Venous Hemodynamics of the Lower Extremities in Response to Electrical Stimulation

Pouran D. Faghri, MD, John J. Votto, DO, Christopher F. Hovorka, BS


Objective: To evaluate the calf muscle pump function using an air plethysmograph (APG) applied to the lower leg of subjects during three different tiptoe exercises.

Design: A controlled trial design was selected to compare the hemodynamic effects of three exercise conditions on a group of able-bodied, healthy patients.

Setting: Testing was performed in an outpatient clinic at a rehabilitation hospital.

Subjects: Patient groups were selected from a convenience sample of 10 healthy volunteers with normal venous capacitance and no reflex, determined through impedance plethysmography before the study.

Interventions: Three exercise conditions undertaken by each subject consisted of loaded and unloaded lower leg muscle contractions produced by (1) voluntary contraction (VOL), (2) electrical stimulation of the gastrocnemius-soleus and tibialis anterior muscles (ES), and (3) combined ES and VOL (ES/VOL).

Main Outcome Measure: Hemodynamic measurements of venous filling index upon standing from the supine (VFI), ejection fraction (EF), ejection volume (EV), residual volume (RV), and residual volume fraction (RVF) were recorded after each protocol. These results were used to compare the lower leg hemodynamic effects of the treatments.

Results: Combined ES/VOL single tiptoe exercise produced the highest EV (97.8mL), followed by VOL (80.6mL) and ES (51.7mL) (p < .0008). The EF was also highest for combined ES/VOL (73.1%), followed by VOL (64.5%) and ES (37.8%) (p < .0001). Ten tiptoe ES exercises produced the highest RV (96.2mL), followed by ES/VOL (44.7mL) and VOL (28.2mL) (p < .0001). RVF was also highest in the ES group (71%), followed by ES/VOL (33.4%) and VOL (22.8%) (p < .0001).

Conclusion: Periodic single ES-induced calf muscle contractions produced significant muscle pump function and could be used to improve venous blood flow and reduce stasis in the lower leg. Continuous ES-induced contractions, on the other hand, could improve lower leg peripheral perfusion while eliciting the physiologic venous muscle pump. Higher RV and RVF after 10 ES-induced contractions in this sample of healthy subjects with normal VFI may be caused by an increase in arterial blood perfusion after repeated ES-induced contractions.

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DURING UPRIGHT STANDING, sitting, or periods of prolonged dependency, an increase in hydrostatic pressure (the gravitational force exerted by the column of blood between the heart and the foot) within the blood vessels of the lower extremities leads to increased transcapillary filtration into the intersitial space. Concomitantly, the reabsorption of interstitial fluid is reduced. During this time, there is a slow volume change in the legs because of the resulting increase in extravascular fluid volume. Continuation of this situation may eventually cause venous insufficiency, subsequent deep-venous thrombosis, and pulmonary embolism.

The following mechanisms have been associated with intravascular thrombus formation: (1) reduction of intravascular blood flow (stasis), (2) primary lesions of endothelial cells, and (3) changes of blood coagulability. Among the risk factors that have been associated with the development of this condition, physical inactivity, occupation, work posture, and long-distance flight (economy class syndrome) have been reported as possible causes. Immobility, shifts of body fluid, orthostatic stress, and compression of the popliteal vein at the edge of the seat are some conditions that may lead to the above pathologic conditions. Stasis, which might contribute to the swelling of the dependent part of the body, has also been described as causing endothelial lesions.

It is generally assumed that the deep-venous system carries 90% of the blood from the lower limb, and transition from rest to normal rhythmic exercise, such as walking, is accompanied by dramatic changes in pressure and flow in the veins of the lower limb. When a subject moves from a supine to a standing position, the foot venous pressure rises from 15 to around 115mmHg because of the hydrostatic pressure. The action of the calf muscle pump has an important effect in reducing venous pressure. The normal functioning of the calf muscle pump (also called venous pump of the calf) is defined as the ability to keep venous outflow from the lower leg equal to arterial inflow during exercise, without undue dilution of the veins of the lower leg. The muscular pumping mechanism has important functional connotations: it drastically lowers the venous and capillary pressures and reduces the blood volume contained within the veins of the leg. The veins also act as a reservoir that releases stored blood during muscular exercise, momentarily accelerating the return of venous blood from the leg at the onset of toe raise exercise. The musculovenous pump also prevents the development of edema in the lower extremities by promoting lymph flow in an upright posture.

