Increasing blood flow before exercise in spinal cord-injured individuals does not alter muscle fatigue

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Olive, Jennifer L., Jill M. Slade, C. Scott Bickel, Gary A. Dudley, and Kevin K. McCully. Increasing blood flow before exercise in spinal cord-injured individuals does not alter muscle fatigue. J Appl Physiol 96: 477–482, 2004. First published September 23, 2003; 10.1152/japplphysiol.00577.2003.—Previous studies have shown increased fatigue in paralyzed muscle of spinal cord-injured (SCI) patients (Castro M, Apple D Jr, Hillegass E, and Dudley GA. Eur J Appl Physiol 80: 373–378, 1999; Gerrits H, Hopman MTE, Sargeant A, and de Haan A. Clin Physiol 21: 105–113, 2001). Our purpose was to determine whether the increased muscle fatigue could be due to a delayed rise in blood flow at the onset of exercise in SCI individuals. Isometric electrical stimulation was used to induce fatigue in the quadriceps femoris muscle of seven male, chronic (>1 yr postinjury), complete (American Spinal Injury Association, category A) SCI subjects. Cuff occlusion was used to elevate blood flow before electrical stimulation, and the magnitude of fatigue was compared with a control condition of electrical stimulation without prior cuff occlusion. Blood flow was measured in the femoral artery by Doppler ultrasound. Prior cuff occlusion increased blood flow in the first 30 s of stimulation compared with the No-Cuff condition (1,350 ± 680 ml/min, respectively; P < 0.001), although blood flow at the end of stimulation was the same between conditions (1,260 ± 140 vs. 1,160 ± 370 ml/min, Cuff and No-Cuff condition, respectively; P = 0.511). Muscle fatigue was not significantly different between prior cuff occlusion and the control condition (32 ± 13 vs. 35 ± 10%; P = 0.670). In conclusion, increased muscle fatigue in SCI individuals is not associated with the prolonged time for blood flow to increase at the onset of exercise.

spinal cord injury; Doppler ultrasound; electrical stimulation

SPINAL CORD- INJURED (SCI) individuals have been shown to have significant vascular and muscle changes that are thought to contribute to increased muscle fatigue. Vascular changes in SCI individuals include reduced diameter size (5, 26), reduced blood flow (25), decreased venous capacity (18), and decreased vascular reactivity (27) in the affected areas. Extreme muscle atrophy (7) as well as fiber type transformation toward fast-fatigable fibers has been well documented in SCI individuals (15, 22, 35). Furthermore, decreased mitochondrial content (22, 35), increased capillary-to-fiber ratio (22), and a greater clumping of muscle fibers (6) have been documented in SCI individuals, leading to a greater heterogeneity in blood flow and possibly inadequate oxygen diffusion across the tissues. Any combination of these factors may contribute to increased muscle fatigue in SCI individuals.

We have been interested in the potential role of inadequate blood flow in explaining the increased muscle fatigue in SCI patients. Large changes in arterial diameter and peak blood flow do occur after SCI but are in proportion to muscular atrophy (26). We have recently shown that the magnitude of blood flow relative to muscle mass is not reduced in SCI compared with able-bodied subjects during electrical stimulation and cannot explain the increased muscle fatigue (28). However, we did find a fivefold slower rate in the increase in blood flow at the onset of stimulation in SCI compared with able-bodied individuals (28). Thus inadequate oxygen delivery at the start of stimulation could possibly explain the increased fatigue in SCI patients. In support of this idea, altered inspired oxygen content has been shown to affect muscle metabolism in able-bodied humans (17, 33). However, other studies have suggested that augmenting oxygen delivery at the start of exercise does not improve oxygen utilization in able-bodied humans (2, 21, 40) and in animals (13). Given the metabolic differences between SCI patients and able-bodied controls, it is not clear what effect the reduced blood flow at the onset of exercise would have on muscle fatigue in SCI patients.

The purpose of this experiment was to determine whether the prolonged half time to peak blood flow found in SCI individuals contributes to the increased muscle fatigue typically found in this population. To test this hypothesis, both thighs of SCI individuals were electrically stimulated while blood flow and muscle fatigue were measured. In one leg, blood flow was increased rapidly via cuff occlusion to raise blood flow to exercise levels before the onset of exercise. In a separate test on the other leg, blood flow was measured in response to electrical stimulation without cuff occlusion. We hypothesized that enhancing blood flow at the onset of electrical stimulation would not reduce muscle fatigue in SCI patients. This would suggest that muscle fatigue in SCI patients is not a result of reduced oxygen delivery.

