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Effect of Graded Electrical Stimulation on Blood Flow to Healthy Muscle

DEAN P. CURRIER,
CYNTHIA REED PETRILLI,
and A. JOSEPH THRELKELD

The purpose of this study was to determine whether 2,500-Hz sine-wave electrical stimulation modulated at 50 bursts per second producing graded muscular responses affects blood flow. Healthy volunteer subjects were assigned randomly to an Experimental group (n = 14) that received bursts of electrical stimulation to the gastrocnemius muscle or to a Control group (n = 14) that received no treatment. Using a Doppler device, pulsatility index (PI) values were determined for multivariate statistical analysis. Electrical stimulation graded to simulate isometric torques equivalent to 10% and then 30% of the subjects' isometric maximum voluntary contraction resulted in respective mean increases in PI values of 20.5% and 19.6% over prestimulation PI values. We found no significant difference in PI values between the two levels of torque. No significant change in PI values was found among the Control group subjects. Our results indicate that electrical stimulation, as used in this study, can alter the blood flow to the muscle being stimulated.

Key Words: Blood circulation, Electric stimulation, Physical therapy.

Electrical stimulation has many forms and uses. Much of the current popularity of electrical stimulation as a therapeutic and training mode may be attributed to the Russian investigator Kots. His claims concerning the benefits of electrical stimulation include that of increased blood flow (hyperemia) to the muscle being artificially contracted. This article explores the use of specific electrical current to achieve increased blood flow to the stimulated muscle.

Blood flow to skeletal muscle during and after volitional exercise is controlled principally by local metabolism. The magnitude and frequency of active muscular contractions also affect the blood flow. Because muscle metabolism increases in response to voluntary contractions, blood flow to the active musculature also increases. Because electrical current produces muscle contractions, the metabolism of the muscle being stimulated also should increase. As a result, blood flow to a muscle should increase in response to the electrical stimulation of that muscle.

Some research has investigated the effects of different magnitudes of muscle force on blood flow. Early research by Barcroft and Millen showed that weak, sustained static contraction of the triceps surae muscles at 10% of maximum voluntary contraction (MVC) caused increased blood flow to the muscle during contraction without a further increase in the flow after exercise ceased (postexercise hyperemia). The authors also reported that voluntary muscular contractions of 30% of MVC resulted in decreased blood flow during the contractions but that blood flow increased greatly after the contractions. Later research by Barcroft and Dornhorst showed that, during rhythmic voluntary muscle exercises, blood flow decreased during the sustained contraction phase in a manner similar to that of a static contraction. The immediate postexercise increase in blood flow, however, was less with rhythmic exercise than with static exercise. Recently, Richardson and Shewchuk reported that a postexercise increase in blood flow to the calf muscles was augmented by increasing the frequency and force of active muscular contractions.

Investigators before Kots have shown that blood flow is altered in response to electrical stimulation. Wakim applied both direct and percutaneous continuous electrical stimulation of the nerve to specific canine leg muscles. He reported that maximum blood flow to the muscle resulted from stimulation frequencies in the range of 8 to 32 Hz. Randall et al reported that greater hyperemia resulted from continuous electrical stimulation of a canine muscle than from active contraction of the muscle. Folkow and Halicka found that blood flow to the gastrocnemius muscle of the cat progressively increased when they increased continuous electrical stimulation at rates of 1, 2, and 4 Hz. Blood flow progressively decreased, however, as the stimulus frequency was increased to 8, 16, 20, 30, and finally 60 Hz. Using various stimulus frequencies and levels of muscular contractile forces, Petrofsky et al found that, in cats, blood flow increased during most levels of contractile force. They also reported that, at all levels of contractile force and stimulus frequencies, blood flow increased to an even greater extent immediately after contraction.