Active voluntary calf muscle contraction is the necessary first step in activation of venous pump function to prevent venous blood flow stasis during inactivity and prolonged standing. Christopoulos and coworkers measured the effects of voluntary calf muscle contractions on the venous blood flow by means of toe raise exercises. Using an air plethysmograph
(APG), a significant edema-preventing effect was observed when the calf venous ejection volume exceeded 60%. In situations in which voluntary calf muscle pump function is not attainable (ie, paralysis, sedentary lifestyle, occupation, or posture), other means of muscle pump activation need to be investigated. Electrical stimulation (ES)—induced muscle contractions of the lower limb muscles could cause activation of calf muscle pump function and has been shown to increase blood flow in healthy subjects, in patients undergoing surgery, and in individuals with spinal cord injury. The purpose of this study was to evaluate the effects of ES-induced calf muscle pump function and combinations of ES-induced and voluntary calf muscle pump function on venous blood flow in healthy individuals and compare their effects with voluntary contractions alone using the APG.

**MATERIALS AND METHODS**

**Subjects**

Twenty legs of 10 healthy volunteers (six women, four men) with normal venous capacitance and no reflux were tested in this study. None of the subjects had a history of disorders of the heart or circulation. All subjects gave written informed consent to participate in the study in accordance with our university's and our hospital's human subjects institutional review boards. Physical characteristics of the subjects are depicted in table 1.

**Instruments**

**Air plethysmograph.** Volume changes of the legs of each subject were measured by a noninvasive APG, model CIC-1000CP, according to the technique described by Christopoulos and colleagues and the protocol outlined by Katz and associates and Christopoulos and colleagues. A polyurethane cuff was placed around the calf of each subject's leg below the ankle. The cuff was connected to a pressure transducer in which a signal was transmitted to an amplifier that processed the information as an analog display onto a laptop personal computer. The cuff was inflated to 60 mmHg to ensure contact with the calf and to stabilize the cuff. Before each measurement, the cuff and computer were calibrated by injection of 100 mL of air into the inflated cuff. The resulting pressure increase in the cuff was adjusted and recorded by the computer. Throughout the measurement procedure, a continuous, real-time graphical display of calf volume was available. All the measurements were recorded at room temperature (22°C to 24°C).

Venous volume (VV), ejection volume (EV), ejection fraction (EF), residual volume (RV), residual volume fraction (RVF), and venous filling index (VFI) were measured for each subject’s leg with the APG during each of the three exercise protocols. Minimum venous volume (MinVV) was determined when each subject was lying supine with the test leg elevated to a 45° angle. This allowed gravity to empty the leg veins. The maximum venous volume (MaVV) represented the greatest volume in the leg veins and was achieved when each subject stood for 5 minutes with the knee slightly flexed to unload the test leg and open the lower leg vasculature. The gastrocnemius-soleus and tibialis anterior muscles remained relaxed in this position, allowing gravity to facilitate accumulation of VV in the lower leg veins. The EV is the volume of blood that is ejected by a single tiptoe maneuver in the standing position during the exercise protocol (fig 1). The EF represents the EV of blood divided by the MaVV multiplied by 100% (EF = EV/MaVV × 100%). The RV represents the volume of blood in the calf during 10 tiptoe movements. RVF is the RV divided by MaVV multiplied by 100% (RVF = RV/MaVV × 100%). The VFI represents the filling rate of the leg veins in milliliters per second. Normal VFI is less than 2 mL/sec. The higher the VFI, the greater the reflux and subsequent risk for venous stasis.

**Electrical stimulator.** An Empi Respop Select Dual Channel Neuromuscular Electrical Stimulator provided surface electrical stimulation to each leg of all subjects. Channels 1 and 2 were used for gastrocnemius-soleus and tibialis anterior stimulations, respectively. Self-adhesive reusable carbonized skin electrodes (5000 Series) were applied to the lower extremities of each subject by the same tester according to the procedures described by Benton and coworkers.

Stimulation parameters were set at 35 pulses per second (pps), balanced continuous symmetrical biphasic waveform, and stimulation intensities were administered according to subject tolerance. Each subject was habituated to electrical stimulation for two to three contractions and the maximum tolerable stimulation intensity was recorded.

**Procedure**

All subjects participated in each of three exercise protocols after a minimum 3-hour rest period between tests (no forceful tiptoe exercise).