METHODS

Subjects

Seven male, chronic (>1 yr postinjury), complete (American Spinal Injury Association, category A) SCI subjects (6 paraplegic, 1 tetraplegic) volunteered to participate in the study. The physical characteristics of the SCI subjects are in Table 1. These subjects had previously participated in a similar study (28). Subjects were excluded if they reported that they were smokers. None of the subjects had any history of disease or other confounding factors. Medications were recorded, and the only medication that was used was antispasticity medication (baclofen) in two of the SCI individuals. Baclofen has no...
known reported vascular effects. The study was conducted with the approval of the Institutional Review Board at the University of Georgia, and all subjects provided written, informed consent.

**Protocol**

Subjects were asked to abstain from fatty foods, caffeine, and alcohol for at least 12 h before testing. This was done to eliminate the potential for diet to confound the results (29). Blood pressure was measured in the arm throughout the entire testing period by using an automated blood pressure machine (Datascope, Mahwah, NJ). Resting artery diameter and blood flow were measured in the femoral artery of both legs by using Doppler ultrasound. Each subject underwent one 4-min trial of muscle stimulation on both legs. Electrical stimulation occurred in one leg after 10 min of cuff ischemia (Cuff condition), whereas in the other leg, no cuff was used before electrical stimulation (No-Cuff condition). Ischemia was induced in the distal thigh and leg by inflation of a 12-mm blood pressure cuff just proximal to the knee to a pressure 100 Torr above systolic pressure. Before cuff release, one stimulation train was delivered to the muscle to increase microvascular perfusion to the quadriceps femoris muscle (10, 11, 39). Cuff inflation and deflation were rapid (1–2 s) and performed by using a Hokanson (Bellevue, WA) device.

**Electrical stimulation.** Subjects were asked to sit on a custom-built chair with a rigid lever arm positioned 70° below horizontal. Mounting a load cell perpendicular to and 12 in. from the axis of rotation of the lever arm established a moment arm of 33 cm. The dynamometer was calibrated by hanging known weights on the load cell. The leg was secured to the rigid level arm with an inelastic strap. Surface electrical stimulation of the quadriceps femoris muscle was conducted by using a commercially available stimulator (TheraTouch model 4.7, Rich-Mar, Inola, OK). Isometric force was recorded on a computer using MacLab 4E (Castle Hill, Australia). Surface electrodes (Uni-Patch, Wabasha, MN) were placed on the quadriceps femoris muscle, one 2–3 cm above the superior aspect of the patella and the other lateral to and 30 cm above the patella on the vastus lateralis muscle. Subjects received electrical stimulation to evoke isometric knee extension (30-Hz train of 450–µs biphasic pulse, 50–µs phase delay) at a current that would elicit ~30 N·m of torque as previously described (28). This was done to elicit similar muscle activation between subjects and legs (1, 16). The work-rest cycle was 1:4 s, which was chosen to allow adequate time between contractions for blood flow measurements by Doppler ultrasound. The electrical stimulation lasted 4 min. Blood flow was measured until it returned to baseline or for 10 min, whichever was shorter. Muscle fatigue was calculated for each exercise bout as a percent decline in force production from the first exercise bout as a percent decline in force production from the first exercise bout as a percent decline in force production from the first exercise bout as a percent decline in force production from the first exercise bout as a percent decline in force production from the first exercise bout as a percent decline in force production from the first exercise bout as a percent decline in force production from the first exercise bout as a percent decline in force production from the first exercise bout as a percent decline in force production from the first exercise bout as a percent decline in force production from the first exercise bout as a percent decline in force production from the first exercise bout as a percent decline in force production from the first exercise bout as a percent decline in force production from the first exercise bout as a percent decline in force 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exercise bout as a percent decline in force production from the first exercise bout as a percent decline in force production from the first exercise bout as a percent decline in force production from the first exercise bout as a percent decline in force production from the first exercise bout as a percent decrease in force production from the first five contractions to the last five contractions.

**Blood flow.** Blood flow was measured in the femoral artery using quantitative Doppler ultrasound (General Electric LogIQ 400CL). A linear array transducer was used at a frequency of 6–9 MHz. The imaging site was located immediately distal to the femoral bifurcation and was marked to ensure replication of probe placement. Resting flow and diameter (during diastole) measurements were made before any exercise. Pulsed Doppler ultrasound was recorded in the longitudinal view by using an insonation angle between 45 and 60°. The velocity gate was set to include the entire arterial diameter.