The cited studies did not use a "new generation" electrical stimulator similar to that used by Kots because until 1979 such instrumentation was unavailable to researchers outside of Russia. This new...
instrumentation delivers a high intensity (>150 V, 0-100 mA output), interrupted sine wave. The purpose of this study was to determine whether electrical stimulation similar to the type used by Kots would produce alterations in blood flow to a muscle during or after electrically produced contractions. The null hypotheses were 1) subjects who receive electrical stimulation with a new generation stimulator would have blood flow no different than that of nonstimulated control subjects and 2) there would be no difference in the blood flow of experimental subjects who receive electrical stimulation that produces graded isometric muscular contractions equivalent to 10% versus 30% of MVC.

METHOD

Subjects

Twenty-eight subjects (10 men and 18 women) ranging in age from 20 to 35 years participated in this study. All subjects were healthy volunteers with normal resting heart rates and blood pressures. All subjects signed an informed consent statement after being briefed on the purpose and procedures of the study. The subjects then were randomly assigned to either an Experimental group (6 men and 8 women) or a Control group (4 men and 10 women). Table 1 gives the demographic information about the subjects.

Measurement of Ankle Torque

Baseline torque measurements were made for the purpose of determining the MVC torque and the intensity of electrical stimulation required to stimulate 10% and then 30% of MVC torque in the Experimental group subjects. No torque or electrical stimulation measurements, therefore, were collected from the Control group subjects.

During measurements of torque, Experimental group subjects sat on a test table in the long sitting position with their knees extended. No backrest or handgrips were provided and the subjects placed their hands behind them on the table with their arms extended to support their trunk. We secured the foot of the subject's test leg to the isokinetic dynamometer in the neutral position (0° of dorsiflexion) and the subject's calf to the test table with a webbed strap. The dynamometer was adjusted to record isometric torque (0°/sec). All subjects in the Experimental group executed three isometric MVCs of their ankle plantar flexor muscles. We recorded the resultant torques with a dynamometer interfaced with a strip chart recorder.* A rest period of two minutes was interposed between contractions. The highest torque score of the three contractions was used as the subject’s MVC.

While the subjects were still seated with their feet secured to the dynamometer, their right posterior calf muscles were electrically stimulated with surface electrodes (8-cm diameter, flexible rubber) secured with Velcro straps to the skin over the motor points of the medial and lateral portions of the gastrocnemius muscle. Moistened sponges inserted between the skin and the electrodes served as conductive couplings. The stimulus intensities (current strength) were adjusted to produce torque scores equivalent to 10% (\(X = 11.2\) mA, range = 6-20 mA) and then 30% (\(X = 14.8\) mA, range = 8-24 mA) of the subjects' measured MVC. The individual stimulus intensities were recorded and duplicated during the stimulation phase of the experiment.

Measurement of Blood Flow, Heart Rate, Blood Pressure, and Skin Temperature

The subjects in the Experimental group maintained a prone position on a treatment table for about 15 minutes while prestimulation preparations and measurements were made. We used a dual-frequency, continuous wave, ultrasonic directional Doppler device† to quantify the blood flow of the calf muscles. The pencil probe of the Doppler device was hand-held and positioned on the skin over the popliteal artery of the prone subjects so that an angle of about 45 degrees was formed with respect to the horizontal plane. The probe position was adjusted empirically by one of the investigators (C.R.P.) for each measurement to yield a maximum signal. This technique of Doppler placement has been shown to be a reliable measure of blood flow changes over time.12 Twenty to 25 consecutive signals, each representing a complete cardiac (pulse) cycle, were transcribed from the Doppler device to a multichannel pen recorder, and the resultant tracings later were analyzed. Prestimulation Doppler recordings (t = 0 minutes) were obtained from both groups. Subsequent Doppler recordings were made at intervals of 1, 5, and 10 minutes after the onset of stimulation (t = 1, 5, and 10) and at intervals of 1, 3, and 5 minutes after electrical stimulation was terminated (t = 11, 13, and 15).

