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Passive Leg Movements and Passive Cycling Do Not Alter Arterial Leg Blood Flow in Subjects With Spinal Cord Injury

Background and Purpose. Subjects with a spinal cord injury (SCI) are at increased risk for cardiovascular disease–related secondary complications, such as pressure ulcers and attenuated wound healing. It has been suggested that passive exercise enhances blood flow via mechanical pump effects or reflex activation. The purpose of this study was to assess the effects of passive leg movements and passive cycling on the arterial circulation in subjects with SCI. **Subjects.** Eight men with motor complete SCI and 8 male control subjects participated. **Methods.** Echo Doppler measurements were obtained to measure leg blood flow at rest, during and after 10 minutes of standardized passive leg movements, and during and after 20 minutes of passive leg cycling. Blood pressure was measured continuously, and total vascular resistance and leg vascular resistance were calculated. **Results.** In both groups, no changes in leg blood flow, vascular resistance, or blood pressure were observed during or after the 2 interventions. **Discussion and Conclusion.** The results of the study demonstrate that passive leg movements and passive cycling do not alter the arterial peripheral circulation in subjects with SCI or control subjects. Although the results do not support the use of passive movements or exercise for the prevention of cardiovascular disease–related secondary complications, physical therapists should not be dissuaded from using these techniques to address musculoskeletal concerns. [Ter Woerds W, De Groot PCE, van Kuppevelt DHJM, Hopman MTE. Passive leg movements and passive cycling do not alter arterial leg blood flow in subjects with spinal cord injury. *Phys Ther.* 2006;86:636–645.]

Key Words: *Blood circulation, Spinal cord injuries.*

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Aspinal cord injury (SCI) results in the paralysis of major muscle groups, autonomic nervous system dysfunction, and loss of sensation. In people with chronic SCI, extensive adaptations of the central and peripheral circulatory systems occur. These vascular adaptations are characterized by decreased venous capacity and increased venous flow resistance,^{1,2} loss of left ventricular mass,³ diminished capillary formation,⁴ increased vascular resistance,⁵ and reductions in vessel diameter,⁶⁻⁹ blood flow,^{6,7} and arterial compliance⁸ of the femoral artery. In addition, almost doubled shear stress levels have been found in the leg arteries of subjects with SCI,^{6,8,10} a finding that may be indicative of endothelial dysfunction.¹¹ Endothelial dysfunction, decreased arterial compliance, and increased venous flow resistance are vascular characteristics in subjects with SCI that are directly linked with atherosclerosis and deep vein thrombosis (DVT).¹¹⁻¹³

Excessive pressure as well as other factors, such as an inadequate nutrition and impaired circulation,^{14,15} are risk factors for pressure sores. The elevated vascular resistance in subjects with SCI⁵ will reduce peripheral tissue perfusion and capillary blood flow further and subsequently contribute to the development of pressure ulcers¹⁴⁻¹⁶ and to poor wound healing.¹⁷

These secondary complications are a frequent cause of morbidity and mortality and lead to a decrease in the quality of life in people with SCI.¹⁸⁻²⁰ Moreover, they account for a major part of the treatment costs.²¹ Therefore, prevention of secondary complications in people with SCI has become an important topic. Previous studies of people with SCI showed that the detrimental adaptations in the circulatory system are partially reversible by functional electrical stimulation training.^{5,22} Moreover, several studies indicated that passive exercise leads to circulatory²³⁻²⁷ and muscular^{28,29} adaptations in people who are healthy as well as in people with SCI. Possible mechanisms by which passive exercise may

enhance leg blood flow (LBF) could be related to activation of the passive muscle pump or ankle pump, of mechanoreflexes, or of the autonomic nervous system.³⁰⁻³⁴ Passive leg movements carried out by a physical therapist and passive cycling are 2 forms of passive exercise frequently used in the rehabilitation of people with SCI. Whether these interventions positively affect the arterial peripheral circulation is unknown. Therefore, the purpose of this study was to assess peripheral circulatory responses during and after passive leg movements and passive cycling in subjects with SCI and control subjects who were healthy by using protocols like those used in the clinical setting in rehabilitation centers. We hypothesized that these passive interventions would enhance LBF in both groups.