Subjects began each protocol relaxed in the supine position (fig 1). The hip of the examined leg was externally rotated and the knee was slightly flexed. The patient's heel rested on a sponge to avoid artifact caused by the cuff contacting the examination table. The subject rested in this position for at least 5 minutes. The resting period stabilizes the patient and leg cuff with the room temperature and allows for arterial inflow. The calibration procedure was then performed. After calibration, the leg was elevated to 45° to empty the leg veins and achieve a MinVV within the lower extremity. Upon plateau of the VV (within 20 sec), which is an indication of zero functional VV (functional VV is the volume change [mL] from the supine position to the standing position), the patient was guided off the examination table and instructed to bear full weight on the opposite leg. A standing balance frame was used for support. The tested leg was relaxed with the knee slightly flexed approximately 20° and the forefoot lightly touching the floor. Subjects remained in the standing position until the leg veins filled to MaVV. Once MaVV was achieved, the patient stood on both feet and performed one tiptoe maneuver. On completion, the subject returned to the original standing position with the body weight on the opposite leg. A decrease in volume (mL) was observed during the tiptoe exercise because there was less blood in the calf due to the muscular contraction. Refill of the calf volume was recorded after the tiptoe maneuver.

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**Table 1: Physical Characteristics of the Subjects**

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<tr>
<th>Subject</th>
<th>Age (yrs)</th>
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<th>Weight (kg)</th>
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Fig 1. Testing protocol. Subjects begin lying supine with the tested leg elevated to achieve minimum venous volume (a). Subjects stand bearing full body weight on the leg opposite the tested leg until maximum venous volume is achieved (b). One or two single tiptoe contractions are performed, followed by a rest period until maximum venous volume is achieved (c, d). Ten consecutive tiptoe contractions are performed (e), followed by lying supine to reestablish minimum venous volume (f). 90% VV, 90% of venous volume; VFT 90, 90% of venous filling time; VFI, venous filling index; EV, ejection volume; VV, venous volume; EF, ejection fraction; RV, residual volume; RVF, residual venous fraction.

Exercise Protocols

Voluntary exercise protocol (VOL). Each subject performed voluntary contractions of the calf muscles by performing tiptoe exercises. Subjects were instructed to begin exercise during this protocol by standing on their heels and then contracting the calf muscles to rise as tall as possible onto both forefeet for 10sec. Subjects transferred their weight to their heels and completed the exercise by flexing the knees of the tested extremities, applying minimal weight to the test legs. Each subject then performed 10 sequential tiptoe exercises.

ES exercise protocol. The gastrocnemius-soleus and tibialis anterior muscles of the test leg of each subject was electrically stimulated to contract, substituting ES for voluntary muscle contraction. Muscle contractions were induced to a non-weight-bearing lower leg that was not touching the floor. The single toe rise exercise was performed by costimulation of the gastrocnemius-soleus and tibialis anterior muscle bellies of the testing leg (with hip externally rotated and knee in flexed position) to maximum tolerable ES intensity to produce plantar flexion (visible foot movement) for 10sec.

The 10 sequential toe rise contractions (2sec on, 2sec off) were performed by ES to produce plantar flexion with maximum tolerable levels of stimulation intensity.

Combined exercise protocol (ES/VOL). Each subject performed voluntary toe raise exercises in addition to electrically stimulated cocontractions of the calf and tibialis anterior muscles by surface ES. Subjects were instructed to follow the voluntary exercise protocol. Subjects initiated voluntary tiptoe exercises as soon as they felt the ES at the threshold level. The intensity of ES was then increased to the maximum tolerable level, and the voluntary contraction was increased as well. Subjects were instructed to maintain the toe raise position for approximately 2sec—the duration of stimulation of the muscles at maximum intensity. When the stimulation ramped down and the subject no longer felt the electrical muscle stimulation, each subject was instructed to lower from the toe raise and return to
the unweighted leg position. For the 10 toe raise exercises, the subject returned to a position with both feet flat on the floor and waited for stimulation cycle before performing each toe raise. After the tenth contraction was completed, the subject returned to the unweighted leg position.

Statistical Analysis

The hemodynamic responses that were recorded for each treatment were the EF (%), EV (mL), RV (mL), RVF (%), VV (mL), and VFI (mL/sec). The data were analyzed as a completely randomized design using SAS, 1994. One-way analysis of variance (ANOVA) was used to compare the treatment means. The treatment means were then compared using least significant difference (LSD). The level of significance was set at $p \leq .05$. The treatment VOL was used as a control for comparison, and the percentage change in response to the other two treatments was calculated. The table of statistical analysis is illustrated in table 2.

RESULTS

Ejection fraction. There was no difference between the EFs produced after VOL and after ES/VOL. The EF produced after ES-induced contraction was significantly lower than that produced after VOL and ES/VOL; however, ES-induced contraction produced up to 60% of EV produced by voluntary contraction (fig 2 illustrates the EF during the three protocols).

Ejection volume. The EV was significantly higher in the ES/VOL group than in the other two treatment groups. ES-induced exercise produced up to 60% of EV produced by voluntary contraction (fig 3).