Except for the time we spent determining the ankle torque, all subjects remained in the prone position for the electrical stimulation and Doppler readings. Prestimulation resting heart rates and blood pressures were obtained from the right arms of all subjects. Heart rate was measured by palpation of the radial artery. Blood pressure was measured with a sphygmomanometer while auscultating the brachial artery. Skin temperature was monitored with a telethermometer§ and a thermistor disk attached to the plantar surface of the forefoot of each subject. Temperature readings were recorded before and after the experimental period.

Electrical Stimulation

An Electrostim 180-21 stimulator (simulated new generation electrical stimulator) provided the electrical stimuli to produce isometric contractions of the right calf musculature of the subjects in the Experimental group. The stimulator produced carrier sine waves at a frequency of 2,500 Hz in modulated bursts at a fixed rate of 50 bursts per second. The bursts of stimuli had an intensity that was finely ramped (rise or surge) so that the current of each series of bursts gradually increased over a five-

### Table 1

<table>
<thead>
<tr>
<th>Group</th>
<th>Age (yr)</th>
<th>Weight (kg)</th>
<th>Height (cm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Experimental</td>
<td>22.5</td>
<td>64.4</td>
<td>172.6</td>
</tr>
<tr>
<td>Control</td>
<td>24.5</td>
<td>60.5</td>
<td>169.9</td>
</tr>
</tbody>
</table>

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* Cybex, Div of Lumex, Inc. 2100 Smithtown Ave, Ronkonkoma, NY 11779.
† Model 909, Parks Electronics Lab, Beaver, OR 97095.
‡ Model 7, Grass Instrument Co. 101 Old Colony Ave, Quincy, MA 02169.
§ Model 47 Ta, Yellow Springs Instrument Co. Box 279, Yellow Springs, OH 45387.
¶ Micromed Instruments, 4996 Place de la Savane, Montreal, Canada H4B 1R6.
second period but had an abrupt intensity fall time ("off" ramp). A 15-second period of stimulation was followed by a 50-second rest period (15/50 duty cycle). The intensity of the stimulus bursts was adjusted to produce graded muscular contractions equivalent to 10% and 30% of the subject's isometric MVC torques as measured before this experimental phase with an isokinetic dynamometer. The subject's right foot was secured in a fixed position against a barrier with the ankle maintained in the neutral position in order to produce isometric contractions.

Serial observations of the Doppler values of the first five Experimental group subjects indicated that, after the 5-minute poststimulation (t = 15) blood flow measurement (10% MVC), a 10-minute recovery period was sufficient time for the return of blood flow to prestimulation resting levels. After this period of recovery, we conducted a second series of electrical stimulations and measurements. During this second phase of the experiment, we used a stimulus intensity sufficient to produce 30% of MVC. Upon termination of the 5-minute poststimulation Doppler measurements (30% MVC), we again recorded the subjects' heart rates, blood pressures, and skin temperatures.

We used a similar procedure to record the positioning and blood flow measurements of the Control group subjects. The exception was that the Control group subjects were not measured for torque and did not receive any electrical stimulation to elicit contractions of the plantar flexor muscles or any other form of treatment.

**Data Analysis**

We used the pulsatility index (PI) as a quantitative measure of blood flow. Gosling and King defined PI as the mean peak-to-peak magnitude of the Doppler signal divided by the mean cardiac-cycle time averaged over several cardiac cycles. We used the mean amplitude and mean cardiac-cycle times of the centermost 15 cycles of 20 to 25 signals recorded for each measurement of each subject in our analysis. That is, each PI value represents the mean of 15 complete analog signals recorded for each group of Doppler measurements and converted to a single value. This technique provides a ratio that is both a valid measure of peripheral circulation and a normalized signal that is relatively independent of the Doppler probe angle, distance of the probe from the vessel, and segment of the vessel being measured.

A two-way, fixed-effects, multivariate analysis of variance (MANOVA) was performed using the PI data. The Duncan method of a posteriori analysis was used for significant F ratios.