Velocity measurements were autocalculated every heart beat by General Electric’s advanced vascular program software for the General Electric Electric LogIQ 400 CL. The minimum, maximum, and time averaged maximum velocity measurements were saved directly to a computer, allowing data acquisition on a beat-by-beat basis. Images were saved to magnetic optical disks for measurement of vessel diameter by custom-made software (Labview 6i, Austin, TX). Blood flow was calculated as the product of femoral artery cross-sectional area and the time-average maximum velocity. The time-average maximum velocity was used in this study despite that it overestimates the mean blood flow response (~40%), because the General Electric autocalculation program could not calculate the mean blood flow response throughout the entire testing protocol. The resting vessel diameter was used for calculation of blood flow, because no dilatation was found in the femoral artery during exercise. The observation that the femoral artery does not dilate with exercise has been reported previously (31) and has been verified in our laboratory with exercise (28) and cuff occlusion (26). Conductance was calculated by dividing the blood flow by the mean arterial pressure. Half time to peak blood flow was determined as the time where blood flow increased from one-half the difference of flow at the onset of exercise to maximal flow during exercise. The time for blood flow to increase is related to the muscle pump, vasodilators, and metabolic demand during exercise (32, 38).

**Statistical Analysis**

Independent samples t-tests (SPSS version 10.0) were conducted to compare for differences in blood pressure, fatigue, blood flow, and conductance between conditions. Levene’s test was conducted to determine equality of variances and was corrected for if inequality was found. If multiple t-tests were conducted they were corrected by the Bonferroni method. A mixed-model repeated-measures analysis was used to determine differences between conditions across time for blood flow and conductance. Mauchly’s test was conducted to determine if sphericity was violated. If sphericity was violated, the repeated-measures ANOVA was corrected by using the Greenhouse-Geisser correction factor. The data were analyzed to verify normality and to test for any outliers. All analyses were conducted at a significance level of 0.05.

**RESULTS**

**Baseline Data**

There was no significant difference in resting blood flow between conditions in the subjects (330 ± 145 ml/min and 380 ± 180 ml/min, No-Cuff and Cuff conditions, respectively). There was no significant difference in mean arterial pressure between resting, exercise, or cuff conditions, which allowed us to report blood flow instead of conductance. Subject

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**Table 1. Individual characteristics of SCI individuals and mean data for AB and SCI groups**

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age, yr</th>
<th>Body Mass, kg</th>
<th>Height, cm</th>
<th>Time Since Injury, yr</th>
<th>Level of Injury</th>
<th>Mean Arterial Pressure, mmHg</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>36</td>
<td>72.7</td>
<td>178</td>
<td>20</td>
<td>$T_6$</td>
<td>107</td>
</tr>
<tr>
<td>B</td>
<td>26</td>
<td>63.6</td>
<td>173</td>
<td>3.5</td>
<td>$T_7$</td>
<td>91</td>
</tr>
<tr>
<td>C</td>
<td>32</td>
<td>65.9</td>
<td>170</td>
<td>13</td>
<td>$T_9$</td>
<td>100</td>
</tr>
<tr>
<td>D</td>
<td>39</td>
<td>75.0</td>
<td>173</td>
<td>21</td>
<td>$T_{7/10}$</td>
<td>102</td>
</tr>
<tr>
<td>E</td>
<td>33</td>
<td>109.1</td>
<td>178</td>
<td>8</td>
<td>$T_2$</td>
<td>104</td>
</tr>
<tr>
<td>F</td>
<td>37</td>
<td>113.6</td>
<td>191</td>
<td>7</td>
<td>$T_4$-$T_5$</td>
<td>96</td>
</tr>
<tr>
<td>G</td>
<td>31</td>
<td>68.2</td>
<td>180</td>
<td>8</td>
<td>$C_5$</td>
<td>75</td>
</tr>
<tr>
<td>Average</td>
<td>33±4</td>
<td>81.2±21.0</td>
<td>178±7</td>
<td>12±7</td>
<td></td>
<td>96±11</td>
</tr>
</tbody>
</table>

Values are means ± SD. SCI, spinal cord injured; AB, able bodied.
G was an outlier on resting blood flow, mean arterial pressure, and resting conductance. This subject, however, was not an outlier on fatigue, blood flow, or conductance responses to exercise. Elimination of the subject from the data set did not change the statistical significance of any analyses, and thus the subject was maintained in the data set.

Muscle Fatigue

Electrical stimulation resulted in significant amounts of muscle fatigue (Fig. 1). There was no difference in the amount of fatigue in the No-Cuff and Cuff conditions throughout the electrical stimulation period \([F(1,12) = 0.840, P = 0.378]\). Nor was there a significant difference in the fatigue between conditions at the end of the exercise bout \([t(12) = 0.437, P = 0.670]\).