Method

Subjects

Eight men with SCI and 8 male subjects who were healthy (control subjects) were included in the study. A control group was included so that we could generalize the results as well as obtain insight into responsible mechanisms. All of the subjects with SCI had motor complete lesions (American Spinal Injury Association class A or B³⁵) with injury levels between T2 and L1, and time since injury varied from 1 to 17.5 years ($\bar{X}=8.3$, $SD=6.1$). All subjects were between 20 and 49 years of age and had no history of diabetes, cardiac diseases, recent DVT, or recent pressure ulcers. Two subjects with SCI had a history of DVT 9 years previously; however, the DVT was in the left leg, and all measurements in the present study were obtained in the right leg. Five subjects with SCI had had pressure ulcers in the past (2, 2, 5, 9, and 9 years previously, respectively). Subjects with SCI exercised 5.7 ± 3.9 hours ($\bar{X}\pm SD$) per week, mainly endurance-type exercise, whereas control subjects exercised 4.7 ± 2.3 hours per week. Descriptive characteristics of the study subjects are summarized in Table 1.¹³

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Dr De Groot, Dr van Kuppevelt, and Dr Hopman provided concept/idea/research design. Mr Ter Woerds and Dr De Groot provided writing and data collection. Mr Ter Woerds provided data analysis. Dr Hopman provided project management and fund procurement. Dr van Kuppevelt provided subjects and facilities/equipment. The authors acknowledge the participation of all subjects in the study.

The Medical Ethical Committee of the Radboud University Nijmegen Medical Centre, the Netherlands, approved this study.

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Table 1.
Subject Characteristics

Subject No.	Age (y)	Height (cm)	Weight (kg)	Body Surface Area (m ²)	Exercise (h/wk)	Lesion Level	ASIA Class ^a	Time Since Injury (y)	Spasms ^b
Spinal cord injury									
1●	33	180	70	1.87	10	T6	A	10	+
2▲	30	178	80	1.98	1.5	T11	B	2.5	-
3△	21	178	73	1.91	10	T6	A	1	-
4△	38	186	58	1.78	4.5	T12/L1	B	16.5	-
5○	49	187	94	2.19	5	T6	A	17.5	+
6■	34	179	69	1.87	1.5	T4-T5	A	5.5	++
7○	43	178	70	1.87	2.5	T8	A	7.5	+++
8□	34	173	65	1.76	10.5	T2-T4	A	5.5	+++
\bar{X}	35 ^c	179	72 ^c	1.91 ^c	5.7			8.3	
SD	8.4	4.6	11	0.14	3.9			6.1	
Control									
1	26	183	83	2.05	4				
2	24	198	95	2.30	7				
3	22	183	74	1.95	3.5				
4	23	196	84	2.17	4				
5	21	197	85	2.19	8				
6	29	185	85	2.08	6				
7	30	180	72	1.91	0.5				
8	34	180	79	1.98	4.5				
\bar{X}	26	187	82	2.08	4.7				
SD	4.5	8	7.2	0.14	2.3				

^aThe symbols of the subjects with spinal cord injury correspond to the individual symbols in Figures 1 and 2. American Spinal Injury Association (ASIA)³⁵ score is used to classify the completeness of the lesion: A=sensory and motor complete; B=sensory incomplete but motor complete.

^b- =no spasms; +=rarely; ++=regular (daily); +++=severe.

^cSignificantly different from value for control subjects at $P<.05$.

Four subjects with SCI used medication: 2 subjects used tolterodine to control bladder spasms, and 2 subjects used methenamine and nitrofurantoin to control urinary tract infections. In order to cope with muscle spasms, 1 subject used baclofen. One subject in the SCI group smoked 5 cigarettes per week. In the control group, 1 subject used medication for allergy, and none of the subjects smoked. Written informed consent was obtained from all subjects before the experiment.

Protocol

Subjects were asked to refrain from use of caffeine, alcohol, and nicotine 12 hours before testing and not to perform exhaustive exercise 24 hours before testing. The tests were performed between 8:30 AM and 1:00 PM. All measurements were obtained in a room in which the temperature was kept constant at 23.5° to 25°C. The same investigator performed the test procedures, and passive leg movements were applied by 1 physical therapist in exactly the same sequence and manner for all subjects.

Successively, each subject underwent 2 interventions, which consisted of passive leg movements and passive cycling. The control subjects were instructed to relax the muscles of the lower extremities during both experiments. To ensure that the control subjects did this,

myofeedback (Myomed 432*) was used during passive cycling. The myofeedback device gave audiovisual feedback from the activity levels of the rectus femoris muscle and the vastus lateralis muscle of the quadriceps femoris musculature. During passive leg movements, the physical therapist detected muscle contractions manually and gave verbal feedback to the control subjects. The entire procedure was practiced before testing began.

