV (Fig. 3B) and MBF (Fig. 3C) so only MBF data are described here. There were main effects of intensity and time and an interaction between the two variables (all $p < 0.001$) and consequently the three contraction levels showed distinctive MBF responses to exercise. Blood flow began to increase from the onset at 30% MVC, whereas there was an initial reduction in flow at 60 and 100% MVC (not statistically significant at 60% MVC). After this transient decrease during the first 10s, flow increased slowly at 60% MVC but rapidly at 100% MVC such that flow was similar at 30 and 100% MVC and both had greater flow than 60% MVC in the second half of exercise (Fig. 3C). Changes to vascular conductance (Fig. 4) were remarkably similar to those seen for MBV and MBF. That is, there was an immediate increase at 30% MVC but an initial reduction at 60 and 100% MVC. Conductance was significantly greater at 30 than 60% MVC throughout exercise, whereas 100% was less than 30% for the initial 20s, but similar to 30% and greater than 60% for the second half of the fatigue protocol. After cessation of the contraction, there was a large and immediate hyperemic response. The peak value for MBF after 30% MVC (185.5 ± 53.9 mL/min) was lower ($p <$
than the equivalent values after 60 and 100% MVC (280.3 ± 67.0 and 253.8 ± 49.1 mL/min, respectively) (data not shown).

**Oxygen saturation**

The rate of decrease in TOI at the onset of exercise, as assessed by the half-time of desaturation, was progressively faster \((p < 0.001)\) with increasing contraction intensity (21.8 ± 2.9, 16.0 ± 1.6, 12.3 ± 1.2 ms for 30, 60 and 100% MVC, respectively). Results of the two-way ANOVA indicate TOI was affected by both intensity and time and that the two factors interacted with one another \((all \ p < 0.001)\). With the exception of the first 10s at 30% MVC, oxygen saturation was lower than baseline throughout exercise (Fig. 5). Saturation was higher at 30% compared to both 60 and 100% MVC at all time points and higher at 60 than 100% MVC during the first 20s of exercise (Fig. 5). Finally, with maintained muscle ischemia (Day 3), oxygen saturation reached a nadir of 14.3 ± 6.4%. This value was significantly lower than the minimal levels reached during the 60 and 100% MVC fatiguing contractions \((p < 0.05)\). The large post-exercise increase in MBF was reflected in the saturation data. The peak value was lower after 30 than 100% MVC (109.2 ± 6.9% and 120.2 ± 9.0% of the resting value, respectively; \(p < 0.05\)), whereas 60% MVC (116.4 ± 11.4) was equivalent to the other intensities (data not shown).

**DISCUSSION**

The major findings of the study were: 1) blood flow was not occluded at the level of the conduit artery during any of the contraction intensities, and indeed was increased overall from rest during the majority of the latter half of the sustained contractions; 2) blood flow was not linearly graded according to contraction intensity; 3) despite continued or increased blood flow through the conduit artery during the contraction, local muscle oxygenation levels declined rapidly and
considerably; and 4) the pattern and extent of muscle deoxygenation was similar at moderate (60% MVC) and maximal contraction intensities.

Occlusion caused by intramuscular pressure

In a variety of muscle groups (e.g., elbow flexors, knee extensors, plantar flexors and dorsiflexors), data collected indirectly using isotope clearance (22), VOP (25) or fMRI (31) suggest that the IMP generated during isometric exercise will occlude MBF at a relative contraction strength of 50-65% MVC (60% MVC in the dorsiflexors of young males; 31). We expected that ultrasound of the narrow (3.5 ± 0.2 mm) anterior tibial artery would enable confirmation of this result via direct measurement of blood flow during a sustained isometric contraction. However, our data illustrate that even a maximal contraction of the dorsiflexors fails to generate sufficient IMP to cause more than transient reductions (<10s) in diameter and blood flow through the artery (14% and 40%, respectively). Nevertheless, the similarity of NIRS-derived oxygen saturation at 60 and 100% MVC during exercise (Fig. 5) and the comparable post-exercise hyperemic response of MBF and TOI may (see below for a conflicting perspective) support the suggestion (31) that IMP at 60% MVC is sufficient to occlude blood flow to the contracting muscle, most likely at the lower order arterioles or the capillary bed (15, 28).