Exercise Hyperemia

Blood flow was increased during electrical stimulation in both conditions. During cuff ischemia, blood flow was reduced to \(\sim 25\%\) of resting flow because of the proximal placement of the Doppler probe relative to the cuff. On release of the cuff, there was an initial burst of blood flow with the first heartbeat and a large hyperemic response. The initial burst of blood flow was not used in data analysis. A representative blood flow response to exercise under both conditions can be seen in Fig. 2. Note in Fig. 2 that blood flow increases gradually during the first 120 s of electrical stimulation during the No-Cuff condition. The half time to peak blood flow in the No-Cuff condition was 40 \(\pm\) 12 s. In the Cuff condition, blood flow increased immediately on cuff release.

Blood flow was significantly lower in the No-Cuff compared with the Cuff condition at 30 s \([t(10) = 5.053, P < 0.001]\) and 60 s \([t(12) = 4.3018, P = 0.002]\) into electrical stimulation (Fig. 3). In the No-Cuff condition, blood flow at 30 and 60 s was significantly reduced compared with 120, 180, or 240 s \((P \leq 0.009)\). In both conditions, by 120 s blood flow plateaued for the rest of the stimulation period. There was a significant interaction in blood flow between condition and time \([F(4,40) = 15.943, P < 0.001]\); thus separate analyses were
conducted to determine whether blood flow was significantly different between conditions and between time periods. Blood flow during the Cuff condition was elevated above that of electrical stimulation alone and decreased to a steady level within the first 30–60 s of stimulation. Thus the cuff was successful in increasing blood flow during the first minute of exercise, and it eliminated the delayed rise in blood flow seen in SCI individuals during exercise.

**DISCUSSION**

The primary finding of this study was that muscle fatigue was not significantly reduced when blood flow was enhanced at the start of exercise in SCI patients. Without prior cuff occlusion, the half time to peak blood flow at the onset of electrical stimulation was ~40 s. We reported previously that SCI individuals had adequate blood flow per muscle mass but a delayed rise in blood flow at the start of electrical stimulation (42 s) compared with able-bodied individuals (8 s) (28). Furthermore, SCI individuals had higher rates of muscle fatigue (3- to 5-fold) compared with able-bodied individuals. Thus the purpose of this experiment was to determine whether the delayed rise in blood flow at the onset of exercise was related to the increased muscle fatigue.

Our results indicate that a delayed rise in blood flow at the onset of exercise is not a potentially limiting factor to muscle function in SCI individuals. This conclusion is based on the assumption that we increased blood flow to the stimulated muscles through the use of cuff ischemia to the lower leg. To ensure that the quadriceps microvasculature received increased perfusion during the electrical stimulation bout, a 1-s stimulation train was applied to the quadriceps immediately before cuff release. This was done as motor unit recruitment has been shown to cause vasodilation of the microvasculature (39). Furthermore, prior contractions before an exercise bout has been shown to accelerate microvascular oxygen exchange (4). The dispersion of the muscle fibers that make up a motor unit over the muscle allows for the stimulation of one motor unit to lead to perfusion of several microvascular units. Modeling suggests that as little as 18% of muscle fibers need to be activated to perfuse the entire muscle during electrical stimulation (11). We are confident that we activated at least 18% of the muscle fibers of the activated muscle mass because the SCI individuals were stimulated at near maximal force generation.

Supporting our finding are experiments in which neither increased blood flow (13) nor enhanced oxygen diffusion (13) to electrically stimulated canine muscle altered muscle fatigue, lactate accumulation, or muscle oxygen uptake kinetics. In healthy humans, muscle oxygen delivery exceeded oxygen demand during knee extensor exercise, suggesting intracellular limitations to muscle oxygen extraction rather than oxygen delivery limitations (2). Furthermore, increased oxygen exchange that occurred during muscle contraction was related to intracellular mechanisms of the muscle rather than increased muscle blood flow (4).

The control and importance of blood flow at the onset of exercise are complex. In one study, blood flow at the onset of exercise was not significantly correlated to muscle oxygen uptake in healthy individuals and yet was significantly correlated in individuals with diabetes or heart disease (3). However, other literature has suggested that blood flow at the beginning of exercise is significantly related to muscle oxygen uptake in healthy individuals (19). These findings suggest that disease status may impact the role of blood flow and oxygen uptake in the muscle. Furthermore, blood flow at the onset of exercise is increased after 10 days of exercise training, indicating that training status is also important in the regulation of blood flow kinetics (36). This study provides evidence that SCI individuals who are extremely detrained compared with healthy individuals have reduced blood flow at the onset of exercise and that it does not affect the fatigability of the exercising muscle.