Passive leg movement protocol. Subjects were studied in the supine position. The protocol consisted of 3 parts: 20 minutes of rest, 10 minutes of passive leg movements, and 10 minutes of recovery. During the intervention, the right leg was passively moved according to the protocol shown in Table 2. Passive movement therapy was performed in a standardized sequence with similar durations for the different movements and with the same intensities of stretching by the experienced physical therapist. For all movement patterns, the full range of motion was applied. Two subjects in the SCI group had a limited range of motion; that is, 1 subject had a maximum flexion angle in the knee joint of 115 degrees (normal: $\pm 160^\circ$), and 1 subject had a range of motion in the ankle joint of 40 degrees (normal: $\pm 70^\circ$).

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Table 2.

Passive Movement Protocol, With Beginning and Ending Times for Each Movement Intervention

Movement	Time (min)
Hip flexion/extension	0–1.5
Stretching hamstring muscles	1.5–2.5
Ankle plantar flexion/dorsiflexion	2.5–3
Knee flexion/extension	3–5
Ankle plantar flexion/dorsiflexion	5–5.5
Hip abduction/adduction	5.5–7.5
Ankle plantar flexion/dorsiflexion	7.5–8
Hip flexion/extension	8–10

Blood flow (as determined by echo Doppler evaluation) was measured after 20 minutes of rest, every 2.5 minutes during passive movements, and at 0, 1, 2, 5, and 10 minutes during recovery. During the measurement period (2.5, 5, and 7.5 minutes), plantar flexion and dorsiflexion of the ankle were performed. The systolic and diastolic diameters of the common femoral artery were measured at rest (ie, immediately before initiation of passive leg movements) and at 1 and 10 minutes during recovery (ie, after passive leg movements). Blood pressure and heart rate were recorded continuously throughout the entire protocol (Portapres device[†]).

Passive cycling protocol. After the passive leg movements, the subjects moved actively from the supine position to the sitting position and transferred over a distance of 1.5 m to the bicycle. During passive cycling, the subjects were seated in a wheelchair or a standard chair in front of the cycle ergometer (Reck Motomed Viva I[‡]). After 20 minutes of rest, the subjects cycled passively for 20 minutes, followed by a 10-minute recovery period. Passive cycling was performed on an electrically driven bicycle ergometer at a speed of 35 repetitions per minute. The feet of the subjects were attached to the pedals with Velcro[§], and the legs were secured just below the knee. Subjects were allowed to rest against a back support. For measurements during passive cycling, the ergometer was stopped briefly (approximately 10 seconds) in the maximally extended leg position. The location at which the femoral measurements were obtained was marked to ensure rapid measurements. Blood flow in the right leg was measured at the end of the 20-minute rest period, after 1 minute of cycling, and subsequently every 2.5 minutes during cycling. During recovery from cycling, measurements were performed at

0, 1, 2, 5, and 10 minutes. The diastolic and systolic diameters of the common femoral artery were measured at rest (ie, immediately before initiation of passive cycling) and at 1 and 10 minutes during recovery (ie, after passive cycling). Blood pressure and heart rate were recorded continuously with the Portapres device.

Measurements and Data Analysis

Red blood cell velocities and the systolic and diastolic diameters of the right common femoral artery were measured by Doppler sonography (Megason^{||}) with a 5- to 7-MHz linear transducer. Measurements in the common femoral artery were obtained below the inguinal ligament, about 2 cm proximal to the bifurcation into the superficial and profundus branches, and the angle of inclination was consistently below 60 degrees. During every measurement of red blood cell velocities, 2 images were stored, for a total of 6 to 8 velocity profiles. For diameter measurements, 2 images each were frozen at the peak systolic phase and the end-diastolic phase. Off-line, 1 investigator who was unaware of group assignment (SCI or control) or measurement intervention (passive movement or passive cycling) analyzed all velocity profiles and arterial diameters.

The velocity profiles were traced manually, and an average of these velocity profiles was calculated for mean red blood cell velocity. For diameter, 3 measurements were obtained manually from each arterial diameter image, and average values were calculated. *Lumen diameter* was defined as the distance between the far wall boundary, that is, the lumen-intima interface, and the near wall boundary, corresponding to the media-adventitia interface. The reproducibility of the echo Doppler measurements in our research was reported previously,⁶ and the coefficients of variation for vessel diameter and blood flow measurements for the common femoral artery were 1.5% and 8%, respectively.

