Blood flow and muscle fatigue in SCI individuals during electrical stimulation

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Olive, Jennifer L., Jill M. Slade, Gary A. Dudley, and Kevin K. McCully. Blood flow and muscle fatigue in SCI individuals during electrical stimulation. J Appl Physiol 94: 701–708, 2003.—Our purpose was to measure blood flow and muscle fatigue in chronic, complete, spinal cord-injured (SCI) and able-bodied (AB) individuals during electrical stimulation. Electrical stimulation of the quadriceps muscles was used to elicit similar activated muscle mass. Blood flow was measured in the femoral artery by Doppler ultrasound. Muscle fatigue was significantly greater (three- to eightfold, P < 0.001) in the SCI vs. the AB individuals. The magnitude of blood flow was not significantly different between groups. A prolonged half-time to peak blood flow at the beginning of exercise (fivefold, P < 0.001) and recovery of blood flow at the end of exercise (threeto-fivefold, P < 0.009) was found in the SCI vs. the AB group. In conclusion, the magnitude of the muscle blood flow to electrical stimulation was not associated with increased muscle fatigue in SCI individuals. However, the prolonged time to peak blood flow may be an explanation for increased fatigue in SCI individuals.

spinal cord injury; Doppler ultrasound

SPINAL CORD-INJURED (SCI) individuals are at high risk of heart disease, diabetes, and obesity due to extreme inactivity inherent with their condition (35). Physical activity has been reported to decrease the risk of heart disease by 50% in sedentary, healthy individuals, as well as decrease obesity and the risk of developing diabetes and increase bone strength (1). Thus current research has been interested in the use of exercise training via electrical stimulation in SCI individuals to lower their risk of disease. However, numerous problems exist with exercise training of SCI individuals, such as increased muscle fatigue (10, 20, 24), muscle (11) and vascular (Refs. 29, 38; J. L. Olive, G. A. Dudley, and K. K. McCully, unpublished observations) atrophy, as well as reduced cardiac output (15).

Muscle fatigue during volitional exercise can be defined as the failure to maintain force output that leads to a reduction in performance (19). Muscle fatigue can be explained by numerous mechanisms, which may include alterations in the neural system, muscle blood flow and availability of metabolites to the muscle, or muscle mechanical properties (18). The increased fatigability of affected skeletal muscle is associated with an increase in fast-fatigable fibers after injury due to extreme inactivity (11, 22, 59). Furthermore, impaired blood flow (17) and altered blood flow redistribution to affected muscles during exercise have been reported in SCI subjects and may contribute to increased muscle fatigue (15, 28). Limitations to maximal exercise performance in SCI individuals have been related to peripheral or muscle limitations and not to heart or lung limitations (27). Other possibilities for increased fatigue may include inadequate delivery of substrates (25, 47) or the buildup of metabolic by-products (3, 14).

Submaximal exercise elicits increases in blood flow to match the metabolic demands of the working skeletal muscle (5, 65, 66, 68), regardless of muscle size (52), contraction rate (41), or type of stimulation (34), in able-bodied (AB) individuals. During submaximal exercise, blood flow will increase rapidly until oxygen delivery matches demand (49, 65) and then plateau. The initial, rapid increase in blood flow at the onset of exercise is dependent on the muscle mechanical factors (first contraction) (62, 63) followed by vasodilation due to motor unit recruitment (first 5 s) (34, 45, 55, 66) or vasodilators such as adenosine, nitric oxide, or potassium ions (16, 31, 45).

SCI individuals have been shown to have significant vascular changes that include reductions in blood flow and arterial diameter size by as much as 50% to affected areas (7, 29, 38, 61). This reduction in diameter size is linked to muscle atrophy in SCI individuals (Olive et al., unpublished observations). SCI individuals have also been shown to have impaired vascular reactivity (inability to regulate vascular tone) (42) as measured by half time to recovery of blood flow (Olive et al., unpublished observations; Ref. 40). Thus it is not known whether alterations in the vasculature of SCI individuals impair muscle function.

Therefore, the purpose of this experiment was to measure muscle blood flow and muscle fatigue in SCI individuals during exercise via electrical stimulation. Doppler ultrasound was used in this study, a method...
that allows for the determination of absolute blood flow as well as blood flow kinetics (44, 45). Blood flow was measured during and after three different duty cycles of electrical stimulation while fatigue levels were measured. We hypothesized that the magnitude of blood flow would be lower in SCI individuals, which could contribute to their increased muscle fatigue. A second hypothesis was that SCI individuals would have a delayed recovery to exercise via electrical stimulation compared with the AB individuals.

