Blood Redistribution during Exercise in Subjects with Spinal Cord Injury and Controls

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ABSTRACT

THIJSSEN, D. H., S. STEENDIJK, and M. T. HOPMAN. Blood Redistribution during Exercise in Subjects with Spinal Cord Injury and Controls. *Med. Sci. Sports Exerc.*, Vol. 41, No. 6, pp. 1249–1254, 2009. **Introduction/purpose**: During exercise, redistribution of blood takes place to enhance blood flow to exercising muscles. To examine the role of sympathetic control in blood redistribution, we assessed blood flow in inactive regions (leg–splanchnic area) during arm-crank exercise in controls and in subjects with spinal cord injury (SCI) who lack central sympathetic control. **Methods**: SCI with a low lesion (≤T7; SCI-L) have no leg sympathetic control and SCI with high lesion (≥T6; SCI-H) lack sympathetic control in the legs and splanchnic area. This enables us to compare inactive regions between subjects with (controls, SCI-L; splanchnic) and without sympathetic innervation (SCI-L: leg, SCI-H: leg–splanchnic). Before and every 5 min during a 25-min arm-crank exercise bout at 50% of the individual maximal capacity, portal vein and femoral artery blood flow were measured. **Results**: Before exercise, portal vein blood flow was not different among groups. Arm-crank exercise induced a significant decrease in portal vein blood flow in subjects with splanchnic sympathetic control (able-bodied controls + SCI-L; ANOVA, $P < 0.05$), whereas SCI-H showed no change in portal vein blood flow. Baseline femoral artery blood was significantly lower in SCI compared with able-bodied controls. Exercise increased femoral artery blood flow in subjects with leg sympathetic control (controls; ANOVA, $P < 0.05$) but not in persons lacking sympathetic control in the leg (SCI). Leg vascular conductance did not change during exercise in both groups. **Conclusions**: In summary, portal vein blood flow decreases in subjects with sympathetic control of the splanchnic area, whereas exercise-induced changes in femoral artery hemodynamics did not differ between groups. These observations primarily indicate the presence of regional differences regarding the magnitude of exercise-induced blood redistribution *in vivo* in humans. **Key Words:** FEMORAL ARTERY, PORTAL VEIN, SPLANCHNIC AREA, SYMPATHETIC NERVOUS SYSTEM

During exercise, substantial cardiovascular adjustments are present. To optimally meet the increased metabolic demands of the contracting muscles, peripheral blood flow to inactive regions is relatively attenuated, resulting in a redistribution of blood directed toward the active areas (1,4,20). In healthy men, blood redistribution is demonstrated to be mainly present in the splanchnic area and in nonactive muscle vascular beds (14,17,20). Because the splanchnic circulation is under sympathetic control (3), and the observation that exercise increases sympathetic nerve activity (8,23), it is suggested that the exercise-induced reduction in splanchnic perfusion results from an elevation in sympathetic nerve activity (20). This physiological response contributes to the elevation of oxygen transport and substrates to sustain strenuous physical activities in humans.

Subjects with spinal cord injury (SCI) demonstrate an impaired ability to perform physical exercise (9,11). We previously reported that individuals with paraplegic demonstrate blood pooling in the inactive lower limbs during arm-crank exercise (10,13), which at least partly contributes to the limited increase in stroke volume in individuals with SCI during exercise (12). The lower stroke volume concomitantly results in an impaired cardiac output, which contributes to the impaired submaximal performance in SCI (9,11). The lack of sympathetic control below the level of the lesion in SCI in both the arterial and the venous systems is hypothesized to cause blood pooling in the limbs (13), which consequently attenuates stroke volume. However, no study has ever directly assessed blood flow changes in inactive regions during exercise in conditions with and without sympathetic control.

