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ORIGINAL ARTICLE

Post-exercise heart rate recovery in individuals with spinal cord injury

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Study design: Prospective comparison of spinal cord injured (SCI) subjects and ambulatory subjects.
Objectives: To determine the effects of the presence and level of SCI on heart rate recovery (HRR).
Setting: Outpatient SCI center.
Methods: HRR was determined in 63 SCI subjects (26 with tetraplegia, 22 with high-level paraplegia, 15 with low-level paraplegia) and 26 ambulatory subjects. To adjust for differences in heart rate reserve between groups (HRpeak minus HRrest), HRR was also 'normalized' to a range of 1 at peak heart rate and to 0 at 8 min, and the shapes of HRR curves were compared.
Results: Although absolute HRR was similar between high- and low-level paraplegia, it was significantly more rapid in participants with paraplegia at 2, 5 and 8 min after exercise than in those with tetraplegia (39 ± 14 vs 29 ± 14 b.p.m., $P<0.05$; 51 ± 14 vs 33 ± 16 b.p.m., $P<0.01$ and 52 ± 16 vs 36 ± 17 b.p.m., $P<0.01$, respectively). HRR among ambulatory subjects was more rapid than among those with tetraplegia at all time points in recovery. However, when normalized for heart rate reserve, HRR was significantly more rapid in tetraplegic subjects ($P<0.001$ vs paraplegia and ambulatory subjects).
Conclusion: In SCI, HRR is strongly associated with the peak exercise level and peak heart rate achieved during exercise testing.
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Keywords: exercise testing; heart rate; heart rate recovery; oxygen uptake; paralysis

Introduction

The degree of autonomic dysfunction is an important mediator of physical function and overall health in persons with spinal cord injury (SCI). Autonomic nervous system (ANS) imbalance cannot be quantified directly, but is usually inferred by functional classification systems based on level and completeness of injury, tests of functional status or noninvasive measurements such as head-up tilt testing or heart rate variability.[1,2] In recent years, the rate at which heart rate recovers from exercise (termed heart rate recovery, or HRR) has been used in ambulatory individuals to reflect the integrity of the ANS and thus is considered as an index of cardiovascular health. Impaired HRR has been associated with ANS dysfunction in a wide range of conditions, including diabetic neuropathy, hypertension, coronary artery disease and chronic heart failure.[2,3] Using pharmacologic manipulation (β- and α-blockade) of ANS function in athletes and patients with chronic heart failure, Imai *et al*.[4] observed that the rapidity of HRR was mediated primarily by vagal reactivation in the immediate post-exercise period. Numerous subsequent studies over the past decade have reported a strong association between vagal tone as evidenced by HRR and mortality in patients with cardiovascular disease and other conditions.[2,5,6]

Individuals with SCI potentially represent a good model to study ANS dysfunction during and after exercise, because these individuals are characterized by a disruption of the normal autonomic cardiovascular control mechanisms.[1,7] For example, in many subjects with SCI, the heart rate response to an exercise stimulus is blunted, disruptions in vasomotor tone lead to abnormalities in blood pressure and cardiac rhythm disorders are common.[8] However, there are no systems in common clinical use to quantify autonomic function. HRR has the potential to better characterize the degree of autonomic imbalance in SCI, and several groups have recently used HRR for this purpose.[9–11]

Previous studies on HRR have generally compared only heart rate at a given point in recovery (for example, 1 or 2 min) between patients with favorable and poor outcomes. Some investigators have theorized that the transition

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processes from sympathetic control of heart rate at peak exercise to vagally mediated heart rate at rest are reflected in the shape of the HRR curve, providing additional insight into autonomic balance.[12–14] It is also well known that SCI individuals have a reduced heart rate reserve (the difference between peak and resting heart rates), particularly among tetraplegic subjects whose loss of sympathetic motor function limits the ability to increase the heart rate. Among ambulatory subjects, heart rate reserve has been suggested to have a significant effect on HRR, the shape of the HRR curve and its association with outcomes.[12–15]

In this study, we used a more population-specific method in which HRR was normalized for heart rate reserve,[12–14] and compared the shape of the HRR curves between subjects with tetraplegia and paraplegia with those of an age-matched group of ambulatory subjects. Our objectives were (1) to characterize HRR in SCI individuals relative to ambulatory subjects; (2) to determine the association between HRR and level and completeness of injury in persons with SCI and (3) to assess the clinical and exercise test determinants of HRR in persons with SCI.