METHODS

Subjects

Nine male chronic (>1 yr postinjury), complete (American Spinal Injury Association, category A) SCI subjects (8 paraplegics, 1 tetraplegic) and eight male AB subjects volunteered to participate in the study. The physical characteristics of the SCI subjects can be found in Table 1. All of the subjects in this study were part of another study (Olive et al., unpublished observations). The AB subjects, who served as controls, were included if they were similar to the SCI group in age, height, and weight and if they did not report exercising >3 days/wk and were not engaged in any formal exercise-training program for 6 mo before the study. Exercise history in the AB subjects was obtained by self-report. 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 in either group was antispasticity medication in the SCI group. Magnetic resonance (MR) image collection for all subjects occurred at Shepherd Center, Atlanta, GA, or Health South, Athens, GA. The study was conducted with the approval of the Institutional Review Board at the University of Georgia, and all subjects provide 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 (43). Blood pressure (BP) was measured in the arm throughout the entire testing period by using an automated BP machine (Datascope, Mahwah, NJ).

Electrical stimulation. Subjects were asked to sit on a specially made chair with a rigid lever arm positioned 70° below horizontal. A moment arm of 33 cm was established by mounting a load cell perpendicular to and 12 in. from the axis of rotation of the lever arm. 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 right 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 with the use of 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 over the vastus lateralis muscle. Subjects then received electrical stimulation inducing 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. This was done to elicit similar muscle activation between subjects (2, 24). Subjects underwent three different electrical stimulation bouts with a work-to-rest cycle of 1:6 (Low), 1:4 (Moderate), and 1:2 (High). These levels were chosen to obtain comparable levels of fatigue among groups while allowing adequate time between contractions for blood flow measurements by Doppler ultrasound. The electrical stimulation periods lasted 3 min each. The recovery period between exercise bouts was determined by a minimum of 5 min or as the time at which blood flow returned to baseline values, whichever was shorter. The AB group did not return to resting blood flow within the 5-min recovery period, whereas, in the SCI group, the time to return of baseline blood was ~4–10 min. The Low bout was always performed first and the High bout last to minimize the amount of fatigue in the subjects and to ensure that they could complete all exercise bouts. Muscle fatigue was calculated for each exercise bout as a percent decline in force production from the first five contractions to the last five contractions.

Blood flow. Blood flow was measured in the femoral artery by 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 heartbeat by GE’s advanced vascular program software for the GE Logiq 400 CL. The minimum, maximum, and time-averaged maximum velocity measurements were saved directly to a computer, thus 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 (CSA) and the time-average maximum velocity. The time-average maximum velocity was used in this study, despite the fact that it overestimates the mean blood flow response (~40%), as it was considered a more reliable measure of the blood flow response. The resting vessel diameter was used for calculation of blood flow, as no dilation was found in the femoral artery during exercise. The observation that the femoral artery does not dilate with exercise has been reported previously (44) and has been verified in our laboratory with exercise (unpublished observations) and cuff occlusion (40). Conductance was calculated by the blood flow divided by the mean arterial...
pressure. Half time to peak blood flow was determined as the time at which 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 (45, 63). The half time to recovery was determined as the time at which blood flow dropped to one-half the magnitudes between maximum flow and resting flow. This was used as an index of vascular reactivity, as it indicates the ability of the arteries to return blood flow to resting values (42).

**MR imaging.** All MR images to calculate muscle volume were used in a previous study (Olive et al., unpublished observations). Skeletal muscle average CSA was determined by using proton-weighted MR imaging. This method has been shown to be highly reproducible and reliable for determination of skeletal muscle composition (37). MR images of both legs and thighs were collected with a 1.5-T magnet (500-ms repetition time, 14-ms echo time, 40-cm field of view, 1 number of excitations, 256 × 256 matrix; General Electric, Milwaukee, Wisconsin). Transaxial images, 1 cm thick and 0.5-cm slice gap, were taken from the hip joint to the knee joint (thigh) by using a whole body coil. Each of the subject’s feet was strapped to a brace to maintain the ankle joint at ~90° and the knee and hip joint extended.