Independent t tests (two-tailed) were used to determine differences between mean prestimulation (t = 0) PI values. Paired t tests (two-tailed) were used to determine differences between the mean initial and final heart rates, blood pressures, and skin temperature variables for both groups of subjects. Independent t tests (two-tailed) were used to determine prestimulation and poststimulation differences between the mean heart rate, blood pressure, and skin temperature values of the two groups. The significance level was set at .05.

**RESULTS**

Descriptive data for the PI values are shown in Table 2. Univariate F tests revealed no significant differences between the mean prestimulation (t = 0) PI values of the Experimental group and the Control group (Tab. 3). The first minute of electrical stimulation (t = 1) of the gastrocnemius muscle at both 10% and 30% of MVC resulted in significantly increased PI values when compared with the PI values of the Control group. The Experimental group's mean PI values remained significantly higher than the Control group's mean PI values between the 5-minute stimulation (t = 5) and 5-minute poststimulation (t = 15) intervals at both 10% and 30% of MVC (Figs. 1, 2). We observed no significant difference between the mean increase in PI values at 10% of MVC and the mean increase in PI values at 30% of MVC in the Experimental group at any of the time intervals during which measurements were recorded (Fig. 3).

Table 4 presents data on heart rate, blood pressure, and skin temperature. The mean heart rate of the Experimental group decreased significantly over time between the prestimulation and the poststimulation measurements. The mean prestimulation heart rate of the Experimental group also was significantly higher than the mean prestimulation heart rate of the Control group, but we found no significant difference.

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**TABLE 2**

Means and Standard Deviations of Pulsatility Index

<table>
<thead>
<tr>
<th>Group</th>
<th>Torque Level</th>
<th>Interval in Time (min)</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Prestimulation</td>
<td>0</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Stimulation Intervals</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Poststimulation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Experimental</td>
<td>10% MVC*</td>
<td>X</td>
<td>11.2</td>
<td>13.3&lt;sup&gt;b&lt;/sup&gt;</td>
<td>12.4&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td></td>
<td>s</td>
<td>4.3</td>
<td>4.5</td>
<td>4.6</td>
</tr>
<tr>
<td></td>
<td>30% MVC*</td>
<td>X</td>
<td>11.2</td>
<td>13.5&lt;sup&gt;b&lt;/sup&gt;</td>
<td>13.0&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td></td>
<td>s</td>
<td>4.3</td>
<td>4.8</td>
<td>4.4</td>
</tr>
<tr>
<td>Control</td>
<td></td>
<td>X</td>
<td>9.9</td>
<td>10.0</td>
<td>9.9</td>
</tr>
<tr>
<td></td>
<td></td>
<td>s</td>
<td>2.2</td>
<td>1.7</td>
<td>1.8</td>
</tr>
</tbody>
</table>

<sup>a</sup> Maximum voluntary contraction.

<sup>b</sup> Significantly higher than control, p < .025.
between the mean poststimulation heart rates of the two groups. The initial and final mean heart rates of the Control group subjects were not significantly different.

We observed no significant change between the initial and final mean systolic blood pressure measurements in either the Experimental group or the Control group. We did find a significant difference, however, between the initial mean diastolic blood pressures of the two groups (p < .05). This difference between groups was not found for the final mean diastolic blood pressures.

We found no significant difference between the initial mean skin temperatures of the Experimental group and the Control group. The mean skin temperatures of both groups decreased significantly between the initial and the final measurements.

**DISCUSSION**

Our finding of increased PI values is in agreement with other researchers who found increased blood flow to muscle during volitional contraction and electrical stimulation. The amount of relative circulatory change in our study, however, was far less than that reported by other researchers. We found no poststimulation hyperemia, whereas other researchers consistently reported this postexercise response.

A general increase in blood flow during muscular contractions at low force levels (less than 20% of MVC) has been reported in humans and in animals. Our data show a rapid initial increase in blood flow followed by a plateau of the PI values that was maintained at a relatively steady state throughout the period of electrical stimulation (muscle contraction). An initial increase in blood flow is an expected result of volitional muscular contractions (eg, exercise).