The mean diameter was calculated as one third of the systolic diameter plus two thirds of the diastolic diameter; the resulting value was used to calculate the cross-sectional area ($0.25 \times \pi \times \text{mean diameter squared}$). Mean LBF (in milliliters per minute) was calculated as mean red blood cell velocity (in meters per second) times cross-sectional area (in meters squared) times 6×10^7 . Leg vascular resistance (LVR) (in millimeters of mercury per milliliter per minute) was calculated as mean arterial pressure (MAP) divided by LBF. For these calculations, we assumed that central venous pressure was low and remained constant throughout the protocol.

Heart rate and blood pressure were measured with the Portapres device. A finger cuff was attached to the third

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[‡] Eureva, Saturnusstraat 95, 2516 AG, Den Haag, the Netherlands.

[§] Velcro USA Inc, 406 Brown Ave, Manchester, NH 03103.

^{||} Pie Medical Benelux BV, Philipsweg 1, 6227 AJ Maastricht, the Netherlands.

Table 3.
Individual Baseline Values for Subjects With Spinal Cord Injury and Control Subjects^a

Subject No.	Leg Blood Flow (mL/min)	V _{mean} (cm/s)	Diameter (cm)	Leg Vascular Resistance (mm Hg/mL/min)	Mean Arterial Pressure (mm Hg)	Total Peripheral Resistance (mm Hg/mL/s)	Cardiac Output (L/min)
Spinal cord injury							
1●	157	9.3	0.60	0.56	89	0.93	5.4
2▲	317	17.9	0.66	0.30	94	0.69	6.9
3△	353	13.4	0.74	0.25	89	1.17	5.4
4△	152	10.3	0.61	0.60	91	0.96	5.3
5○	105	2.8	0.89	0.92	96	0.52	11.4
6■	203	7.8	0.69	0.47	96	0.97	6.1
7○	96	5.7	0.75	0.96	91	0.72	6.8
8□	146	7.1	0.56	0.66	96	0.86	6.0
\bar{X}	191 ^b	9.3	0.69 ^c	0.59 ^c	93	0.85	6.6
SD	95	4.7	0.11	0.26	3	0.2	2.0
Control							
1	409	8.7	0.98	0.23	95.3	0.69	8.2
2	288	6	1.05	0.28	81.9	0.53	9.3
3	287	6	0.97	0.37	105.5	0.68	9.3
4	261	6	0.95	0.41	106.4	0.73	8.9
5	334	7.4	1	0.25	84.9	0.92	5.5
6	247	4	1.02	0.41	100.6	1.22	5
7	175	4.15	1	0.48	83.9	0.66	7.7
8	420	10	1.14	0.20	93	0.81	6.1
\bar{X}	303	6.5	1.02	0.33	93	0.78	7.5
SD	82	2.1	0.07	0.10	10	0.21	1.7

^a Values were measured in the supine position. V_{mean}=mean red blood cell velocity in the common femoral artery.

^b Significantly different from value for control subject at $P < .05$.

^c Significantly different from value for control subject at $P < .01$.

finger of the right hand. Data were collected beat to beat at a rate of 100 Hz. The MAP was computed as the true integrated mean of systolic and diastolic pressures. Previous studies^{36,37} showed that the Portapres device accurately reflects arterial blood pressure. Modelflow, a pulse-counting method, was used to calculate stroke volume³⁸ and, subsequently, cardiac output (CO)=stroke volume (SV) × heart rate, in liters per minute, and total peripheral resistance (TPR) (MAP/CO, in millimeters of mercury per milliliter per second).

Previous studies on SCI showed that with functional electrical stimulation, blood flow values increased more than 350%.³⁹ In people who are healthy, leg exercise may increase blood flow more than 10-fold.⁴⁰ In the current literature, inconsistent results have been reported regarding the effects of passive movements on blood flow, with only 1 study showing an increase²⁴ and other studies reporting no effect.⁴¹ We hypothesized that, if there is an effect, it would be relatively small, because passive exercise involves no active muscle contractions. A 20% to 40% increase would be clinically relevant, because values in the presence of SCI would approach values for resting flow in the control subjects. Therefore, on the basis of power analyses, we included a total of 8 subjects with SCI and 8 control subjects in the present study (a 20%–40% increase in LBF with a

standard deviation of 60 mL per minute during exercise can be considered clinically relevant; with $\alpha = .05$, power is .80, and the number of subjects needed is 8). A Student *t* test for independent groups was used to test for differences between groups with regard to demographic and baseline circulatory characteristics.