Data were downloaded to a disk and analyzed on specifically designed software (X-Vessel, East Lansing, MI). The methodology for MR image data analyses has been reported previously (33). In brief, images were automatically segmented into fat (high-intensity), muscle (mid-intensity), and background and bone (low-intensity) regions as follows. A preliminary segmentation of each two-dimensional slice into fat and nonfat regions was obtained by simplex optimization of the correlation between a Sobel-gradient image computed from the original image vs. the corresponding gradient images computed from single-threshold, fat-segmented images (21). This first-pass segmentation was used to correct for intensity variations across the original image caused by radio-frequency heterogeneity (13). The corrected original image was then resegmented into the three intensity components by using a fuzzy c-mean clustering algorithm (58). Manual selection of any pixel of muscle subsequently highlighted all muscle pixels in the region and provided a total number of muscle pixels exclusive of any fat or low pixels. A manual tracing was drawn around the outside edge of the quadriceps muscles to determine quadriceps muscle mass to allow for determination of active muscle mass. For each subject, thigh CSA was determined with the first slice not containing gluteal muscle and continuing distally toward the knee until the patella could be seen. The individual collecting the data performed two trials on sample images to determine test-retest reliability ($r = 0.99$). Pixel number was converted to area by multiplying by the field of view and dividing by the total number of pixels in the entire image.

**Statistical Analysis**

Independent sample $t$-tests (SPSS version 10.0) were conducted to compare for differences in subject characteristics and blood vessel diameter between the two groups. The data were analyzed to verify normality and to test for any outliers. 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 the AB and SCI groups across exercise bouts for blood flow, conductance, half time to peak flow, or half time to recovery. Mauchly’s test was conducted to determine whether sphericity was violated. If sphericity was violated, the repeated-measures ANOVA was corrected by using the Greenhouse-Geisser correction factor and the value for epsilon ($\epsilon$) was reported. Effect size was calculated using Cohen’s $d$, $\eta^2$, or $\epsilon^2$. Missing data for half time to recovery or half time to peak blood flow were replaced by using a regression model (51). All analyses were conducted at a significance level of 0.05.

**RESULTS**

**Baseline Data**

The AB and the SCI groups were similar in age, height, and weight (Table 1). There were no significant differences between the SCI and AB groups at rest in heart rate (HR) or BP (BP: 126/76 vs. 124/78 Torr; HR: 71 vs. 75 beats/min for AB vs. SCI). The tetraplegic subject was not a statistical outlier on any parameter and thus was included in the analyses. The SCI individuals had significantly smaller (~40%) quadriceps muscle CSA ($t(15) = 5.560, P < 0.001, d = 2.7$) and volume ($t(15) = 5.094, P < 0.001, d = 2.5$) than the AB group (Fig. 1). Total thigh muscle CSA ($t(15) = 4.264, P = 0.001, d = 2.1$) and volume ($t(15) = 4.250, P = 0.001, d = 2.1$) were significantly smaller (~40%) in the SCI compared with the AB group as well. Absolute resting blood flow (ml/min) was not different between groups (180 ± 120 vs. 179 ± 93 ml/min for AB vs. SCI).

**Muscle Fatigue**

There was a significant group-by-exercise bout interaction for muscle fatigue [$F(2,30) = 23.028, P < 0.001, \eta^2 = 0.606$]. Thus independent sample $t$-tests were conducted to determine differences between groups. The SCI group had three to eight times the amount of fatigue compared with the AB group during each exercise bout ($t(15) = 4.070, P = 0.001, d = 2.0$; $t(15) = 4.628, P < 0.001, d = 2.3$; and $t(15) = 7.863, P < 0.001, d = 3.9$ for Low, Moderate, and High, respectively). Repeated-measures ANOVAs were conducted to determine differences in fatigue across workloads for each group. Fatigue increased significantly with increasing workload in the AB [$F(2,14) = 67.464, P < 0.001, \eta^2 = 0.84$].

![Fig. 1. Largest quadriceps muscle cross-sectional area (CSA) and total quadriceps muscle volume in spinal cord-injured (SCI) and able-bodied (AB) individuals. Values are means ± SD. *Significant difference between groups, $P < 0.001$.](image-url)
Exercise Hyperemia

All subjects were able to complete all three stimulation periods. There were no significant differences in HR or BP between the groups or during the stimulation periods. A representative blood flow response to a Low workload is shown for a SCI and an AB individual (Fig. 3). Blood flow increased significantly during each exercise bout from resting flow values ($t(12) = 7.299, P < 0.001, d = 2.5$; $t(12) = 6.823, P < 0.001, d = 2.5$; and $t(12) = 6.790, P < 0.001, d = 2.5$ for Low, Moderate, and High, respectively) and was maximal at the end of the exercise bout. The peak blood flow at the end of exercise increased with increasing intensity of exercise [$F(2,30) = 19.118, P < 0.001, \eta^2 = 0.560$] (Fig. 4).