The Electrostim 180-2 stimulator apparently is capable of simulating the effect of volitional muscular contractions of 10% and 30% of MVC because it produced a sudden increase in blood flow. The results we obtained, thus, support Richardson’s report of increased blood flow concomitant with the onset of volitional muscular contractions that reached steady-state levels within 30 seconds. Folkow and Halicka’s data show steady-state levels of blood flow during intermittent muscular contractions.

**TABLE 3**

Summary of Multivariate and Univariate Analyses of Variance Significance Tests

<table>
<thead>
<tr>
<th>Group effects at 10% MVC vs control</th>
<th>Multivariate</th>
<th>Univariate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Main Effects and Dependent Variable</td>
<td>df</td>
<td>F</td>
</tr>
<tr>
<td>Group effects at 10% MVC vs control</td>
<td>6, 21</td>
<td>2.61</td>
</tr>
<tr>
<td>- Prestim</td>
<td>1, 26a</td>
<td>6.73</td>
</tr>
<tr>
<td>- Stim</td>
<td>1 min</td>
<td>5.71</td>
</tr>
<tr>
<td>- 5 min</td>
<td>1 min</td>
<td>6.95</td>
</tr>
<tr>
<td>- 10 min</td>
<td>5 min</td>
<td>12.22</td>
</tr>
<tr>
<td>Group effects at 30% MVC vs control</td>
<td>6, 21</td>
<td>3.08</td>
</tr>
<tr>
<td>- Prestim</td>
<td>1, 16a</td>
<td>6.26</td>
</tr>
<tr>
<td>- Stim</td>
<td>1 min</td>
<td>11.21</td>
</tr>
<tr>
<td>- 5 min</td>
<td>3 min</td>
<td>15.78</td>
</tr>
<tr>
<td>- 10 min</td>
<td>5 min</td>
<td>15.78</td>
</tr>
<tr>
<td>Group effects at 10% vs 30% MVC</td>
<td>6, 8</td>
<td>1.64</td>
</tr>
<tr>
<td>- Prestim</td>
<td>1, 13a</td>
<td>0.03</td>
</tr>
<tr>
<td>- Stim</td>
<td>1 min</td>
<td>0.50</td>
</tr>
<tr>
<td>- 5 min</td>
<td>10 min</td>
<td>0.22</td>
</tr>
<tr>
<td>- Poststim</td>
<td>1 min</td>
<td>0.25</td>
</tr>
<tr>
<td>- 3 min</td>
<td>5 min</td>
<td>3.69</td>
</tr>
<tr>
<td>- 5 min</td>
<td>0.01</td>
<td>NS</td>
</tr>
</tbody>
</table>

- Maximum voluntary contraction.
- Degrees of freedom for all intervals of time.

**Fig. 1.** Means and standard deviations of pulsatility index values of the popliteal artery during prestimulation, stimulation, and poststimulation phases at current amplitudes producing 10% of maximum voluntary contraction of the gastrocnemius muscles of Experimental group subjects. Control group subjects did not receive electrical stimulation.
These investigators, however, also found that stimulus frequencies between 30 to 60 Hz caused considerable "squeezing" effects that decreased blood flow during muscle contractions. Similar observations of decreased blood flow during muscle contractions that generated forces of 20% of MVC were ascribed to mechanical compression of the blood vessels by the muscle tissue. Donald et al labeled these interfering pressures as mechanical "nipping" of the distributing arteries. This explanation implies that a steady rise in blood flow during exercise is mechanically attenuated and masked during contraction, then resurges during the postexercise period as the postexercise hyperemia. This additional postexercise rise in blood flow was not seen in our study.