For the variables LBF, LVR, TPR, MAP, and CO, 2-factor repeated-measures analyses were applied with time (rest, intervention, and recovery values) as the within-subject factor and group (SCI and control) as the between-subject factor. The level of statistical significance for all tests was set at $P < .05$. Data are presented as mean ± standard deviation unless otherwise stated.

Results

Resting Properties

At supine rest, the arterial diameter and blood flow of the common femoral artery were decreased significantly in subjects with SCI compared with control subjects. The LVR in subjects with SCI was significantly higher than that in control subjects. No differences between the groups were seen in any other variable at rest. Individual values are shown in Table 3.

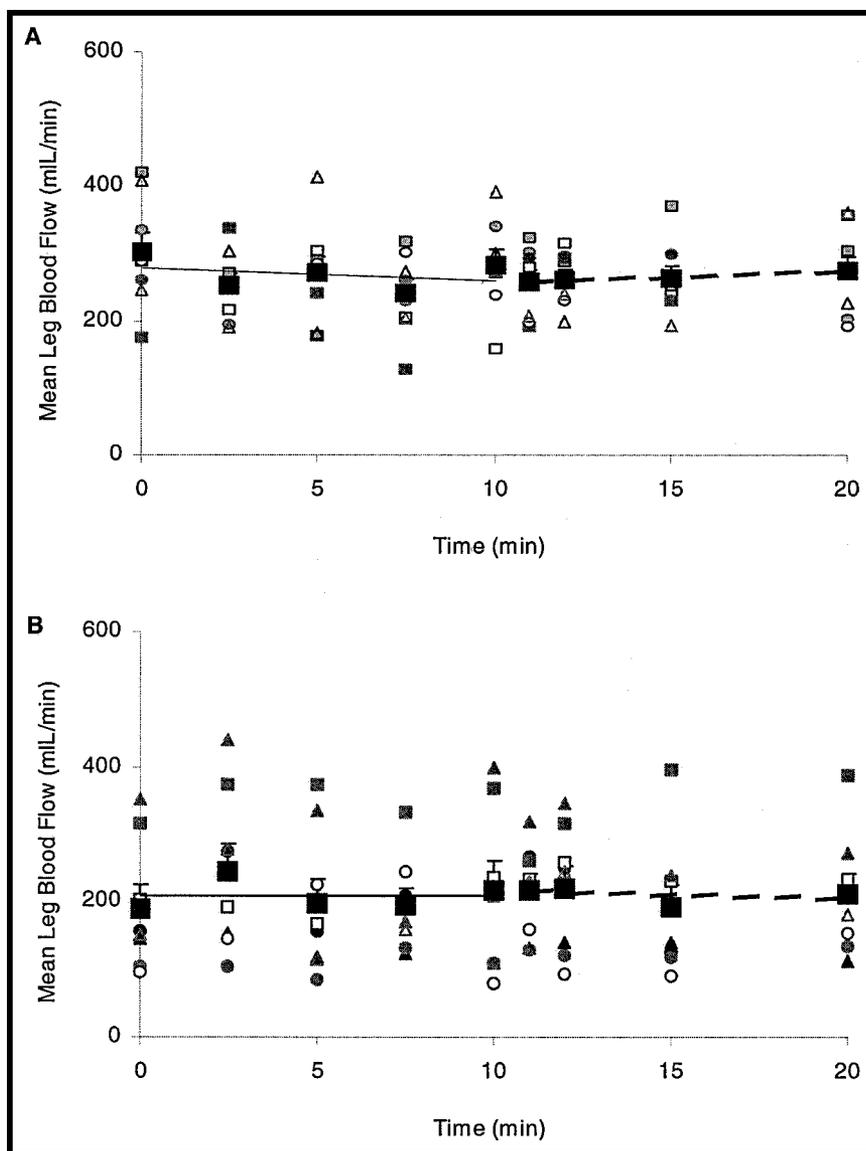


Figure 1. Individual results for mean leg blood flow before (0), during (0–10 minutes), and after (10–20 minutes) passive movements in control subjects (A) and subjects with SCI (B). Large black squares represent mean group values (\pm SEM). Regression lines are plotted for mean blood flow during treatment (solid line) and during recovery (dashed line). See Table 1 for explanation of symbols for subjects with SCI.