Residual volume and residual volume fraction. The RV and RVF values of the subjects during the three treatment protocols were significantly different from each other. ES-induced exercise produced the highest RV and RVF, followed by ES/VOL (figs 4 and 5).

Venous filling index. All subjects had normal VFI (<2mL/sec). The mean ± SD for VFI recorded during the three treatment protocols were .85 ± .40mL/sec (VOL), .77 ± .36mL/sec (ES), and .71 ± .27mL/sec (ES/VOL) (fig 4).

Venous volume. The EV was significantly higher in the ES/VOL group than in the other two treatment groups. ES-induced exercise produced up to 60% of EV produced by voluntary contraction (fig 3).

Residual volume and residual volume fraction. The RV and RVF recorded for each of the three treatment protocols were significantly different from each other. ES-induced exercise produced the highest RV and RVF, followed by ES/VOL and VOL (figs 4 and 5).

DISCUSSION

The hemodynamics of the arterial circulation are relatively simple to understand, dominated by the pumping function of the heart. In contrast, the hemodynamics of venous return of the lower limb, against gravity, are more complicated and multifactorial, dominated by function of the calf muscle contraction. In limbs with primary varicose veins, the calf muscle pump is like a heart with a normal stroke volume but increased preload because of reflux. In limbs with deep-venous disease, it is like a heart with reduced stroke volume because of destruction of the veins. This causes a reduction of the volume of the deep veins of the calf, increased afterload because of high outflow resistance caused by venous obstruction, and increased preload as a result of reflux in the deep or superficial veins.

The impaired function of the calf muscle pump is responsible for venous hypertension, which leads to excessive accumulation of fluid and fibrinogen in the subcutaneous tissue, resulting in swelling, lipodermatosclerosis, and finally ulceration.

In their study, Bellinger and colleagues showed that healthy, able-bodied working humans had an average increase in lower leg volume of 50mL between morning and afternoon. In the same study, subjects with chronic venous insufficiency showed a threefold increase in swelling rate. These results support the assumption that muscle inactivity is the principal factor in swelling of the lower leg. A person sitting or standing for prolonged periods normally experiences less muscle activity or sometimes complete muscle relaxation, which may affect venous and lymphatic backflow from the lower limbs and lead to more venous stasis and swelling. Therefore, normal leg activity is an important factor in prevention of swelling. Noddeland and Winkelman found that the swelling of the leg with normal leg activity in sitting humans is .33mL/100mL/h, increasing to .71mL/100mL/h in the same subjects when their legs are completely immobile. However, normal muscle activity/

### Table 2: Statistical Analysis of Hemodynamic Responses to Three Treatment Protocols

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<th>Measured Responses</th>
<th>Treatment Protocol</th>
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<tr>
<td></td>
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<td>37.8</td>
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<td>51.7</td>
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<td>97.8</td>
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<td>Residual volume (mL)</td>
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exercise is not always possible for healthy persons. In healthy individuals, situations associated with muscle inactivity (ie, sedentary occupations or prolonged airline travel) may induce the development of lower limb blood flow stasis and venous thrombosis. ES has been shown to improve the blood flow circulation in paralyzed patients and those under anesthesia. 

In this study, we investigated the effects of ES alone and in combination with voluntary contraction on the lower leg hemodynamics of healthy subjects. As displayed by continuous APG measurements, the greatest amount of flow in the leg veins occurs when combined electrically stimulated cocontraction of the gastrocnemius-soleus and tibialis anterior muscles in conjunction with voluntary toe raise exercises are performed. The average volume of blood moved by the ES was 51.7 ± 25.5mL. ES in combination with voluntary contraction produced the highest EV (97.8 ± 37.5mL). The amount of blood moved by ES alone was equal to the amount of blood accumulated in the lower leg during normal standing. ES-induced contraction alone produced EF of up to 38%. Studies have shown that EF is in the range of 60% to 90% in normal limbs, 30% to 60% in limbs with primary varicose veins, and 10% to 50% in limbs with deep-venous disease. The 38% EF that was produced by ES and was tolerated in this group of healthy subjects could have added a significant effect in returning a large amount of blood to the heart. This addition could particularly affect persons with deep-venous disease. The effects of EF on venous ulceration have also been investigated. The incidence of ulceration in limbs without venous obstruction is 0% for...
venous ulceration may exist because not all limbs with severe minimal reflux (VFI < 5mL/sec), 46% for moderate reflux from 63% to 30% in limbs with moderate reflux and from 70% to 41% in limbs with severe reflux. Poor EF (EF < 40%) results in a significantly high incidence of ulceration (32%). The addition of ES to voluntary contraction in our study group resulted in a significantly high EP of 73%. The EF produced by ES alone in eight of the subjects in this study was more than 40%. These results represent a nearly protective effect in preventing venous stasis complications through surface electrical muscle stimulation alone. It should be cautioned that only healthy subjects free of venous pathology were tested for this study. Further research is needed to document these effects in a symptomatic group of individuals with documented venous pathology.