Patients and methods

Subjects

A total of 63 individuals with SCI and 26 ambulatory subjects participated in the study. Subject characteristics are presented in Table 1. Ambulatory subjects were age matched specifically to the tetraplegic subjects. In all 26 individuals with tetraplegia, 22 with high paraplegia (injury level T2–T6), 15 with low paraplegia (injury level T7–S1) and 26 ambulatory subjects were included in this study. All subjects with SCI sustained injuries at least a year before the study and were functionally nonambulatory (that is, no capacity to ambulate or a limited capacity to ambulate for weight-bearing purposes only). The subjects were generally sedentary, but activity status was not used as an exclusion/inclusion criteria. Subjects were excluded from participation if they had comorbid neurologic conditions, other serious medical conditions or were currently taking β-blockers. Written informed consent was obtained using a protocol approved by the Stanford University Institutional Review Board.

Exercise testing

All subjects with SCI completed symptom-limited exercise tests using a manually incremented arm ergometry protocol, and testing was performed in the upright-seated position using an arm ergometer (Ergometrics 800; Ergoline, Bitz, Germany or Monark Rehab Trainer 881E; Varberg, Sweden). Each subject's personal wheelchair was used for the test, positioned securely by brakes and, when necessary, a technician held the chair steady. The height of the ergometer was adjusted so that the fulcrum was horizontal with the shoulder. In subjects with tetraplegia who had difficulty grasping the handlebars, gloves were used to secure the hands. Work increments were individualized (from 1 to 10 W min^{-1}) such that the targeted test duration was between 8 and 12 min. The mean work rate increments were 1.0 ± 0.88, 4.60 ± 3.1 and 4.86 ± 2.6 W min^{-1} for tetraplegic, high paraplegic and low paraplegic subjects, respectively. Subjects were requested to maintain cadence at 60 r.p.m. throughout the test. All tests were continued to the point of volitional fatigue. A 12-lead electrocardiogram and cardiopulmonary exercise responses were recorded at rest, throughout exercise and for an 8 min period after completion of the test while the subject remained upright.

Ambulatory subjects underwent treadmill testing using an individualized ramp protocol (Schiller CS-200; Baar, Switzerland). Treadmill testing was used for ambulatory subjects to compare these responses to previous studies in ambulatory subjects, and because in the seminal studies on HRR, this response was shown to predict outcomes independently of peak heart rate and exercise level achieved.[5] A pretest questionnaire was used to determine the appropriate work rate for each individual such that the targeted test duration was between 8 and 12 min.[16] Standard 12-lead electrocardio-

Table 1 Demographic information

	Ambulatory subjects (n = *26*)	*Low paraplegia* (n = *15*)	*High paraplegia* (n = *22*)	*Tetraplegia* (n = *26*)	P-*value*[a]
Age (years)	50 ± 10	54 ± 15	50 ± 11	50 ± 10	0.65
BMI (kg m^{-2})	27.8 ± 4.3	25.8 ± 4.7	27.8 ± 6.6	26.3 ± 5.6	0.50
Weight (kg)	86.5 ± 17.5	80.1 ± 16.8	88.3 ± 25.5	82.1 ± 19.7	0.55
Height (cm)	176.2 ± 7.6	174.0 ± 9.9	176 ± 9.4	175.0 ± 10.5	0.88
Duration of injury (years)	NA	22 ± 12	13 ± 12	19.0 ± 12	0.08
ASIA class					
A: *n* (%)	NA	6 (40.0)	13 (59.1)	10 (38.5)	—
B: *n* (%)	NA	3 (20.0)	3 (13.6)	6 (23.1)	—
C: *n* (%)	NA	5 (33.3)	4 (18.2)	6 (23.1)	—
D: *n* (%)	NA	1 (6.7)	0	3 (11.5)	—
Unknown: *n* (%)	NA	0	2 (9.1)	1 (3.8)	—
Level of injury	NA	T7, 2; T8, 3; T10, 1; T12, 5; L1–5, 4	T2, 2; T3, 3; T4, 6	T5, 7; T6, 4	C5, 12; C6, 7; C7, 7