The aim of this study was to assess acute changes in blood flow in the brachial artery, portal vein, and femoral artery during arm-crank exercise in able-bodied controls, individuals with SCI with low (SCI-L, no leg sympathetic control) and high thoracic lesions (SCI-H, no leg + splanchnic sympathetic control). This enables us to compare exercise-induced blood flow changes in active (arm) and inactive regions (leg + splanchnic) between subjects with

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(controls and splanchnic area in SCI-L) and without sympathetic innervation (leg in SCI, leg + splanchnic area in SCI-H). We hypothesize that the presence of sympathetic control results in a decrease in blood flow in nonactive areas during arm-crank exercise and, therefore, affects blood redistribution during exercise.

METHODS

Subjects

Ten male individuals with SCI with a complete thoracic spinal cord lesion and 10 male able-bodied healthy controls participated in the study. On the basis of the presence of splanchnic sympathetic control, which is present when the spinal cord lesion is lower than T6, individuals with SCI were divided into high (SCI-H; T1–T6) and low thoracic lesion groups (SCI-L: T7–T12; Table 1). All individuals had normal arm function, were otherwise healthy, and performed physical activities on a regular basis. No subject reported having been diagnosed with cardiovascular disease, diabetes, increased insulin resistance, or cardiovascular risk factors such as hypercholesterolemia or hypertension. The study procedures were approved by the Ethics Committee of Radboud University Nijmegen Medical Center and adhered to the Declaration of Helsinki, and all subjects gave prior written consent.

Experimental Design

Participants reported twice to the laboratory. First, subjects performed an incremental, maximal arm-crank test to determine the individual maximal workload. On a second day, subjects performed arm-crank exercise at 50% of their individual maximal workload for 25 min. Before and within 5 s after this exercise session, brachial artery blood flow was assessed using echo Doppler. In addition, before and every 5 min during the test, portal vein and femoral artery blood flow were examined to assess vascular changes in active (brachial artery) and inactive vascular beds (portal vein and femoral artery), with (controls, portal vein in SCI-L) and without (femoral artery in all SCI, portal vein in SCI-H) sympathetic control during arm-crank exercise.

TABLE 1. Subject characteristics of able-bodied participants (C, n = 10) and of SCI subjects with lesion below T6 (SCI-L, n = 5) or above T6 (SCI-H, n = 5).

	C (n = 10)	SCI-L (n = 5)	SCI-H (n = 5)	P
Age (yr)	28 ± 4	29 ± 5	31 ± 5	0.44
Weight (kg)	78.1 ± 9.7	65.2 ± 14.5	74.6 ± 9.3	0.13
Height (cm)	182 ± 9	185 ± 3	183 ± 7	0.79
Systolic blood pressure (mm Hg)	140 ± 14	128 ± 8	125 ± 24	0.19
Diastolic blood pressure (mm Hg)	70 ± 7	77 ± 15	69 ± 23	0.65
Sport activity ($h \cdot wk^{-1}$)	4.3 ± 3.5	3.1 ± 2.6	2.4 ± 2.3	0.51
Duration of lesion (yr)		9.4 ± 2.8	9.6 ± 4.7	0.94
Peak workload arm-crank test (W)	135 ± 30	130 ± 7	106 ± 18	0.10
Oxygen uptake submaximal test ($mL\ O_2 \cdot kg^{-1} \cdot min^{-1}$)	18.4 ± 2.0*	19.5 ± 3.8*	14.6 ± 2.1	0.02

Data are presented as mean ± SD.
* *Post hoc* significant from SCI-H at $P < 0.05$.

Experimental Procedures

Day 1: Maximal arm-crank test. Exercise testing was performed in a temperature-controlled room (23 ± 1°C). During each test, exercise was performed using an electromagnetic arm-crank ergometer (Angio300; Lode, Groningen, the Netherlands). Subjects (controls as well as SCI) were seated in a wheelchair in front of the arm ergometer. The axis of the arm-crank was at shoulder level, with the elbows slightly flexed at the point of maximal extension while cranking. An incremental arm-crank test was performed, starting at 10 W and increasing the workload with 10 W every minute until voluntary exhaustion, to assess maximal performance.