Passive Leg Movements

Repeated-measures analyses were applied to the data, and no significant main effect was found for blood flow ($P=.68$), LVR ($P=.71$), TPR ($P=.59$), MAP ($P=.46$), or CO ($P=.65$), indicating that the variables did not change over the time period examined. In addition, no significant effect of an interaction of group and time was found for blood flow ($P=.35$), LVR ($P=.22$), TPR ($P=.68$), or MAP ($P=.57$). The mean blood flow values during and after passive movements in the control subjects and the subjects with SCI are shown in Figures 1A and 1B, respectively.

Passive Leg Cycling

Repeated-measures analyses revealed no changes over time for blood flow ($P=.14$), LVR ($P=.34$), TPR ($P=.59$), MAP ($P=.37$), or CO ($P=.84$). No significant effect of an interaction of group and time was found for blood flow ($P=.68$), LVR ($P=.35$), TPR ($P=.35$), MAP ($P=.57$), or CO ($P=.83$). The mean blood flow values during and after passive leg cycling exercise in the control subjects and the subjects with SCI are shown in Figures 2A and 2B, respectively.

Discussion

Using echo Doppler sonography, we found that blood flow in the common femoral artery did not change during or after 2 different passive exercise interventions in the subjects with SCI or the control subjects. The results of the study demonstrate that passive exercise does not affect the peripheral circulation and therefore will not improve tissue perfusion, a low level of which is one of the risk factors for pressure ulcers¹⁶ and poor wound healing¹⁷ in people with SCI. However, it is important to realize that passive movements are useful for musculoskeletal concerns, such as mobility and prevention of contractures and spasms.

In order to compare the subjects with SCI with the control subjects, it was necessary for the control subjects to relax their muscles during the interventions. During passive cycling, myofeedback was used to achieve this goal. With audiovisual feedback, it appeared to be possible to correct the contractions immediately. During passive leg movements, the physical therapist was able to sense muscle contractions manually and to correct them by means of verbal feedback. Normally, when muscle activity is present, cortical brain activity causes autonomic nerve activity and a subsequent increase in heart rate.⁴² In the present study, heart rate did not increase; therefore, all subjects were assumed to be in a passive state.

Resting Properties

The significantly lower blood flow in the supine position and the smaller arterial diameter of the femoral artery in the subjects with SCI than in the control subjects were in accordance with the results of previous studies.^{6,7,43} In