Abbreviations: ASIA, American Spinal Injury Association; BMI, body mass index.
[a]*P*-value reflects main effect between groups by ANOVA.

grams and cardiopulmonary responses were obtained throughout the exercise test and for an 8 min period during recovery. Heart rate was recorded at rest (15 min in the supine position), during peak exercise and during recovery at 2, 5 and 8 min. The Borg 6–20 perceived exertion scale was used for both SCI and ambulatory populations to quantify subject effort at 1 min intervals. Exercise was continued until volitional fatigue; no heart rate targets were used to terminate the tests.

Oxygen uptake (VO_2) and other cardiopulmonary exercise responses were obtained using the Quark K4b^2 system (Cosmed, Rome, Italy). The oxygen and carbon dioxide sensors were calibrated before each test using gases with known concentrations, and the flow sensor was calibrated before each test using a 3 liter syringe. Data were acquired breath-by-breath and expressed as rolling 30 s averages printed every 10 s.

Heart rate recovery

Heart rate recovery was quantified in two ways. First, it was expressed in a conventional manner as the absolute decrease in heart rate after exercise as: (peak heart rate−heart rate at 2, 5 and 8 min in recovery). This is termed absolute HRR. HRR curves were then derived by dividing HRR into two elements: a normalized recovery curve that characterizes how quickly peak heart rate (HR_{peak}) recovers to a posttest resting rate, and an amplitude scaling term defined by the difference between HR_{peak} and post-exercise HRrest as described previously.[12,13] This is illustrated in Figure 1. To compare the shape of the normalized recovery curves, we standardized HRR to a uniform range of 1.0 at peak heart rate and to 0 at 8 min into recovery (HRR_8). HRR_8 was subtracted from each HRR value and the difference was divided by ($HR_{peak}-HRR_8$). This normalization process supports the comparison of the shape of the recovery curve independent of the amplitude scaling factor related to changes in HRpeak and HRrest. This is termed normalized HRR, and reflects the percentage change in recovery heart rate over the transition from peak exercise to late resting recovery.

As an example to illustrate normalized heart rate, consider a subject who reaches a peak heart rate of 140 b.p.m., recovers to 120 b.p.m. at 2 min into recovery and has a stable heart rate of 95 b.p.m. at 8 min into recovery. At the 2 min recovery point, the patient's normalized HRR is (120−95)/(140−95) = 55%; the patient's heart rate is still 55% above the stable resting recovery rate. The method allows recovery comparisons to be made without the confounding influence of variations in HRpeak.

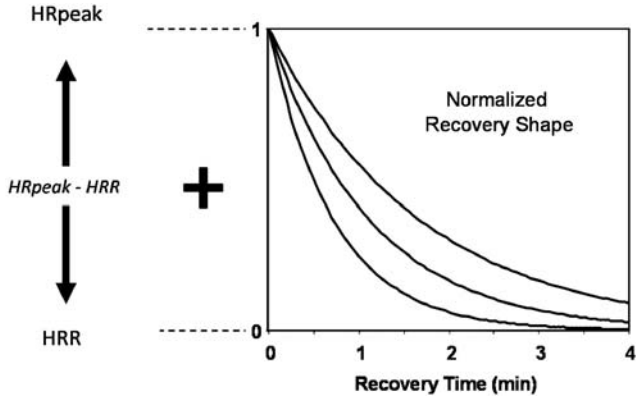

Figure 1 An example of the calculation of normalized heart rate recovery curves.[12–14] HR_{peak}, peak heart rate achieved; HRR, heart rate recovery.