Day 2: Exercise bout. Subjects were seated in a wheelchair in front of the arm ergometer, using the setting previously applied during the maximal arm-crank test. A back support was present to assist in the stabilization of the trunk and to minimize leg contractions. In addition, the legs were slightly elevated, so that the legs could not be used as a fulcrum. During a familiarization session, we instructed subjects to avoid leg stabilization movements that could eventually impact femoral artery blood flow measurements. To examine the level of lower limb muscle activity during arm-crank exercise, which might influence femoral artery blood flow measurements in the control subjects, we repeated our protocol and studied EMG activity of the quadriceps and hamstring muscle in three subjects. We expressed EMG activity relative to EMG activity during a maximal voluntary contraction. Muscle activity in the quadriceps and hamstring muscles was slightly higher during arm-crank exercise (1.0 ± 0.2% to 2.6 ± 1.5% maximal voluntary contraction (MVC) and 0.8 ± 0.3% to 1.1 ± 0.2% MVC, respectively).

After a resting period of 25 min, baseline diameter and velocity of the femoral and brachial artery were assessed. For the assessment of the femoral artery at rest and during arm-crank exercise, the trunk was kept at 60° with the seat. Using a 5-MHz echo Doppler ultrasound device (SSA 270A; Toshiba, Tokyo, Japan), blood flow and vessel diameter were assessed by a well-trained sonographer. The transducer was held consistently at an angle of 60°. To assess brachial artery blood flow, the right forearm rested on a table at heart level and supported by foam. Brachial artery blood flow was examined at the distal one third of the right upper arm. The portal vein was distinguished from other vessels by flow pattern, flow direction, and anatomy. Portal vein blood flow was measured a little to the right of the midline, below the rib cage (subcostal), or between the ribs (intercostal). Measurements were made at the level of the intrahepatic part of the common portal vein (18). The femoral artery was measured 2 cm proximal to the bifurcation into the deep and superficial femoral artery of the right leg.

After baseline measurements, subjects started with the exercise session, each person at 50% of their individual maximal arm-crank workload. Every 5 min, the diameter and velocity of femoral and portal vein arteries were recorded

immediately after each other in the last minute of each 5-min bin. In addition, mean arterial blood pressure was measured manually in the lower limb. Owing to a limitation in time, no diameter and red blood cell velocity recordings were performed during the last interval of the exercise bout. Within 5 s after cessation of the exercise protocol, brachial artery diameter and red blood cell velocity were measured.

Data Analysis

One investigator analyzed offline recordings of arterial images and red blood cell velocities. Resting diameters were measured from three consecutive images and were then averaged for each individual vessel. Furthermore, from the corresponding Doppler spectrum waveforms, the mean blood velocity V_{mean} ($cm \cdot s^{-1}$), defined as the average velocity of the enveloping Doppler spectrum during the entire cardiac cycle, was calculated and averaged. Subsequently, mean blood flow was calculated from the product of the arterial cross-sectional area (πr^2) and the V_{mean} (5).

Statistics

Statistical analyses were performed using SPSS 14.0 (SPSS, Chicago, IL) software. All data are reported as mean ± SD, and statistical significance was assumed at $P \leq 0.05$. A paired *t*-test was used to examine the effect of exercise on brachial artery blood flow. A two-way repeated-measures ANOVA was used to assess the impact of arm-crank exercise on blood flow in nonactive regions during exercise (within-subject variable) between subjects with and without sympathetic control of the vascular region of interest (between-subject variable). This means that, for the portal vein, we compared controls + SCI-L (with sympathetic) versus SCI-H (without sympathetic), whereas for the femoral artery, we compared controls (with sympathetic) versus SCI-L + SCI-H (without sympathetic). We calculated the effect size (ES = mean difference between groups / pooled SD) for the exercise-induced changes in portal vein and femoral artery blood flow to gain better insight into the (clinical) relevance of a given finding (22). For tables and figures, we presented the data for each individual group separately (controls, SCI-L, and SCI-H).

RESULTS

Baseline subject characteristics were not different among groups, except oxygen uptake during the submaximal exercise bout, which was similar between controls and SCI-L but lower in SCI-H (Table 1). The maximal workload during the arm-crank test was not different among groups. Resting arterial pressure did not differ among groups, whereas mean blood pressure increased significantly during the exercise bout in able-bodied controls only (Table 2). Baseline brachial artery blood flow and vascular conductance were not different between controls, SCI-H and SCI-L. Arm-crank exercise increased brachial artery blood flow and vascular conductance after exercise (Table 3).