There was no difference in blood flow between groups ($P = 0.352$). Conductance was significantly different within exercise periods [$F(2,30) = 17.805, P < 0.001$, $\eta^2 = 0.543$], with no difference between groups. The conductance followed the trends of blood flow, as would be expected, because there were no changes in mean arterial pressure throughout exercise.

As demonstrated in Fig. 3, the magnitude of blood flow was similar between groups, but the half time to peak and recovery of blood flow was significantly prolonged (18 vs. 54 s, 8 vs. 42 s, and 8 vs. 38 s for the AB vs. SCI group at Low, Moderate, and High, respectively) in the SCI individual. The SCI individual illustrated in Fig. 3 has a prolonged half time to peak flow compared with the average response, but the figure is illustrative of the type of responses that were seen in each population. The onset and recovery of blood flow were prolonged across all exercise bouts in the SCI compared with AB groups, as measured by half time to peak blood flow [$F(1,14) = 15.508, P = 0.001, \eta^2 = 0.526$] (Figs. 3 and 5) and half time to recovery [$F(1,15) = 52.905, P < 0.001, \eta^2 = 0.779$] (Figs. 3 and 6). The half time to recovery was approximately three
times longer in the SCI compared with the AB group. Half time to recovery of resting blood flow was longer with increasing intensities of exercise for both groups \( F(1.450,21.743) = 6.857, P = 0.009, \eta^2 = 0.314, \epsilon = 0.725 \), with significant differences occurring between the Moderate- and High-exercise bouts \( P = 0.001 \).

**DISCUSSION**

We found that the magnitude of blood flow response was similar between SCI and AB individuals at three exercise bouts, even though the SCI group had significantly more muscle fatigue than the AB group. This result suggests that the magnitude of blood flow is not limiting during electrical stimulation of the quadriceps muscles and did not explain the increased fatigue seen in SCI individuals. Second, SCI individuals had prolonged blood flow kinetics at the onset of exercise and during recovery from exercise, which may be evidence of abnormal oxidative ability and impaired vascular reactivity.

A unique aspect of this study was that we activated similar amounts of muscle mass (~16 cm\(^2\)) in both groups, allowing comparison of blood flows during exercise between SCI and AB individuals. To do this, we stimulated all subjects to the same absolute torque (~30 N·m), as isometric torque has been highly correlated to stimulated CSA as determined by transverse relaxation time MR imaging for SCI and AB individuals (unpublished laboratory observations) (2, 24). Calculation of activated muscle indicated that we activated ~24% of AB and 42% of the SCI individuals' quadriceps muscle mass. It is possible that activating different proportions of muscle mass may influence blood flow response to electrical stimulation. This is a potential limitation to the study and is a consequence of attempting to compare subjects who have different muscle sizes. An alternative approach would have been to activate the same proportion of muscle mass and normalize flow values to the amount of activated muscle mass. Comparing subjects in this manner, however, requires determination of activated muscle mass and presents difficulties with scaling (4, 60).

We found a three- to eightfold increase in the rate of muscle fatigue in the SCI compared with the AB group at three work-rest cycles, a finding consistent with previous literature (10, 20, 24). This increase in fatigue after SCI has been associated with changes in myosin heavy chain structure from slow-oxidative to fast-glycolytic fibers (22, 50, 59), decreased mitochondrial content (8, 36), and alterations in the vasculature or impairments in blood flow (15, 17, 27, 28).

We found no difference in the magnitude of the blood flow response at the end of exercise via electrical stimulation, demonstrating that absolute levels of muscle blood flow for a given amount of activated muscle mass are the same between groups with extremely different muscle mass and fatigue rates. Our results indicate that absolute muscle blood flow is not limiting in SCI individuals and does not explain the increased fatigue rates. Alterations in systemic pressure or HR may influence leg blood flow during electrical stimulation in paraplegic subjects (15). However, we did not find any changes in BP or HR during the stimulation bouts for either group, suggesting that systemic alterations did not occur and did not influence leg blood flow during the exercise bouts. The lack of changes in BP or HR during the stimulation is most likely due to the relatively small amount of muscle activation in this study. Blood flow increased significantly with increases in work level in both groups, a finding that is consistent with previous work in which muscle blood flow increases in proportion to the intensity of the exercise bout (5, 65, 66, 68). These findings have been shown, regardless of muscle size (52), contraction rate (41), or type of stimulation (34).