Data analysis

Clinical, exercise and demographic data between groups (SCI with tetraplegia, high vs low paraplegia and ambulatory subjects) were assessed by one-way analysis of variance for continuous variables and by χ^2-tests for categorical data. Because HRR values and other exercise test responses were similar between subjects with high and low paraplegia, the high and low paraplegia groups were combined. Heart rate reserve was defined as ($HR_{peak}-HR_{rest}$). Comparisons of HRR between different groups at each time point in recovery and the normalized HRR curves were assessed by one-way analysis of variance. The Bonferroni procedure was used to perform *post hoc* comparisons between groups. The associations between HRR and other clinical and exercise data were assessed using linear regression. A forward stepwise multiple regression procedure was used to determine clinical and exercise test predictors of HRR. All analyses were performed using NCSS software (Kayesville, UT, USA).

Results

No significant differences in demographic data were observed between the SCI categories, or between SCI and ambulatory subjects (Table 1). Exercise test responses, including absolute HRR at 2, 5 and 8 min, are presented in Table 2. Peak exercise responses were generally higher in ambulatory subjects relative to those with SCI. The responses of subjects with tetraplegia were attenuated relative to both the high- and the low-paraplegia subjects, whereas high- and low-paraplegia subjects were similar.

Absolute HRR responses are illustrated in Figure 2. There was a significant main effect for 2, 5 and 8 min ($P<0.01$), with HRR being greater (more rapid) among ambulatory subjects compared with both SCI groups, and HRR being more rapid in paraplegic compared with tetraplegic subjects. Figure 3 illustrates the HRR curves when normalized for differences in heart rate reserve. In the latter case, the converse was observed; HRR was more rapid among tetraplegic subjects ($P<0.001$ vs ambulatory subjects), and HRR was slowest among ambulatory subjects ($P<0.001$ ambulatory vs paraplegic subjects).

Table 3 presents correlation coefficients between absolute HRR, pretest variables and exercise test responses among subjects with paraplegia and tetraplegia. HRR at 2, 5 and 8 min was significantly associated with peak oxygen uptake, HRpeak and heart rate reserve, but weakly related to body mass index, HRrest and blood pressure. Age was significantly and inversely related to HRR at 2, 5 and 8 min among paraplegic but not tetraplegic subjects. Table 4 presents predictors of 2 min absolute HRR from clinical and exercise

Table 2 Exercise test responses

	Ambulatory subjects (n = *26*)	*Low paraplegia* (n = *15*)	*High paraplegia* (n = *22*)	*Tetraplegia* (n = *26*)	P-*value*[a]
Rest					
HR_{rest} (beats per min)	73.3 ± 18.3	74.7 ± 14.8	74.6 ± 15.2	66.7 ± 12.2	0.23
Resting systolic BP (mm Hg)	132.5 ± 12.6[b]	126.9 ± 16.9	120.1 ± 16.8	106.3 ± 20.7[c]	<0.001
Resting diastolic BP (mm Hg)	86.9 ± 7.5[b]	76.1 ± 11.6	77.8 ± 10.8	74.4 ± 14.4	<0.001
Exercise					
HR_{peak} (beats per min)	166.2 ± 15.8[b]	136.0 ± 29.2	143.8 ± 24.9	106.6 ± 20.5[c]	<0.001
VO_{2peak} (ml O_2 per kg min^{-1})	36.4 ± 10.5[b]	15.1 ± 4.1	12.8 ± 4.6	10.3 ± 4.3	<0.001
VE (l min^{-1})	103.7 ± 27.4[b]	59.1 ± 26.6	50.7 ± 19.7	35.5 ± 13.8[c]	<0.001
RER	1.15 ± 0.13	1.24 ± 0.19	1.22 ± 0.14	1.22 ± 0.28	0.43
Peak power (W)	NA	50.0 ± 38.5	52.0 ± 33.5	18.3 ± 16.7[c]	<0.001
Recovery					
HRR 2 min (beats per min)	43.6 ± 19.6[b]	39.6 ± 17.0	38.8 ± 12.5	28.7 ± 14.4[c]	<0.001
HRR 5 min (beats per min)	69.6 ± 21.3[b]	50.1 ± 14.3	52.2 ± 14.5	33.3 ± 15.5[c]	<0.001
HRR 8 min (beats per min)	71.0 ± 21.4[b]	52.4 ± 17.2	51.7 ± 15.7	36.1 ± 17.4[c]	<0.001