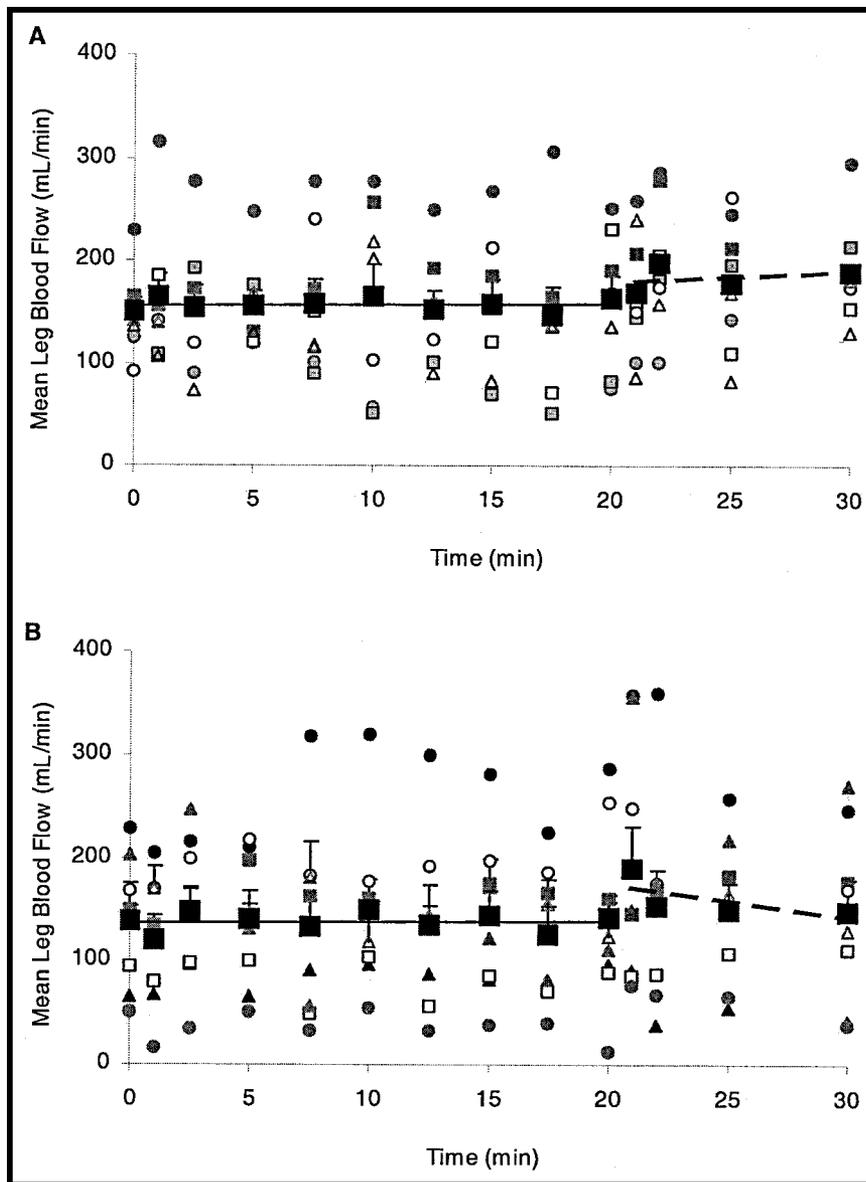


Figure 2. Individual results for mean leg blood flow before (0), during (0–20 minutes), and after (20–30 minutes) passive cycling in control subjects (A) and subjects with SCI (B). Large black squares represent mean group values (\pm SEM). Regression lines are plotted for blood flow during treatment (solid line) and during recovery (dashed line). See Table 1 for explanation of symbols for subjects with SCI.

the present study, the baseline characteristics body surface area (BSA) and age differed significantly between the groups. In a previous study, Sandgren et al⁴⁴ showed that femoral artery diameter is positively related to age and body size. Because of the older age of the subjects with SCI, a larger diameter would be expected in these subjects. In contrast, the larger BSA in the control subjects would imply a larger diameter in these subjects. However, the extensive diameter reduction (30%–40%) in the subjects with SCI must be considered a result of inactivity and paralysis and cannot be explained by these differences in BSA and age between the subjects with SCI

and the control subjects. The femoral artery diameters in both groups also were consistent with results reported from a previous study in which subjects with SCI and control subjects were matched for weight, height, and age.⁴⁵

Passive Leg Movements and Passive Cycling

During and after passive leg movements and passive cycling, no changes in blood flow in the femoral artery were observed in either the subjects with SCI or the control subjects. This finding is consistent with the results found by Svensson et al,⁴¹ who studied subjects with SCI in the spinal shock phase. They applied 30 flexion or extension movements of the leg joints and did not find an increase in calf blood flow, as measured by venous occlusion plethysmography. The present study revealed 2 additional findings. First, in subjects with chronic SCI, passive movements do not seem to be sufficient to improve LBF. Second, even a protocol like that used in rehabilitation centers fails to enhance blood flow. In contrast, Seifert et al²⁴ showed a significant increase in blood flow after passive movements of the paralyzed tibialis anterior muscle in subjects with SCI by using a method based on the detection of radioactive isotopes by a scintillation counter system. This increase continued for 8 minutes after application. Unfortunately, the authors did not describe clearly the types of passive movements that were used. In addition, before passive exercise, active exercise of the upper arm was performed. Because the authors did not report the use of a resting period between the interventions, crossover effects may explain the

increase in LBF. In the present study, none of the interventions caused an increase in blood flow; therefore, a crossover effect could not have been present. Only 1 study²⁵ investigated blood flow with Doppler sonography. The authors reported a significant increase in blood flow at the onset of passive leg extension in subjects who were healthy. However, blood flow was measured in the time before the limb returns (from knee extension to knee flexion) of the first few movements; therefore, the observed increase in blood flow could have been a short-term mechanical effect that passed away quickly.