Electrical stimulation intensities sufficient to elicit isometric contraction forces equivalent to 10% and 30% of MVC resulted in mean PI increases of 20.5% and 19.6% over prestimulation and 5-minute poststimulation values, respectively. The degree of reported blood flow increase differs among the various cited studies. Richardson found an increase in blood flow of about 50% over resting levels in humans performing volitional exercises of 7.5% and 15% of MVC effort. Blood flow increases in animals that were electrically stimulated ranged from 83% to 109% and 35% to 135% in the Wakim and Randall et al studies, respectively. The method used to enhance blood flow was different in each of the studies cited, and this difference may account for the variable alterations of blood flow reported. In our study, the muscle was stimulated indirectly through the skin, whereas in the animal studies the muscles were stimulated directly with electrodes inserted into the muscle belly. Increased (poststimulation or postexercise) hyperemia apparently occurs to meet the metabolic demands of musculature partially

**Fig. 2.** Means and standard deviations of pulsatility index values of the popliteal artery during prestimulation, stimulation, and poststimulation phases at a torque level of 30% of maximum voluntary contraction of the gastrocnemius muscles of Experimental group subjects. Control group subjects did not receive electrical stimulation.

**Fig. 3.** Means and standard deviations of pulsatility index values of the popliteal artery during prestimulation, stimulation, and poststimulation phases at torque levels of 10% and 30% of maximum voluntary contraction of the gastrocnemius muscles of Experimental group subjects.

### TABLE 4

Means and Standard Deviations of Physical Characteristics of Subjects

<table>
<thead>
<tr>
<th>Group</th>
<th>Torque (N·m)</th>
<th>Heart Rate (bpm)</th>
<th>Blood Pressure (mm Hg)*</th>
<th>Temperature (°C)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Prestimulation</td>
<td>Poststimulation</td>
<td>Prestimulation</td>
<td>Poststimulation</td>
</tr>
<tr>
<td>Experimental</td>
<td>108.3</td>
<td>83.9</td>
<td>75.4</td>
<td>123.3/78.7</td>
</tr>
<tr>
<td></td>
<td>39.6</td>
<td>9.2</td>
<td>10.6</td>
<td>5.7/3.8</td>
</tr>
<tr>
<td>Control</td>
<td>X</td>
<td>72.4</td>
<td>69.1</td>
<td>120.1/73.9</td>
</tr>
<tr>
<td></td>
<td>s</td>
<td>12.6</td>
<td>12.3</td>
<td>15.7/6.8</td>
</tr>
</tbody>
</table>

* Systolic/diastolic value.
deprived of adequate blood supply by mechanical interference during exercise. The threshold for this phenomenon seems to be when static contractions are produced at tension levels of 20% or more of MVC. In the poststimulation phase of our study, the PI did not increase above the steady-state levels that we recorded during the stimulation phase and that produced torques equivalent to 10% and 30% of MVC. The placement of the surface electrodes in our study probably elicited greater contraction of the posterior calf muscle nearest the electrode (ie, the gastrocnemius muscle). Because the gastrocnemius muscle is composed mostly of fast glycolytic fibers, its metabolism would be primarily anaerobic. Multiple contractions would cause a large oxygen debt in the gastrocnemius muscle, resulting in the poststimulation hyperemia reported by other researchers.

Each stimulation cycle in our study consisted of 15 seconds of electrical stimulation followed by 50 seconds of rest (no electrical stimulation). These electrical stimulation conditions may have been insufficient to occlude mechanically or interfere with the blood supply. That is, the low isometric contraction intensity (10% and 30% of MVC) and the short duration of the electrical bursts (15 seconds), coupled with an increased nutritive flow to the muscle over a 50-second rest period, may not have allowed the gastrocnemius muscle to release or to accumulate sufficient amounts of vasactive substance to produce postexercise hyperemia. Our results agree with the findings of Folkow and Halicka that muscle that was intermittently contracted with brief interruptions (at frequencies of 8, 16, 20, 30, and 60 per sec) reached a steady state of blood flow to the muscle. Other researchers who did report hyperemia after electrical stimulation used stimuli that were applied continuously to the muscle. Wakim stimulated the muscle continuously for 15 minutes, and Randall et al stimulated the muscle for periods of 0.5, 1.0, and 2.0 minutes.