Abbreviations: BP, blood pressure; HR, heart rate; HRR, heart rate recovery; RER, respiratory exchange ratio; VE, minute ventilation.
[a]*P*-value for ANOVA main effect between groups.
[b]$P<0.05$ vs paraplegia and tetraplegia.
[c]<0.05 compared to paraplegia.

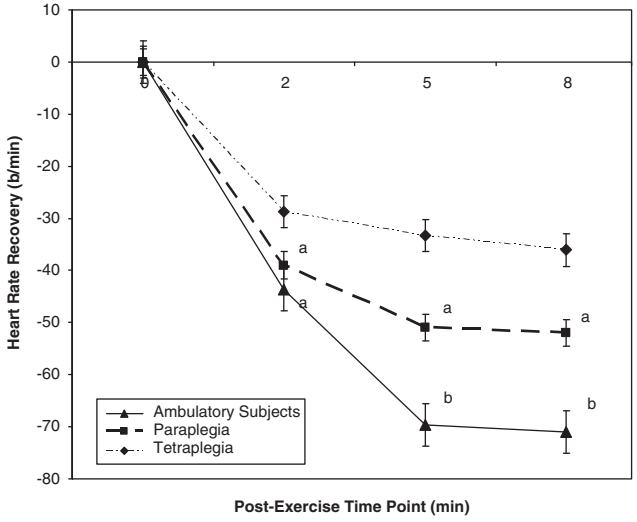

Figure 2 Comparison of heart rate recovery responses at 0, 2, 5 and 8 min between tetraplegia, paraplegia and ambulatory subjects. [a]$P<0.05$ vs tetraplegia; [b]$P<0.05$ vs tetraplegia and paraplegia.

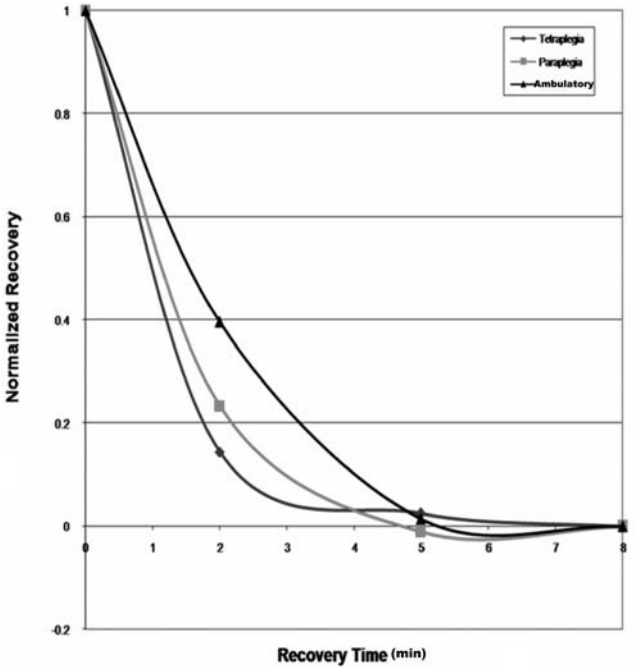

Figure 3 Normalized heart rate recovery curves for tetraplegic, paraplegic and ambulatory subjects. The decline in normalized heart rate recovery (HRR) was significantly more rapid among both tetraplegic and paraplegic subjects compared with ambulatory subjects ($P<0.001$ for both).