The time for blood flow to increase was significantly prolonged (approximately equal to fivefold) in the SCI group (Figs. 3 and 5). The time for blood flow to increase is dependent on muscle mechanical factors, vasodilator ability, and oxidative capacity (45, 62, 63, 65). A prolonged time to peak flow could indicate that one
or more of these mechanisms are impaired in SCI individuals. The prolonged rise in blood flow could be explained by decreased muscle pump activity, which has been reported in SCI subjects (64). A second explanation could be that blood flow response is slowed due to decreased oxidative capacity (31, 44), as is seen by reduced oxidative enzyme capacity and alterations of muscle fiber type from slow-oxidative fibers to fast-fatigable fibers in SCI individuals (8, 36). Last, the prolonged rise in blood flow could be due to impaired vessel reactivity, evidenced either as reduced vessel response to vasodilators or a reduction in the amount of vasodilators produced. Impaired vessel reactivity is prevalent in diseased populations (9, 12, 56). Furthermore, our laboratory has demonstrated that the reactive hyperemic response is impaired in SCI subjects, indicating reduced vascular reactivity (Olive et al., unpublished observations; Ref. 40).

A delayed or reduced blood flow response at the onset of exercise alters oxidative metabolism in healthy individuals (23, 26, 30) and results in increased muscle fatigue in healthy working skeletal muscle (25, 48). Limited blood flow during exercise may also result in increased levels of fatigue due to an accumulation of metabolic by-products (3, 14). If flow is limited at the onset of stimulation in SCI patients, it could limit oxidative metabolism and could be one cause of the increased muscle fatigue, even though absolute muscle blood flow was not limiting.

Half time to recovery of blood flow after electrical stimulation was also prolonged (approximately equal to threefold) in the SCI individuals (Figs. 3 and 6). Half time to recovery has been used as an index of vascular reactivity, as the blood flow response after exercise is dependent on the accumulation of metabolic factors and vasoactive substances such as nitric oxide and adenosine (6, 46, 53). Thus a longer half time to recovery after exercise would indicate a greater buildup of metabolic factors and/or vasoactive substances, altered sympathetic activity, and/or a diminished ability to remove them. The prolonged time to recovery is consistent with our previous results in both incomplete (40) and complete SCI individuals (Olive et al., unpublished observations) after cuff occlusion. Activity level has been shown to affect vascular reactivity. Exercise training was found to improve vascular reactivity (42), and inactivity decreased vascular reactivity as evidenced by decreased vasoconstrictor ability (54) and increased vascular resistance after a stressor (32). Furthermore, abnormal vascular reactivity has been associated with many diseases, including diabetes (57) and heart disease (12). Consequences of reduced vascular reactivity could be abnormal oxidative metabolism, increased insulin insensitivity, and/or hypertension (67), all of which are prevalent in SCI individuals (35, 36).

A limitation to this study is that the SCI subjects may not have reached peak blood flow by the end of the electrical stimulation bout, as suggested in Fig. 3. In a few of the SCI subjects at the Low level, the increase in blood flow was slow enough that an obvious plateau was not reached during the stimulation. A longer stimulation period may have ensured that a plateau in blood flow would be reached during stimulation. However, we did not choose to use longer stimulation periods to minimize the impact of performing repeated trials. The lack of a plateau for the stimulation duration means that we may have underestimated the peak blood flow values in our SCI group. We do not think that the underestimation of peak blood flow would have changed our results significantly for the following reasons. First, the blood flow during stimulation did not plateau in only a few SCI subjects and thus would have only a small effect on the mean peak flow. Second, the slight underestimation of peak blood flow in the SCI group would have made the nonsignificant difference between groups even smaller. Another limitation to this type of study is that blood flow measurements via Doppler ultrasound are difficult to obtain during electrical stimulation. The difficulty in obtaining blood flow, especially at the High workload, made it impossible to calculate the half time to peak blood flow in some subjects, hence the use of a linear regression model to replace a few missing data points for the half time to peak blood flows. Thus confirmation of the onset kinetics needs to be conducted in future studies.

In conclusion, this study has shown that the magnitude of muscle blood flow during exercise via electrical stimulation was similar between groups and does not explain the increased muscle fatigue in SCI individuals. However, the prolonged rise in blood flow at the onset of stimulation may be one explanation for increased fatigue, even though absolute muscle blood flow was not limiting. Last, we found evidence that SCI individuals have decreased vascular reactivity, which may have negative health consequences.

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