TABLE 2. Changes in mean arterial pressure during the 25-min arm-crank exercise protocol at regular intervals of 5 min in able-bodied participants (C, $n = 10$) and in SCI subjects with lesion below T6 (SCI-L, $n = 5$) or above T6 (SCI-H, $n = 5$).

	Baseline	5 min	10 min	15 min	20 min	0–20 min	*P*
C	93 ± 9	120 ± 23	119 ± 10	116 ± 11	113 ± 13	117 ± 8	<0.001
SCI-L	94 ± 12	103 ± 13	93 ± 16	106 ± 17	96 ± 20	99 ± 16	0.56
SCI-H	88 ± 23	98 ± 23	99 ± 15	97 ± 18	111 ± 21	98 ± 18	0.86

P values represent *t*-test between baseline and average blood pressure between 0 and 20 min (0–20 min).
Data are presented as mean ± SD.

TABLE 3. Mean brachial artery blood flow (BF, $mL \cdot min^{-1}$) and vascular conductance (VC, mm $Hg \cdot mL^{-1} \cdot min^{-1}$) before and after the 25-min arm-crank exercise protocol in able-bodied participants (C, $n = 10$), SCI-L ($n = 5$), and SCI-H ($n = 5$).

	Baseline	After Exercise	*P*
Blood flow			
C + SCI-L + SCI-H	300 ± 128	492 ± 286	0.001
C	303 ± 157	556 ± 321	
SCI-L	314 ± 46	602 ± 285	
SCI-H	281 ± 140	345 ± 167	
Vascular conductance			
C + SCI-L + SCI-H	3.3 ± 1.4	5.8 ± 5.2	0.009
C	3.3 ± 1.9	5.2 ± 2.9	
SCI-L	3.2 ± 1.0	11.7 ± 7.9	
SCI-H	3.4 ± 0.5	4.3 ± 1.5	

Because all subjects have sympathetic control of the brachial artery, data are pooled for all three groups (C + SCI-L + SCI-H) and analyzed using a paired Student's *t*-test.
Data are presented as mean ± SD.

Portal vein blood flow during arm-crank exercise. Baseline portal vein blood flow was similar among groups (Fig. 1). The two-way ANOVA reported no impact of exercise on portal vein blood flow ($P = 0.46$) and no interaction-effect was also observed (two-way ANOVA, $P = 0.22$). However, the effect size of exercise on portal vein blood flow between the subjects with and without sympathetic control was 1.1 (0.25/0.22 $mL \cdot min^{-1}$), which is well above 0.8 and, therefore, can be regarded as a large effect (22). In addition, the relative change in portal vein blood flow in subjects with sympathetic regulation (−26 ± 29%)

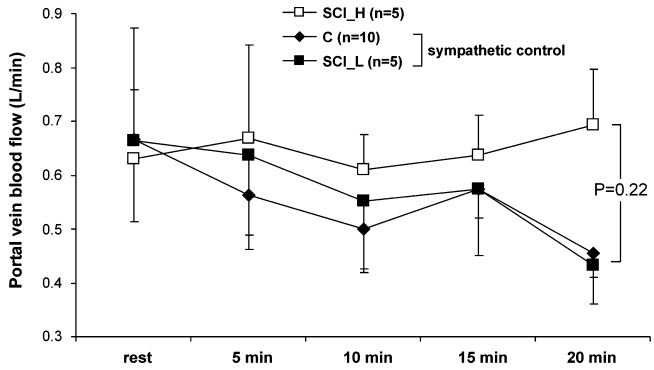

FIGURE 1—Portal vein blood flow ($L \cdot min^{-1}$) in able-bodied controls ($n = 10$; *black diamonds*), SCI-L ($n = 5$, sympathetic splanchnic control; *black squares*), and SCI-H ($n = 5$, no sympathetic splanchnic control; *open squares*) at baseline and at 5-min intervals during the arm-cranking exercise. *P* values reported at the end of each line represent a one-way ANOVA for subjects with (controls + SCI-L, $n = 15$) and without splanchnic sympathetic control (SCI-H, $n = 5$). The other *P* value represents the interaction effect for the two-way ANOVA between these two groups. *Error bars*, represent SE.

was significantly different from the change in subjects without sympathetic control (33 ± 78%, *t*-test; $P = 0.02$).