test data in the ambulatory and SCI groups. In both SCI and ambulatory subjects, heart rate reserve was the strongest predictor of HRR, accounting for 54 and 45% of variance in HRR, respectively ($P<0.01$). In SCI subjects, heart rate reserve accounted for roughly 54, 77 and 76% of variance in HRR at 2, 5 and 8 min, respectively. Similarly, among ambulatory subjects, heart rate reserve accounted for roughly 45, 77 and 84% of the variance in HRR at 2, 5 and 8 min, respectively.

Discussion

The ability of heart rate to recover after exercise is related to the capacity of the cardiovascular system to reverse ANS (withdrawal of sympathetic activity) and baroreceptor (detection of changes in blood pressure and inhibition of sympathetic discharge) adaptations that occur during exercise, often termed vagal reactivation.[4] Vagal predominance, as evidenced by indirect measures such as heart rate variability, tilt table or cold pressor tests, and the heart rate response to exercise and recovery has long been associated with better cardiovascular health.[2,4,17] This has

Table 3 Correlation coefficients between HRR and exercise test responses among SCI subjects

	Paraplegia			*Tetraplegia*		
	HRR 2 min	*HRR 5 min*	*HRR 8 min*	*HRR 2 min*	*HRR 5 min*	*HRR 8 min*
Age	-0.45**	−0.43**	−0.38*	−0.21	−0.20	−0.21
BMI	−0.19	−0.22	−0.20	−0.38	−0.35	−0.38
Resting SBP	−0.14	0.01	0.06	0.09	0.10	0.13
HR_{rest}	0.10	−0.06	0.07	−0.04	−0.27	−0.35
HR_{peak}	0.66**	0.75**	0.78**	0.78**	0.74**	0.76**
$HR_{reserve}$	0.67**	0.87**	0.82**	0.74**	0.81**	0.87**
VO2peak	0.37*	0.50**	0.46**	0.58**	0.53**	0.57**
VE_{peak}	0.18	0.36*	0.39*	0.23	0.41*	0.40*
RER	0.22	0.41**	0.44**	0.06	0.24	0.26
Peak power	0.01	0.21	0.22	0.28	0.33	0.39*

Abbreviations: BMI, body mass index; HR, heart rate; RER, respiratory exchange ratio; SBP, systolic blood pressure; VE, minute ventilation; VO2, oxygen uptake.
*$P<0.05$; ** $P<0.01$.

Table 4 Multiple regression analyses for HRR at 2 min

	R	R^2	*New variance explained (%)*	P-*value*
SCI subjects				
Variables entered				
$HR_{reserve}$	0.73	0.54	54	<0.01
HR_{rest}	0.75	0.56	2	<0.05
Ambulatory subjects				
Variables entered				
$HR_{reserve}$	0.69	0.45	45	<0.01

Abbreviations: HR, heart rate; SCI, spinal cord injury.

been underscored by the long-established observation that recovery of heart rate is faster in athletes,[4] and the fact that autonomic imbalance, principally a deficiency in vagal tone, is associated with higher mortality.[2,17] Although autonomic imbalance is an important feature that characterizes the level and completeness of injury in SCI, the application of HRR to persons with SCI has not been fully explored.