Wakim reported that frequencies of 4, 8, 16, and 32 Hz increased blood flow more effectively than frequencies of 64, 128, and 256 Hz. Randall et al’s results also showed greater blood flow with lower frequencies of 7 and 14 Hz than with the higher frequencies that produced tetanic contractions. Generally, the faster the frequency of electrical stimulation the lower the percentage of increase in blood flow. Our stimulation procedure elicited fused tetanic contractions at a fixed frequency of 50 Hz, which also may account for the smaller percentage of increase in blood flow observed in our study.

Our PI findings indicate that muscle blood flow does not increase significantly when contraction force is increased from 10% to 30% of MVC. Although the 50-Hz frequency of the Electrostim 180-2 unit did bring about a mean 20.5% increase in blood flow at 10% of MVC and a mean 19.6% increase at 30% of MVC, the timing sequence (15 seconds “on” and 50 seconds “off”) may not have been optimal for improving the circulation of blood flow in contracting muscle. Further study is needed to determine the optimum frequency for augmentation of blood flow by indirect electrical stimulation of human muscle. This finding may have considerable clinical relevance as a basis for the therapeutic treatment of soft-tissue injuries if further research shows that a sustained increase in blood flow during and after electrical stimulation to the triceps surae musculature increases the supply of nutrients to the injured area and improves the removal of waste products.

Initially, the Experimental group had a significantly higher mean heart rate than the Control group. We found no significant difference in the mean heart rates of the two groups at the final measurement. The initial difference probably was due to the Experimental group subjects’ anxiety concerning their forthcoming experience with electrical stimulation. Even after brief exposure to electrical stimulation during the electrically induced torque determinations, the mean heart rate of the Experimental group was significantly increased. The 15-minute rest during the preparatory activity before the experiment apparently was not sufficient time to allay the anxieties of the Experimental group subjects; however, by the completion of the stimulation phase, the mean heart rate of the Experimental group had decreased to within statistical equivalence of the Control group subjects. Thus, the Experimental group’s experience with electrical stimulation during the experimental period was sufficient to reduce anxieties and, consequently, their mean heart rate. The Control group subjects did not receive electrical stimulation and their heart rates remained relatively constant throughout the simulated experimental conditions.

The increased prestimulation mean diastolic blood pressure of the Experimental group when compared with that of the Control group also might suggest prestimulation anxiety. No significant difference between the mean diastolic blood pressures of the two groups was found during the final measurement. This finding may further support the existence of prestimulation anxiety among subjects of the Experimental group. Another explanation, however, must be considered; electrical stimulation to the calf musculature of one leg may cause systemic blood flow changes elsewhere in the body. Further research is needed to ascertain whether local muscle exercise that is induced artificially may alter circulatory conditions elsewhere in the body (eg, the upper extremities).

The skin temperatures of both the Experimental group and the Control group subjects were higher at the beginning of the experiment than at its termination. The exposure of the uncovered skin of the treated lower extremity to an ambient temperature of about 22°C (72°F) was the probable cause of the decrease in skin temperature.

CONCLUSIONS

The results of this study allow us to make the following conclusions about electrical stimulation (current characteristics of 2,500-Hz sine-wave frequency modulated at 50 bursts per second) applied to the posterior calf musculature with an intensity sufficient to produce isometric contraction of the plantar flexor muscles equivalent to 10% or 30% of MVC: 1) The blood flow of the popliteal artery increases, 2) the blood flow increases during the first minute of electrical stimulation and maintains a relatively steady-state level through the subsequent stimulation (9 minutes) and poststimulation (5 minutes) phases, and 3) blood flow does not increase further when the intensity of electrical stimulation is increased above the level required to produce 10% of isometric MVC to a level required to produce 30% of MVC.
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