Blood flow and oxygen saturation

It has been established that, during dynamic exercise, blood flow increases to meet the demands of the working muscle (2, 19, 23). However, the results of this and other studies (7, 13, 27, 30, 31) suggest that the same relationship does not exist during sustained isometric exercise. In the present study, there was a progressive increase in blood flow at 30% MVC; no change from rest until an increase in the final 10s of exercise at 60% MVC; and an initial decrease followed by a
progressive increase at 100% MVC (Fig. 3). Thus, our results illustrate a non-linear gradation and differential time-course of blood flow among low (30% MVC), moderate (60% MVC) and maximal (100% MVC) contractions.

In accordance with data collected at comparable contraction intensities in other muscle groups (7-9, 13, 27), a sustained 30% MVC contraction of the dorsiflexors induced a progressive increase in blood flow in the conduit artery, presumably to attempt to meet the energy demands of the task. However, despite the increase in blood flow, oxygen saturation was ~30% lower than the resting value for the final 45s of contraction. Hicks and colleagues (9) reported a comparable reduction in oxygen saturation during wrist flexion at 30% MVC, albeit after a greater delay than in the present study. The absence of even a transient reduction in artery diameter or blood flow suggests that IMP had a negligible impact at this contraction intensity.

Unlike comparable increases in blood flow reported at low (25 or 30% MVC) and moderate contraction intensities (50 or 70% MVC) in the much larger femoral artery (7) or the brachial artery of females (13), we saw marked differences in anterior tibial artery flow between 30 and 60% MVC. Thompson and colleagues (27) demonstrated that sex-related differences in strength impact blood flow as they found equivalent increases in brachial artery flow at 20 and 50% MVC for females (similar to 7, 13), whereas their data collected in males mirror our findings at 30 and 60% MVC. Despite presumed occlusion of the muscle tissue (31), a 1min contraction at 60% MVC failed to alter anterior tibial artery blood flow until an increase during the final 10s of exercise. This suggests that: 1) the energy demands of the contraction are almost met by anaerobic pathways and oxidative phosphorylation fuelled by the oxygen trapped in the muscle at the onset of exercise; or 2) flow to the muscle is not completely occluded (more on this below). Earlier increases in HR and MAP (between 30-40 and 20-30s of exercise, respectively)
than blood flow indicate a modest pressor response which required time to compensate for the
non-significant reduction in flow observed during the first 10s of exercise.

In contrast, the maximal contraction induced a pressor response large enough to increase
anterior tibial artery blood flow above resting levels after 30s of exercise despite the 40%
reduction in flow observed during the first 10s. Although we did not measure IMP in this study,
the initial transient decreases in anterior tibial artery diameter and blood flow as well as vascular
conductance suggest that IMP at the onset of exercise was sufficiently high to overcome the
exercise-induced demand for increased MBF. Unlike the submaximal tasks during which torque
was held constant, the maximal contraction resulted in a linear decrease in torque from 100 to
60%. The presumed decline in IMP is likely to have facilitated the ability of the concurrent
metaboreflex-induced pressor response to increase blood flow to the muscle. Torque remained
above 60% MVC for the entire minute so the elevated flow in the conduit artery may not have
aided (entered) the contracting muscle (31). However, oxygen saturation reached a steady-state
minimum value (~21%) which failed to match the minimum achieved with muscle ischemia
(14%). This suggests that the enhanced blood flow was reaching the muscle tissue because the
reliance on oxidative phosphorylation increases markedly during a sustained 1min dorsiflexor
MVC (16). Moreover, despite greater torque production (energy demand) and blood flow in the
conduit artery at 100 compared to 60% MVC, TOI was equivalent beyond 20s of contraction
(Fig. 5). NIRS-derived oxygen saturation is believed to be representative of the balance between
O₂ delivery and extraction so the data suggest that some of the added blood available at 100%
MVC reached the contracting muscle or else the TOI score would have been lower than at 60%
MVC. However, it cannot be ruled out that there is a technological component to the results.
That is, the NIRS signal represents a weighted average of venous and arterial blood (3) and an exercise-induced shift in the balance could influence the TOI value.