The EP of the calf muscle pump depends on two parameters: EV and VV. The decreased EF in limbs with superficial incompetence is mainly a result of the increased VV caused by varicose veins. In limbs with deep-venous insufficiency, however, the decreased EF results from low EV and increased VV. In postthrombotic limbs, the EV is reduced because of destruction of the venous sinuses and intramuscular veins of the gastrocnemius-soleus. Studies have shown that ES-induced contraction causes a significant effect in emptying these sinuses compared with voluntary contraction. In this study, the addition of ES to voluntary contraction caused a 21% increase in EV. Higher EV could be the result of an additional effect of ES in emptying the venous sinuses in this group. This could benefit patients with deep-venous insufficiency.

After the 10 tiptoe exercises, a steady state (RV) is achieved. RV is the amount of blood expelled from the veins of the leg as a result of each calf muscle contraction. This is equal to the amount of blood that enters the muscles during each period of relaxation from the capillaries and from reflux. The RV is directly related to VFI. Studies have shown that this reduction is between 45% and 90% in normal limbs, between 35% and 50% in limbs with primary varicose veins, and between 0% and 30% in limbs with deep-venous disease. In contrast to EF and EV measurements, which do not depend on reflux, the RVF and RV depend on reflex and EF and express the combined effects of both of these parameters during rhythmic exercise. The RVF is the measure of the volume of blood during exercise. It is responsible for the ambulatory venous pressure, which when extrapolated depends on both reflux and EF of the calf muscle pump. The VFI is considered normal when it is less than 2mL/sec. In our study group, the average VFI was 8.5mL/sec for VOL, 7.1mL/sec for ES, and 4.4mL/sec for ES/VOL. The RV (28.2mL) and RVF (22.8%) were within normal limits in the VOL group. These values were highest for the ES group (96.2mL RV, 70.4% RVF), followed by the ES/VOL group (44.7mL RV, 33.4% RVF). Because the same group of subjects underwent three different protocols and displayed normal VFI, the higher RV and RVF during ES and ES/VOL could be caused by hyperemia induced by ES. Fokkow and Halicka reported a 46% increase in blood flow upon stimulation of the gastrocnemius muscle and a 77% increase upon stimulation of the soleus muscle compared with voluntary contraction.

Blood flow to skeletal muscle during and after volitional exercise is controlled by local metabolism. Local metabolite changes produce alterations in local pH and composition of the interstitial fluid, both of which promote vasodilatation. Continuous ES of the calf muscle at the intensity sufficient to produce the equivalent of 15% maximum voluntary contraction is sufficient to cause redistribution of the blood from nonexercising areas of the body to the contracting muscles. An increase in RV and RVF in our healthy subjects with normal VFI could be secondary to poststimulation hyperemia. This phenomenon may not have occurred during single static ES-induced contraction. Electrical induction of near-maximal contractions may occlude blood supply and induce metabolic deficit to the muscle while accelerating venous blood flow. However, the rest period after each electrically induced contraction may be sufficient for rephosphorylation of adenosine diphosphate and creatinin phosphokinase in the contracting muscles and inhibit vasodilation after static contraction.

CONCLUSIONS

Based on the results of this study, we made the following two conclusions. First, single and static ES-induced contractions of the lower limb may be used as an adjunct to improve the venous blood flow. This treatment may be more effective (increasing venous return) in patients with compromised venous circulation. The level of stimulation used in this study was comfortable to and tolerated by healthy subjects. The application of electrically stimulated muscle contraction may provide an edema-preventing effect in persons at risk for venous stasis and subsequent edema. The system may be used to act as a physiologic muscle pump during imposed inactivity. Further study is needed to confirm these findings. Second, continuous ES-induced contractions might be used to improve the blood flow to the contracting muscles. This may significantly improve the electrically stimulated muscle’s circulation. The ES-induced contraction may be more effective in patients who may be otherwise unable to voluntarily exercise their muscles (ie, those with paralysis). Continuous ES-induced contractions, on the other hand, could improve lower leg peripheral perfusion while eliciting the physiologic venous muscle pump.

References

**Suppliers**

a. ACI Medical Inc., 1857 Diamond Street, San Marcos, CA 92069.