Femoral artery blood flow during arm-crank exercise. At baseline, SCI groups demonstrated a significantly smaller femoral artery diameter, lower blood flow, and vascular conductance than controls (Fig. 2; *t*-test, $P < 0.001$). Arm-crank exercise did not change femoral artery diameter in any group (Fig. 2). Arm-crank exercise increased femoral artery blood flow, with no interaction between both SCI and control (two-way ANOVA; Fig. 2). Because baseline differences in femoral artery blood flow were present, we used relative changes from baseline to calculate the effect size. Because SCI subjects demonstrated an increase of 56 ± 90% and able-bodied controls demonstrated a change of 53 ± 35% (*t*-test, $P = 0.93$) while the effect size was 0.04, this represents no relevant effect (<0.2) (22). Femoral artery vascular conductance did not change significantly and revealed no interaction effect (Fig. 2).

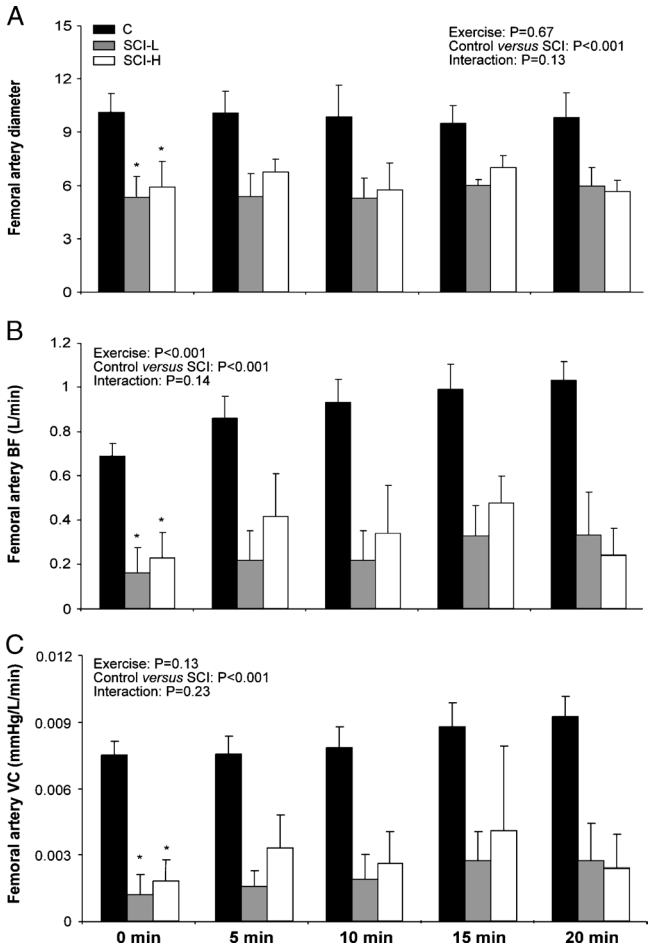

FIGURE 2—Femoral artery diameter (A, mm), mean blood flow (B, L·min^{-1}), and mean vascular conductance (C, L·min^{-1}·mm Hg^{-1}) in able-bodied controls ($n = 10$; *black bars*), SCI-L ($n = 5$, *gray bars*), and SCI-H ($n = 5$, *white bars*) at baseline and at 5-min intervals during the arm-cranking exercise. Data from the two-way ANOVA are presented for each parameter regarding the within-subject factor (exercise), between-subject factor (controls vs SCI), and the interaction effect. Asterisk (*) represent an unpaired *t*-test demonstrating a significant difference compared with controls at $P < 0.001$. *Error bars*, represent SE.