Methodological Considerations

We observed exercise-induced increases in blood flow at the level of the conduit artery, but measurement of gross flow at this site does not indicate how much of the added blood actually perfused the contracting muscle. The concurrent examination of muscle oxygenation allowed us to speculate on perfusion but the combination of Doppler ultrasound and NIRS does not approach the precision of PET which can show the distribution of blood in a muscle during a sustained isometric contraction (14, 20, 21).

Although both appropriate and necessary for the current research question, the 1min contraction duration (imposed by the ability to maintain a constant torque at 60% MVC) precluded a more complete investigation of competing mechanisms during sustained low-intensity contraction.

Perspectives and Significance

The results from this study indicate that, even during a maximal contraction of a distal muscle group housed in a restrictive fascial sheath, increases in IMP do not greatly impede blood flow at the level of a narrow conduit artery. Equivalent oxygen saturation during exercise as well as blood flow after exercise at 60 and 100% MVC may support the proposed (31) occlusion of blood flow to the dorsiflexor muscle tissue at 60% MVC, but downstream of our recording site. However, data from the same research group which show a marked increase in oxidative phosphorylation during a 1min dorsiflexor MVC in young males (16) lead us to interpret our blood flow and oxygen saturation data as evidence that some blood reaches contracting muscle fibres even at MVC. Finally, our results show, in contrast to dynamic (2, 19) or intermittent
isometric (11, 32) exercise, blood flow is not linearly graded with exercise intensity nor directly linked with muscle oxygenation during sustained isometric contractions. Thus, similar to neuromuscular fatigue itself, blood flow responses to sustained isometric exercise demonstrate clear task-dependency.

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GRANTS

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REFERENCES


FIGURE CAPTIONS

Figure 1. Normalized dorsiflexor torque and tibialis anterior EMG during sustained isometric contractions at different levels of maximal torque. Values are means ± SE. Contractions were performed at 30 (○), 60 (□) and 100% (△) of MVC torque. Open symbols are torque, whereas filled symbols are EMG data. Torque data are normalized to the peak value obtained at the start 100% MVC contraction. EMG data during the 30 and 60% MVC contractions are normalized to the value obtained during the brief MVC performed in the recovery period following the 30% MVC contraction. For the 100% MVC contraction, EMG data are normalized to the mean value from the first 10 s of exercise.

Figure 2. Heart rate (A) and mean arterial pressure (B) during sustained isometric contractions at different levels of maximal torque. Values are means ± SE. Contractions were performed at 30 (○), 60 (□) and 100% (△) of MVC torque. Filled symbols indicate data points that are significantly different from Pre (p < 0.05). Asterisks (*) denote time points when 60% MVC differs from 100% MVC (p < 0.05). Double daggers (‡) denote time points when 30% is different from both 60 and 100% MVC (p < 0.05).

Figure 3. Anterior tibial artery diameter (A), mean blood velocity (B) and mean blood flow (C) during sustained isometric contractions at different levels of maximal torque. Values are means ± SE. Contractions were performed at 30 (○), 60 (□) and 100% (△) of MVC torque. Filled symbols indicate data points that are significantly different from Pre (p < 0.05). Asterisks (*) denote time points when either 30 or 60% MVC differs from 100% MVC (p < 0.05). Daggers (†) denote time points when 30% is different from 60% MVC, whereas double daggers (‡) denote time points when 30% is different from both 60 and 100% MVC (p < 0.05).
Figure 4. Anterior tibial artery vascular conductance during sustained isometric contractions at different levels of maximal torque. Values are means ± SE. Contractions were performed at 30 (○), 60 (□) and 100% (▲) of MVC torque. Filled symbols indicate data points that are significantly different from Pre (p < 0.05). The asterisk (*) denotes the time point when 60% MVC differs from 100% MVC (p < 0.05). Daggers (†) denote time points when 30% is different from 60% MVC, whereas double daggers (‡) denote time points when 30% is different from both 60 and 100% MVC (p < 0.05).

Figure 5. Dorsiflexor oxygen saturation during sustained isometric contractions at different levels of maximal torque. Values are means ± SE. Contractions were performed at 30 (○), 60 (□) and 100% (▲) of MVC torque. Filled symbols indicate data points that are significantly different from Pre (p < 0.05). Asterisks (*) denote time points when 60% MVC differs from 100% MVC (p < 0.05). Double daggers (‡) denote time points when 30% is different from both 60 and 100% MVC (p < 0.05).