An explanation for our findings may be that a delay in oxygen utilization reduces the need for oxygen delivery at the start of exercise (24). In addition, small deficits in oxygen delivery could be overcome with increased oxygen extraction (21). Although this study was not designed to determine the mechanism behind the results, the data are consistent with this conclusion. Another explanation for the lack of relation between muscle fatigue and enhanced blood flow in SCI patients is that muscle fatigue may not be directly linked to oxidative metabolism. A previous study has reported that muscle fatigue in SCI individuals was unrelated to fiber type or aerobic-
oxidative enzyme levels (8). This was based on experiments that showed that acute, complete SCI patients had greater fatigue than able-bodied subjects despite having no fiber type transformation and small (~10%) reductions in succinic dehydrogenase (8). However, chronic SCI patients like the ones we tested have been shown to have changes in muscle fiber types from slow-oxidative to fast-glycolytic fibers (15, 34, 37). The change in fiber characteristics also included decreases in oxidative enzymes (15, 22), decreased fiber diameter size (35), decreased mitochondrial content (22, 34), and increased contractile speed (12).

If muscle fatigue in SCI patients is unrelated to oxidative metabolism, other cellular mechanisms might be responsible. Reduced calcium kinetics may be one possible mechanism for muscle fatigue (11, 12). SCI individuals were shown to have an increased amount of the fast-fiber isoform of sarcoplasmic reticulum calcium ATPase, contributing to increased fatigability (37). In addition, impaired calcium uptake by the sarcoplasmic reticulum and a delayed dissociation of cross bridges has been reported in SCI patients resulting in an increased half time of relaxation and fatigue (35). Increased glycolysis and decreased glycogen levels may be other possible mechanisms related to muscle fatigue. A lower free fatty acid uptake has also been reported during moderate exercise in chronic SCI compared with able-bodied subjects, implying a great demand on carbohydrate metabolism and glycolysis (20). Decreased glycogen levels are directly related to a glycogen-dependent failure of sarcoplasmic reticulum calcium release, leading to increased fatigue (9). Additional studies are needed to better demonstrate the mechanism of muscle fatigue in SCI patients and in particular to examine the role of altered calcium kinetics in fatigue in SCI.

Another explanation for our results is that the decreased force that we reported in our study is not muscle fatigue but is actually muscle injury. We are defining fatigue as the loss of force with repeated muscle activation, related to metabolic alterations. Muscle damage is the loss of force with repeated stimulation due to structural changes. In a recent study, we have found that isometric electrical stimulations resulted in evidence of muscle damage in SCI patients (4a). In that study, the stimulation intensity was higher than the one reported here; however, it is possible that the reduction in force was due to muscle damage. Isometric contractions are not usually thought to cause muscle damage (23); however, SCI muscle maybe more susceptible to damage than muscle from able-bodied subjects. Consistent with this theory, skeletal muscle unloading has been shown to increase the vulnerability to contraction-induced muscle injury (30). We did not measure muscle damage in this study, so we can only postulate that this might be an explanation for the increased muscle fatigue in SCI individuals.

The possibility that we activated different amounts of muscle mass between the two legs and conditions might exist, which would make it difficult to compare blood flow between the two conditions. However, we do not believe that this is problematic because we stimulated both legs to the same absolute torque (~30 N·m). Isometric torque has been highly correlated to stimulated cross-sectional area as determined by transverse relaxation time in magnetic resonance imaging for SCI and able-bodied subjects (1, 16). These subjects were used in prior studies, and muscle mass was not found to be significantly different between legs as determined by magnetic resonance imaging (26, 28). This suggests that the absolute and relative amounts of activated muscle mass were similar between legs allowing comparison of blood flow.

In conclusion, this study confirms the results of our previous work that blood flow is delayed at the onset of exercise in chronic SCI individuals (28). This work clearly indicates that the delayed rise in blood flow in SCI individuals does not explain their high levels of muscle fatigue. Additional studies are needed to better understand the causes of increased fatigue in SCI patients. In particular, understanding the role of oxygen delivery in determining muscle function is needed. Also, the role of altered calcium kinetics in determining muscle fatigue is important. And finally, a better understanding of the potential role of structural alterations (muscle damage) in influencing muscle function is needed. These results, taken with our previous work in which the magnitude of blood flow was not related to muscle fatigue (28), indicate that blood flow is not limiting in SCI individuals and does not explain increased muscle fatigue.

GRANTS
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REFERENCES