Muraki and colleagues^{23,46} studied passive cycling in subjects with SCI and reported an increase in SV and CO. These results may suggest that it is possible to enhance LBF by use of passive cycling, through enhancement of MAP as a result of an increase in CO. However, in the present study, we found no changes in LBF during passive exercise. Apparently, an increase in CO during passive cycling may not imply that LBF will increase. A possible explanation for the findings of Muraki and colleagues may be related to the body position during their experiments. In that study, a normal bicycle ergometer was used, whereas in our study, subjects were seated in a wheelchair in front of a cycle ergometer. This difference is a major one, because in a sitting position on a standard bicycle ergometer, the lack of back support requires more muscle activation of the trunk and upper extremity. It is well known that the splanchnic area contains a major portion of the total blood volume and that the activation of, for instance, abdominal muscles may cause an increase in venous return. Through the Frank-Starling mechanism, this scenario may provoke an increase in SV. Moreover, static muscle activation results in increased oxygen demand and a subsequent increase in CO.

Unfortunately, Muraki and colleagues reported no information regarding the activity of muscles in the upper part of the body. They did measure skin blood flow in the lower limbs by laser Doppler imaging during passive cycling and reported no increase in skin blood flow. This finding is in agreement with the lack of changes in the peripheral circulation during passive exercise in the present study. Because we could not detect an effect of passive exercise on peripheral blood flow, tissue perfusion will not be enhanced during or after passive exercise. Tissue perfusion and related capillary blood flow are low in people with SCI and with impaired circulation in the lower part of the body⁵; low perfusion and low blood flow represent risk factors for pressure ulcers and poor wound healing.^{16,17}

During active exercise, neural control of the cardiovascular system is believed to encompass 2 major mechanisms.^{33,42} The first mechanism is called central command. Signals arising in a central area of the brain activate the motor cortex and, in a parallel fashion, activate the cardiovascular control areas in the medulla. This activation causes autonomic nervous system reactions, such as vagal withdrawal at the onset of exercise and a subsequent increase in heart rate. This mechanism serves as a feed-forward system that is related to the volitional component of exercise and therefore will not be activated by passive exercise.

The second mechanism is based on the activation of cardiovascular control areas in the medulla by afferent

information from chemoreceptors and mechanoreceptors in skeletal muscle. Chemoreceptors are activated whenever blood flow to the muscle is restricted, so that the delivery of oxygen and the washout of metabolites are reduced. Because no increase in oxygen consumption and no production of metabolites occur during passive exercise, afferent signals from chemoreceptors will not play a role in the control of the cardiovascular system. The mechanoreflex is a feedback control mechanism, evoked by mechanical stimuli caused by passively moved muscles and joints.^{31,33,47} In people with SCI, peripheral afferent reflexes are absent, because there is a disruption of the afferent pathway. Therefore, chemoreflexes as well as mechanoreflexes do not play a role in cardiovascular responses during passive exercise in people with SCI. In people who are healthy, however, it is possible that mechanoreflexes influence the cardiovascular system during passive exercise.

Another theory that has been postulated to explain possible cardiovascular changes caused by passive movements is the passive muscle pump, in analogy to the active muscle pump.^{31,48} According to this theory, rhythmic lengthening and shortening of muscles can induce cardiovascular responses because of increased venous return. In accordance with the Frank-Starling mechanism, an increase in venous return results in a subsequent increase in SV. A mechanism that is comparable to the passive muscle pump is the so-called ankle pump, in which the displacement of tendons may bring about a sucking action of deep and superficial veins underlying these tendons.^{30,34}

Because the central command and chemoreflexes are absent, only the passive muscle pump and the ankle pump could have induced cardiovascular changes during passive exercise in the subjects with SCI. Finally, it is possible that in the subjects with SCI, autonomic dysreflexia may have been induced by the passive exercise intervention. However, arguments against this theory are that there was no effect on LBF or MAP of any of the interventions and that, moreover, we did not observe any differences in responses between subjects with lesions high in the thoracic area and those with lesions low in the thoracic area.

In the present study, no changes in the peripheral circulation during passive exercise were found; therefore, we conclude that the passive muscle pump or the ankle pump, the mechanoreflexes, and the autonomic nervous system are not sufficient to enhance local blood flow during passive exercise. Although the results of this study do not support the use of passive movements or exercise for the prevention of cardiovascular disease-related secondary complications, physical therapists

should not be dissuaded from using these techniques to address musculoskeletal concerns.

Conclusion

The results of this study demonstrate that passive leg movements and passive cycling do not alter the arterial peripheral circulation in people with SCI or in people who are healthy. These results were indicated by unchanged arterial LBF, LVR, and MAP during and after passive exercise interventions like those typically used in the rehabilitation setting.

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