We observed that absolute HRR was reduced in persons with SCI, and this reduction was accentuated among subjects with tetraplegia (Figure 2). On the surface, this would suggest that persons with SCI have impaired vagal reactivation, which has been repeatedly shown to portend a heightened risk for cardiac events among ambulatory individuals.[2,5,6,17] However, the fact that much of this reduction was attributable to heart rate reserve (that is, HRR was more rapid in subjects with a lower HRrest, a higher HRpeak or both) (Table 4) led us to further explore the association between HRR and heart rate reserve. Considering heart rate reserve in subjects with SCI is important because those with high injury levels in particular tend to have slightly lower resting heart rates and markedly lower peak heart rates.[8,11] By normalizing HRR for differences in HRrest and HRpeak (Figure 1), the effects of differences in heart rate reserve were removed. After normalizing for heart rate reserve, HRR actually declined more rapidly in subjects with tetraplegia (Figure 3). In practical terms, these findings suggest that HRR is strongly related to the exercise level achieved rather than to the level and completeness of injury, and that HRR response reflects a normal pattern of vagal reactivation in subjects with tetraplegia.

Several factors could potentially explain the pattern of HRR in SCI. A reduced HRR may reflect an intrinsic deficiency in vagal reactivation, an impairment in baroreceptor sensitivity, deconditioning associated with high-level SCI, some combination of these factors[1,2,9] or simply a low HRpeak achieved. Although an impairment in baroreceptor sensitivity has been widely described in SCI,[1,17,18] the contribution of deconditioning is suggested by the significant association between peak VO_2 and HRR in SCI subjects in this study (Table 3). In fact, Sedlock *et al*.[10] observed that SCI subjects who were physically active had HRR responses that were similar to those of able-bodied subjects. Duran *et al*.[19] reported a faster HRR at 6 min after exercise after a 16-week training program in a group of thoracic-level SCI subjects. Although we observed a modest association between fitness and absolute HRR in subjects with SCI, we do not have data on activity patterns that would permit a more direct evaluation of the effects of regular exercise on HRR.

The extent to which HRR is related to the peak heart rate achieved has been debated. Recent work among ambulatory subjects from our laboratory[12–14] and others[15] suggests that both the rapidity of HRR and the heightened mortality associated with impaired HRR are largely attributable to heart rate reserve. This contrasts the widely held belief that HRR is principally a function of vagal reactivation, and is independent of exercise capacity or peak heart rate achieved. Although HRR was strongly related to heart rate reserve in this study, much of the variance in HRR was unexplained, particularly in early recovery (Table 4), the time point that has been most closely associated with poor outcomes.[5,6,12,14] It is noteworthy in this context that, similar to HRR, heart rate reserve is also governed by autonomic balance; a lower HRrest is largely related to higher vagal tone, and a higher HRpeak suggests enhanced sympathetic drive, lowered vagal influence or both, at peak exertion. Because of the strong association between reduced HRR and increased risk of cardiovascular and all-cause mortality in ambulatory subjects,[2,5,6] a potential role of HRR as a tool to risk-stratify individuals with SCI exists. However, no such follow-up studies to our knowledge have been performed among persons with SCI. HRR is also considered as a surrogate measure of the integrity of the ANS in ambulatory subjects,[2,4,17] although such data are sparse after SCI. Because the degree of autonomic dysfunction profoundly influences the clinical course and treatment in SCI, the potential of a simple, noninvasive index such as HRR to quantify autonomic dysfunction is attractive. Further studies are needed to assess the role of HRR in SCI and how HRR is influenced by impaired autonomic function associated with different levels of injury.

In summary, absolute HRR is impaired in SCI subjects compared with ambulatory subjects, with the most marked impairment occurring in tetraplegic subjects. However, HRR is strongly related to heart rate reserve, indicating that HRR

is largely attributable to the exercise level and heart rate achieved. This study was limited by the fact that few women were available, and the fact that HRR is only an indirect measure of autonomic function. Although HRR has been suggested to have applications for characterizing autonomic function in SCI, studies considering heart rate reserve and more direct measures of autonomic function (for example, tilt table testing, heart rate variability, pharmacologic manipulation) are necessary to further investigate the relationship between HRR and autonomic impairment before this index is suitable for clinical application in SCI individuals.

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