DISCUSSION

Supraspinal sympathetic control is regarded as a key regulator of blood redistribution during exercise in humans. To the best of our knowledge, this is the first study to examine exercise-induced changes in blood flow in nonactive regions in persons with and without supraspinal sympathetic control of these nonactive areas. Examining the portal vein, which transports blood from the splanchnic region, subjects with and without sympathetic splanchnic regulation demonstrated a large difference (*effect size*) in portal vein blood flow at the end of arm-crank exercise. Whereas subjects with sympathetic control (controls and SCI-L) showed a decrease in portal vein blood flow during arm-crank exercise, persons without sympathetic splanchnic control (SCI-H) demonstrated no change. This finding confirms our hypothesis that the sympathetic nervous system regulates blood redistribution in the splanchnic region during exercise in humans. In the femoral artery, vascular conductance during arm-crank exercise in controls and in SCI individuals did not change. This finding is rather surprising given our previous observations that calf volume decreases in controls during arm-crank exercise. Nonetheless, this finding primarily suggests that different inactive vascular beds, i.e., the splanchnic area and a muscle vascular bed, respond differently to the same exercise stimulus. The splanchnic region, which contains a large blood volume, seems to respond more distinct than the muscle vascular bed regarding blood redistribution during exercise.

During resting conditions, ~50% of the total blood volume in humans is in the splanchnic area, the body's largest blood reservoir (1). Blood redistribution from the splanchnic area, including the kidneys, is the first and most important step to redirect blood to the active muscles. In our study, subjects showed a ~35% decrease in portal vein blood flow during an arm-crank exercise bout at 50% of the individual maximal workload. This change in blood flow is somewhat lower than reported in a previous study, which found a ~50% decrease after 20 min of cycling exercise at 70% of the individual maximal workload (18). Previous research indicated a direct relationship between the relative exercise intensity, the involved muscled mass and magnitude in splanchnic blood flow change during exercise (21). This relationship may explain the somewhat smaller decrease in portal vein blood flow during arm-crank exercise found in our study.

Although previous studies indicate, primarily on the basis of animal studies, that sympathetic control is responsible for blood redistribution from the splanchnic region during exercise (20), this has never been directly examined in humans *in vivo*. A unique aspect of our study is that we examined individuals with SCI who, based on the level of their spinal cord lesion (T1–T6, SCI-H), lack supraspinal sympathetic control. In contrast with the exercise-mediated

decrease of portal vein blood flow in controls and SCI-L individuals, individuals with SCI-H demonstrated no change in portal vein blood flow. This novel finding confirms our hypothesis that the sympathetic nervous system is a key player regarding the exercise-induced blood redistribution from the splanchnic region in humans *in vivo*.

Control subjects demonstrated a minor increase in femoral artery blood flow, without a change in vascular conductance. Therefore, the exercise-associated increase in mean arterial pressure is primarily responsible for the elevated femoral artery blood flow in controls. This finding is in agreement with previous studies. Green et al. (6,7), who examined brachial artery flow in response to an incremental leg cycling exercise protocol, found an increase in blood flow at higher exercise levels. Nonetheless, the small increase in blood flow is somewhat surprising given our previous finding of a decrease in calf volume during arm-crank exercise in healthy subjects (13). Possibly, the observed decrease in calf volume relates more to the venous system than to arterial adaptations.

Interestingly, the arm-crank exercise-induced changes in the femoral artery contrasts with our observation in the portal vein. Whereas portal vein blood flow decreases, femoral artery vascular conductance does not change. This suggests that, during exercise, blood redistribution takes place at a different magnitude in different inactive areas. In humans, such a regional difference has previously been found between both limbs (24). On the basis of our results, regional differences also seem to exist between the splanchnic region and muscle vascular beds. Recent findings in animal studies support this hypothesis. For example, electrical-stimulated physical activity in rats induces less vasoconstriction in skeletal muscles than in kidney, which was related to a neural mechanism (15). In addition, the splanchnic bed is densely innervated with postganglionic, sympathetic adrenergic neurons that terminate in the adventitia of the vascular wall (2), which helps to rapidly redistribute blood from the splanchnic area. Muscle vascular beds are relatively densely innervated with dilating β-adrenergic receptors (19). As a result, circulating epinephrine can antagonize the vasoconstrictor activity of sympathetic stimulation during exercise, primarily in muscle vascular beds. Although speculative, these factors may explain the regional differences in blood redistribution observed during exercise in humans.

Individuals with SCI demonstrated a change neither in femoral artery blood flow nor in vascular conductance during exercise. In line with our findings, we did not expect vascular conductance to change because the legs of individuals with SCI lack sympathetic control and, therefore, cannot increase resistance below the level of the lesion during exercise (13). Femoral artery blood flow, however, was hypothesized to increase as a direct result of the exercise-induced rise in arterial blood pressure. Whereas controls reported an increased femoral artery blood flow, the lower increase in blood pressure in individuals with SCI compared with controls likely explain the unaltered femoral artery blood flow in SCI during arm-crank exercise. Another mechanism that may contribute to blood flow changes during exercise relates to systematically circulating neurotransmitters (e.g., dopamine and neuropeptide Y [23]) or local vasoconstrictors (e.g., endothelin 1 [16]). However, the intensity level of our arm-crank exercise bout may have been too low to induce such changes in our study.

Limitations. Because we did not find a significant increase in brachial artery blood flow in all groups, one may hypothesize that the level of exercise intensity was too low. However, owing to technical restrictions of the echo Doppler technique in measuring the brachial artery during arm exercise, we examined diameter and blood flow before and immediately after the exercise bout. An important limitation of such approach is that brachial artery blood flow rapidly decreases after cessation of the exercise bout, most likely resulting in an underestimation of brachial artery blood flow. When analyzing all subjects in a pooled analysis, a significant exercise-induced increase in brachial artery blood flow is observed. This suggests that the small number of the subgroups for this comparison, but also for the portal vein, could explain the lack of significance level for some comparisons.

A limitation of the present study is that we did not directly examine the sympathetic nervous system to verify the exercise-induced increased activity of this system. However, the exercise bout was performed at 50% of the individual maximal workload, leading to an approximately fivefold increase in oxygen uptake. On the basis of previous studies, much lower levels of intensity are already associated with a marked increase in sympathetic nerve activity (8,23). Therefore, we believe that our exercise protocol induced an increase in sympathetic nerve activity in all subjects. Another limitation related to the fact that we were not able to examine leg muscle EMG activity during our protocol to exclude leg muscle contractions for the stabilization of the trunk in all subjects. However, in three able-bodied subjects, we repeated our protocol with the addition of EMG electrodes on the quadriceps and hamstring muscle. We expressed EMG activity relative to EMG activity during a maximal voluntary contraction. The muscle activity in the quadriceps and hamstring muscles was slightly higher during arm-crank exercise ($1.0 \pm 0.2\%$ to $2.6 \pm 1.5\%$ MVC and $0.8 \pm 0.3\%$ to $1.1 \pm 0.2\%$ MVC, respectively). This modest change from baseline is unlikely to explain the main findings of our study because we found no interaction-effect between SCI (who have paralyzed legs) and controls regarding the effect of arm cranking on femoral artery mean flow, conductance, or shear rate (Figs. 1 and 2).

In summary, the marked differences in portal vein blood flow changes in subjects with (i.e., vasoconstriction) or without sympathetic control (i.e., unaltered blood flow) indicate the importance of supraspinal sympathetic innervation to effectively constrict the splanchnic region to redistribute blood toward the active muscles. In the femoral

artery, a small increase in blood flow in controls and no change in vascular conductance in controls as well as in SCI were found. As controls demonstrated vasoconstriction in the portal vein, but not in the femoral artery, this remarkable observation indicates the presence of regional differences regarding exercise-induced blood redistribution in humans; that is, the splanchnic region, which contains a large blood volume, seems to respond more distinct than an inactive muscle vascular bed. Accordingly, specified blood redistribution in nonactive regions seems to contribute in optimally meeting the altered metabolic demands in humans during exercise.

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